MEDICAL PARASITOLOGY
AND ZOOLOGY
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By
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Foreword by
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Dedicated to

THE MEMORY OF MY PARENTS

and to my former teachers

SIR PATRICK MANSON

an outstanding pioneer in parasitology, tropical medicine, and hygiene

SIR RONALD ROSS

one of his celebrated pupils, both of whom by their research and teaching have inspired others in further developing these subjects
The presentation of the essentials of medical parasitology and zoology to the prospective practitioner of medicine is usually given scant or at best inadequate attention in our medical colleges. This unfortunate neglect of the subject may be ascribed to the overcrowded state of the medical curriculum but further, in such time as is allotted, there may be an unsatisfactory adaptation and coordination of teaching methods to the time available and to the probable future needs of the practicing physician. As a result the average hospital interne too often finds himself incompetent to apply the laboratory aids which are available for the diagnosis of the more common parasitic infections.

It seems to be customary in our medical schools to place responsibility for instruction in this subject with one or another of the several departments of bacteriology, clinical pathology, and public health. At Cornell University Medical College, for a number of years this instruction has been a function of the Department of Public Health and Preventive Medicine. It is given as a separate short course (to the second year students). The lectures, demonstration exhibits and laboratory exercises are systematically coordinated. The objective has been to give the student not only practical experience in the diagnosis of the more common and important parasitic diseases of man, but also to teach him some of the procedures essential to their control and to the prevention and treatment of the pathological conditions caused by their presence.

Because of the limited class time available for such a course, collateral reading is particularly important. Although there are
several works available for the more advanced student in this subject, there appears to be none meeting our requirements of an inexpensive textbook adapted to the specific needs of the medical student, the clinician, and the teacher alike. Accordingly, this volume has been prepared, embodying the accumulated staff experience gained from teaching this subject to medical students for many years and the experience of the author in laboratory diagnosis in temperate as well as in warmer climates.

The importance of a knowledge of medical parasitology to both the clinician and the public health official is increasing with the acceleration of means of travel and transportation. The appearance of certain parasitic diseases in regions where they were before unknown, and the wider spread from previously limited endemic and epidemic areas, is a matter of common knowledge. It would thus seem of growing concern for the protection of the public health that the graduates of our medical schools should have an adequate and precise working acquaintance with present day methods of diagnosis, treatment, and prevention of these diseases.

John C. Torrey

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Animal parasites stand more or less apart from other important disease-producing agencies, such as viruses, the pathogenic bacteria, and parasitic moulds. Except in the protozoa, multiplication of the animal parasite within the primary host does not frequently occur among members of these more highly organized pathogens. Furthermore there is manifested a general tendency to follow a course of development of varying degrees of complexity. Although increase in numbers within the host occurs in most protozoa, encystment is also the rule in order to enhance the chances of successful transfer to a new host. In only a few human intestinal protozoa is true encystment not definitely known to occur, notable examples being *Dientamoeba fragilis* and the trichomonads.

Whereas direct or indirect transfer of the multiplying organisms themselves constitutes a frequent mechanism of infection in case of bacteria, animal parasites must often undergo certain prior and subsequent functional and morphologic changes. This may occur in distant parts of the original host, in the external environment, or in a secondary host or hosts. Such processes or alternate phases of development in the life of a parasite take place in several ways as follows:

1. The larval young may be born within the original host, migrate to other parts of the same host and undergo some further development to await suitable transfer to a fresh host. A striking example of this is *Trichinella spiralis* in trichinosis. In the case of the pinworm (*Enterobius vermicularis*) the pregnant female makes its way to the perianal region to deposit there her eggs. These develop rapidly to larvae within the original egg shell. Transfer to the same or another human host is then often a matter
of hand to mouth carriage. *Hymenolepis nana*, the human dwarf tapeworm, in children especially, may be auto-inoculated similarly also since no intermediate host is required as in most other flatworms.

2. The larvae, or eggs from which larvae subsequently hatch, go through a simple process of development in the external environment before entering another host. Simple examples of this are the hookworms in which there is no multiplication beyond the laying of eggs by the adult female in the intestine; in case of *Strongyloides stercoralis*, however, strikingly similar in many respects to the hookworms, there are also one or more free-living generations, the progeny of which are capable of infecting new hosts.

3. The eggs of the flatworms and sexual forms of certain other highly specialized parasites, such as the malaria plasmodia, reach a second host of another species where a totally different process of development takes place. The host, in which the adult or sexual stage is passed, is referred to as the primary, definitive, or final host; that in which the larval or asexual stage occurs, the intermediate or secondary host. In case of certain protozoa such as *Plasmodium*, where an intermediate host is not required, an interval of time is required after passage in the feces before infection of a new host may occur successfully. During this period a multiplication-development continues preparatory to parasitization of a new host.

Medical parasitology and zoology comprise all those animal organisms which have to do with the production or transmission of disease states in man. For practical purposes this subject may be divided, for the most part, into medical parasitology and entomology. Parasitology may in like manner be subdivided into protozoology and helminthology. The former includes one-celled parasites while the latter is practically limited to the roundworms and flatworms (flukes or trematodes and tapeworms or cestodes).*

* For a general working classification of the organisms with which this treatise deals, the reader is referred to the glossary.
Teaching of this important subject to medical students, in the limited time usually allotted, constitutes a difficult problem. The plan developed at Cornell University Medical College, New York City, seems to us to have the distinct advantages of conciseness, logical appeal, and a certain degree of simplicity. The following outline epitomizes the work in these subjects as is given in ten sessions of three hours each. The first half to three quarters of an hour of each period is usually assigned to one of the several staff members to be devoted to presentation of matter not adequately covered in assigned readings or to questioning members of the class regarding the work of the previous period or that to be undertaken at the current session.

1st session—Amoebiasis: The first forty minutes is devoted to an illustrated lecture by a prominent clinician in tropical medicine and hygiene.

2nd session—Amoebiasis, Intestinal flagellates and ciliates with talk on flagellates, ciliates and differential diagnosis of protozoa.

3rd session—Mosquitoes and disease transmission: (1) Life-cycle of mosquito with talk on mosquitoes. (2) Malaria parasite in mosquito with talk on malaria plasmodia phases in the mosquito.

4th session—Mosquito-borne diseases of man: (3) Malaria parasites in man with talk on malaria plasmodia phases in man. Talk on elephantiasis, dengue and yellow fever.

5th session—Intestinal worms associated with pollution by fecal excrement, particularly of the soil, with talk on roundworm infections.

6th session—Intestinal worms involved in parasitization or contamination of food eaten by man: (1) Trichinella spiralis and Trichinosis with talk on Trichinosis. (2) Trematodes and fluke diseases with talk on the fluke diseases.

7th session—Intestinal worms involved in parasitization or contamination of food eaten by man: (3) Cestodes and tapeworm infections with talk on tapeworm infections.

8th session—Insects (other than mosquitoes) and disease transmission: (1) Mites, ticks and lice with talk on tick- and louse-borne diseases.

9th session—Insects (other than mosquitoes) and disease transmission: (2) Fleas and bugs with talk on flea-borne diseases.
(5) Flies and disease transmission. (6) Fly-larvae and disease; talk on fly-borne diseases and the myiasis.

10th session—Poisonous arthropods, fishes, and snakes: Principal poison-injecting groups among the arthropods, fishes, snakes, and lizards, preceded by illustrated talk on these arthropods and vertebrates. Talk on snakes, nature and treatment of their bites.

Although we have been able to teach in the manner outlined something of the rudiments of these subjects in the thirty hours of class time available, it seems obviously a handicap to both instructor and student to be obliged to do so. A more consistent relative rationing of the students' time devoted to the basic medical sciences would undoubtedly result in the allocation of considerably more time to medical parasitology and closely related subjects.

In view of the rapidly accelerating demands which will be made upon physician, surgeon, and health officer in the wake of the present world-wide conflict, it seems probable that those in authority in all progressive teaching institutions, medical colleges in particular, will soon come to comprehend the true relative value of a practical working knowledge of medical parasitology and zoology to the practitioner of medicine. In fact, it appears entirely likely that the teaching of these and kindred subjects may soon receive the attention which their importance in the enlarged horizons of clinical and preventive medicine strongly suggest.

This text is based on experience acquired in attempts to present the rudiments of medical parasitology and zoology to undergraduate medical students in a minimal number of hours. At its inception it was realized that a guide to these subjects must of necessity contain related material which it would not be practicable to present during the class sessions. In order to subordinate this relevant information, it was deemed desirable to segregate in appendix and glossary the more directly useful of such subject-matter, and thus avoid tempting the student with memorizing it. By so doing, the author aspires to bring to medical students, clinicians, and laboratory technicians alike a practical presenta-
tion adaptable to both instruction and ready reference. Moreover, no attempt is made at elaboration, but rather to clarify, epitomize, and simplify in so far as practicable.

Certain specific nomenclature with reference to cytology, morphology, and life cycle must necessarily be employed. In this respect the author has endeavored to abridge and minimize wherever possible. For more detailed information direct references are made to a few readily available larger standard American and English textbooks. In addition, the appendix is utilized for presenting less essential but useful relevant information, which if incorporated in the body of the text might tend to burden the student unduly. The employment of such a method, it is believed, will tend to rid the subject of some of its terrors. Finally it is hoped that the simplified descriptions, table-summaries and illustrations will serve to carry along the student's or reader's interest and lead to a more practical understanding of the essentials of medical parasitology and zoology.

I am much indebted to Dr. Wilson G. Smillie for material assistance and encouragement in the course of preparation of the manuscript; to Dr. John C. Torrey and Dr. Morton H. Kaln for helpful advice and counsel; to Dr. D. H. Wenrich, Dr. C. H. Curran, and Mr. Karl Kauffeld for reading the chapters on Protozoology, Arthropods, and Poisonous Snakes, respectively; to Dr. Suzanne Howe for assistance in reading page proof; to Dr. Irving Rappaport and Mr. Edward Clifford for invaluable assistance in preparation of illustrations and for devotion to other essential details in the development of material; to Miss Ada Bartoli for help in preparing the bibliography and glossary.

Authors and publishers of whom requests were made for using or reproducing illustrations have been most courteous, and specific acknowledgment of source of such is made throughout the text and appendix. As immediate sources of much valuable factual matter, special reference should be made to Dr. Philip H. Manson-Bahr, editor of Manson's Tropical Diseases; Rear Admiral E. R. Stitt, Surgeon General, Medical Corps, U. S. Navy (Ret.); Colonel Charles F. Craig, U. S. Army Medical Corps (Ret.) and Professor
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PART ONE

PROTOZOA PARASITIC IN MAN
A brief differential study of the five generally accepted intestinal amebae of man will be made in this chapter. These amebae must be distinguished not only from one another but also from all other constituents of stools. Artefacts, for example, are frequently mistaken for amebae or their cysts. It is essential that the observer not be misled by superficial resemblances and that true parasites be identified by their characteristic features.

Amebiasis, which often presents an obscure, uncertain and varied symptomatology, may be diagnosed usually by identification of the causative organism through a simple microscopic examination of the feces. The information obtained, in addition to aiding in establishing a positive diagnosis, is of importance in gauging the effects of treatment and in the control and prevention of this condition.

**Technique of Preparing and Examining Fresh Material**

Before undertaking a comparison of the five amebae as they appear in fresh, unchilled stools, it may be helpful to outline an approved method of preparing the material for microscopic study.

If the stool specimen is not sufficiently transparent to be observed by transmitted light, a diluent must be added. Body-warm, freshly prepared, sterile physiologic salt solution is satisfactory for this purpose. A moderate sized drop of this diluent is placed on a glass slide (1 by 3 inches) warmed to body temperature,

* In order to minimize the amount of textual material, the definitions of technical words, phrases, and zoological classification have been segregated and assembled in a glossary following the appendix.
and then a selected small portion of the material to be examined is stirred into it, care being taken not to have the mixture too concentrated. The optimum density is readily recognized after a little experimentation. A clean thin coverglass (for hygienic reasons, preferably 3/4 inch square) held at an angle (preferably with coverslip tweezers) in a somewhat horizontal position, is then placed, in contact with the slide, with one edge near the prepared drop. The coverglass is brought into contact with the fluid, and allowed to fall gently upon the drop. With this technique a satisfactory fresh preparation may be made after a little practice.

The completed warm preparation is placed on the stage of the microscope for rapid observation. If a longer examination is to be made, some means of keeping the slide at body temperature is desirable. By far the most practical device for this purpose is an electrically heated, warm stage equipped with a satisfactory temperature control mechanism. Such a supplementary stage is shown in Figure 1. A fairly good substitute may be constructed for use with a small alcohol lamp or other flame as the source of heat. Specifications for the construction and employment of such a warm stage are given in Appendix I, Figures 91 and 92.
AMEBAE

MICROSCOPIC STUDY OF FRESH PREPARATION

With a working knowledge of the use of the compound microscope, and attention to the simple details just outlined, the student is prepared to search for and to observe living motile amebae and other protozoa in specimens of feces or other material. In Table I are assembled data relating to the living vegetative or trophic forms of the five human intestinal amebae. These, in lieu of satisfactory graphic methods of presentation, are brief word pictures of what can be observed under the microscope when one or more of these amebae are present in the stool from which the preparation was made.

Aptitude in determining the exact nature of objects observed through the microscope depends on ability to apply this knowledge. Such skill can be obtained only by painstaking attention to details and logical deductions based on the various items of evidence available to the examiner. The method of using the compound microscope in parasitology and the various means of measurement of microscopic objects are described in Appendix II. Failure to observe the practical points outlined there, as well as lack of knowledge of how to measure the size of microscopic objects, may lead to failure to recognize the true nature of objects seen, and to false interpretations or wrong deductions.

IODINE-STAINED SPECIMENS

In the saline suspension prepared as described, cyst forms of these amebae and other intestinal protozoa, if present, may be observed and compared with those in a similar preparation of the stool made in Lugol's solution (preferably half-strength) or other similar preparation of iodine and potassium iodide. The so-called "iodine preparation" method is applicable generally to a direct study of all the intestinal protozoa, particularly when it is employed in conjunction with the saline preparation. Generally speaking, the iodine tends to bring out more or less clearly the chromatin topography of the nucleus and the glycogen distribution in the cytoplasm. However, a satisfactory result with this stain
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<th>3</th>
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<tr>
<td><strong>Average diameter</strong></td>
<td>12 to 35 μ</td>
<td>15 to 30 μ</td>
<td>9 to 13 μ</td>
<td>6 to 10 μ</td>
<td>3 to 12 μ</td>
</tr>
<tr>
<td><strong>Motion</strong></td>
<td>Active progression in a definite direction. Form is elongated in motion</td>
<td>Most strains are not actively progressive but merely change in conformation</td>
<td>Many strains in culture like that of <em>E. histolytica</em>. The majority, however, are like <em>E. coli</em></td>
<td>Some organisms like that of <em>E. histolytica</em> (except the ameba is very small) but the majority merely change in form and do not move progressively</td>
<td>Show indefinite progression or merely change in conformation</td>
</tr>
<tr>
<td><strong>Color</strong></td>
<td>Faint green</td>
<td>Gray</td>
<td>Faint green</td>
<td>Gray</td>
<td>Gray</td>
</tr>
<tr>
<td><strong>Pseudopodia</strong></td>
<td>Finger-like with smooth outline when not in progressive motion. Ectoplasm is clear, glasslike and is easily discernible. When in progressive motion the ectoplasm may not be differentiated. One-third to one-quarter of the parasite is ectoplasm</td>
<td>Usually blunt, but it may be like <em>E. histolytica</em>. The ectoplasm is often not clearly differentiated in the pseudopodia; sometimes more of the bulk of <em>E. coli</em> (tropic) will be clear ectoplasm</td>
<td>Like <em>E. histolytica</em> or very broad with coarsely indented outline. One-half to one-third of the parasite is ectoplasm and is easily differentiated</td>
<td>Like <em>E. histolytica</em>. One-half to one-third of the parasite is ectoplasm and is easily differentiated</td>
<td>Often comprises one-half of the organism. Outline is often indented</td>
</tr>
<tr>
<td><strong>Visibility of nucleus (oil immersion lens)</strong></td>
<td>Usually difficult to visualize except when the nucleus passes into the pseudopodia and is contrasted against the clear ectoplasm</td>
<td>Quite clear. It is much more readily seen than that of <em>E. histolytica</em></td>
<td>The karyosomes can be defined with ease</td>
<td>The karyosomes can be defined with ease</td>
<td>Difficult to distinguish from ingested bacteria</td>
</tr>
<tr>
<td><strong>Endoplasmic inclusions of diagnostic significance</strong></td>
<td>Red blood cells are typical and diagnostic. Degenerated and culture forms contain bacteria</td>
<td>A voracious feeder but usually does not ingest red blood cells. Bacteria and starch grains are the principal inclusions</td>
<td>Bacteria</td>
<td>Bacteria</td>
<td>Bacteria</td>
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depends on having a reasonably fresh solution and a sufficient concentration of the iodine in the final mixture, and emulsifying the fecal material in it in the same manner as for the fresh saline preparation.

Iodine preparations need not be kept at body temperature. Time may be saved by making the two preparations on the same slide. This practice, however, is not recommended where more extended examination of the saline preparation is to be made on the warm stage, since under these conditions the iodine solution tends to evaporate too rapidly. The iodine stain often brings out sufficient nuclear detail to reveal the character and position of the endosome (karyosome) within the nucleus. It also aids in confirming the presence of chromatoidal material observed in the saline preparation by its failure to stain. Iodine, furthermore, reveals the presence of glycogen and other types of vacuole. Not infrequently the information obtained through comparison of the saline and the iodine preparations is sufficient to establish clinical diagnosis.

HEMATOXYLIN-STAINED SPECIMENS

Although for rapid presumptive diagnosis, iodine staining affords an excellent adjunct for observing protozoa microscopically, the results to be achieved through its use fall far short of those to be derived from study of properly prepared permanent iron-alum hematoxylin smears. Examples of the information to be obtained from proper staining of the intestinal amebae by this method are shown in Figure 2 and briefly described in Tables II and III. Comparative study of these data is necessary to an understanding of the more detailed descriptions which are to follow. A simplified rapid method for carrying out the hematoxylin staining technique will be described briefly here; a more detailed account is given in Appendix IV.

Three points of importance in making good permanent stained smears are: (1) careful selection of material to be examined, (2) proper preparation of the smear, and (3) avoidance of drying of the smear before fixing and at all subsequent stages of
Fig. 2. Graphic differentiation of Amebae, × 850 app. This figure illustrates Tables II and III and the more detailed descriptions of the human intestinal amebae in text. (Courtesy of the Army Medical School and Museum.)
staining, differentiating, dehydrating, clearing, and mounting. The necessity for using the greatest care in carrying out these details cannot be overemphasized. In selecting the material, mucus in or on the stool specimen should be included as well as other representative portions, particularly if the preliminary microscopic examinations in saline and iodine suspensions have revealed anything suggestive. The smear should be fairly thin and daublike in appearance; this may be accomplished by thoroughly emulsifying it with the broader (blade) end of a common wooden toothpick, rather than by spreading as in making a blood smear for staining. If the stool material is solid, it should be emulsified upon the slide with physiologic salt solution just prior to smearing. Drying before as well as after fixing must be meticulously avoided at this juncture.

The method to be described for making a permanent smear is not so satisfactory for bringing out the finer morphologic details as is the regular Heidenhain staining technique, but it is excellent for rapid diagnosis in routine work, particularly when there are few organisms in the smear and time is important. After the preparation has been fixed in body-warm Schaudinn's solution * for from five to ten minutes, staining is carried out as follows:

1. Rinse by immersion in 50 per cent alcohol for several minutes.
2. Transfer to 70 per cent alcohol and allow to remain for two minutes.
3. Again transfer to 70 per cent alcohol containing tincture of iodine (enough to give a port-wine color to the alcoholic solution) and allow to remain not less than five minutes. (This procedure is for the purpose of removing the unused mercury salts of the fixative.)
4. Then rapidly pass slide through 70 per cent, 50 per cent, and 30 per cent alcohol into running tap water, allowing about one-half to one minute for passage through each strength of alco-

* Schaudinn's solution or fixative consists of two parts of a saturated aqueous solution of mercuric chloride to one part of 95 per cent alcohol with 2-5 per cent of glacial acetic acid commonly added just before using.
<table>
<thead>
<tr>
<th></th>
<th>2 E. histolytica</th>
<th>2 E. coli</th>
<th>3 I. batesii</th>
<th>4 E. mansa</th>
<th>5 D. fragilis</th>
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<tbody>
<tr>
<td>Average diameter</td>
<td>12 to 35 µ</td>
<td>15 to 30 µ</td>
<td>9 to 15 µ</td>
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<tr>
<td>Nuclear membrane (stains faintly or not at all)</td>
<td>Lined with minute, fairly even sized grains of chromatin which stain deeply</td>
<td>Lined with coarse irregular sized grains or bars of chromatin which stain deeply</td>
<td>A few poorly staining widely separated chromatin grains on nuclear membrane</td>
<td>Chromatin on nuclear membrane in thin line and stains poorly</td>
<td>Chromatin on nuclear membrane in thin line and stains poorly. Commonly two nuclei</td>
</tr>
<tr>
<td>Karyosome (Endosome)</td>
<td>Short rod or globule of small diameter, centrally suspended within the nucleus. Regular outline. Stains deeply and uniformly</td>
<td>Short rod or ball of irregular outline, usually eccentric. Diameter greater than that of E. histolytica</td>
<td>Similar to that of E. mansa but larger and more apt to contain a poorly staining central portion. Causes the nucleus to appear like an eye with a widely dilated pupil</td>
<td>Very large, central or eccentric, composed of one, two or more deeply staining masses in a lighter staining matrix. Outline often irregular and oblong</td>
<td>Composed of several minute deeply staining discrete grains</td>
</tr>
<tr>
<td>Linin network (stains faintly or not at all)</td>
<td>Contains no chromatin grains between the karyosome and nuclear membrane</td>
<td>Sometimes contains grains of chromatin. Region just without karyosome halo often appears cloudy after staining</td>
<td>Like a web when defined by an excellent stain</td>
<td>It is not often discernible. Consists of a few short lines from the karyosome halo to the nuclear membrane. Karyosome usually the only structure visible in the nucleus</td>
<td>Not demonstrable</td>
</tr>
<tr>
<td></td>
<td>AMEBAE: CYSTIC STAGE</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td></td>
<td><strong>1</strong> E. histolytica</td>
<td><strong>2</strong> E. coli</td>
<td><strong>3</strong> I. bütschlii</td>
<td><strong>4</strong> E. nana</td>
<td><strong>5</strong> D. fragilis</td>
</tr>
<tr>
<td>Average diameter</td>
<td>5 to 20 µ</td>
<td>20 to 30 µ</td>
<td>7 to 13 µ</td>
<td>5 to 8 µ</td>
<td></td>
</tr>
<tr>
<td>Number of nuclei</td>
<td>1 to 4, rarely more. Mature cysts contain 4 nuclei</td>
<td>1 to 8, rarely more. Mature cysts contain 8 nuclei</td>
<td>1 or rarely 2</td>
<td>1 to 4, rarely more. Mature cysts contain 4 nuclei</td>
<td></td>
</tr>
<tr>
<td>Visibility of nuclei in the unstained living state</td>
<td>Poor but discernible with the oil immersion lens</td>
<td>Good</td>
<td>Good</td>
<td>Good</td>
<td>Cystic stage unknown</td>
</tr>
<tr>
<td>Shape</td>
<td>Generally spherical or nearly so</td>
<td>Generally longer than broad and one side may be less curved than the other</td>
<td>Great irregularity of shape and outline is common</td>
<td>Irregularity of shape is common. Generally oval</td>
<td></td>
</tr>
<tr>
<td>Reserve food inclusions (these disappear in old cysts and are not constant in young)</td>
<td>Bar-shaped chromatoid bodies in 0–90 per cent of cysts. Sometimes a small amount of glycogen is present in young cysts. It is diffuse and stains a light brown with iodine.</td>
<td>Acicular chromatoid bodies present in about 10 per cent of cysts. A large amount of glycogen may be present and push the nucleus against the cyst wall</td>
<td>Masses, grains or rods of volutin may be present but these are not characteristic. The glycogen, almost invariably present, is large in amount, smoothly outlined, and stains a deep brown with iodine. This iodine body is characteristic and diagnostic</td>
<td>Small granules or masses of volutin and glycogen may be present. Neither is characteristic</td>
<td></td>
</tr>
</tbody>
</table>

**Table III**
hol and enough time in the water to remove all the remaining alcohol.

5. Mordant for ten minutes or longer in 2 per cent to 4 per cent aqueous solution of iron-alum (ferric ammonium sulphate), heated to 37° C.

6. Rinse well in water.

7. Stain for one-half hour or longer in 0.5 per cent aqueous solution of well ripened hematoxylin heated to 37° C.

8. Wash in running water for at least five minutes, or in several changes of water for fifteen minutes.

9. Dehydrate by passing through alcohol of 50 per cent, 70 per cent, 85 per cent, 95 per cent strength.

10. Decolorize or destain in saturated alcoholic (95 per cent) picric acid solution for from four to seven minutes, depending upon the thickness of the smear. Differentiation under the microscope ordinarily is unnecessary.

11. Wash in several changes of 95 per cent alcohol to remove excess of picric acid.

12. Place in 95 per cent alcohol containing a small amount of potassium acetate until the blue color is restored.

13. Immerse in diaphane solvent for one-half to one minute and mount directly in diaphane; or place successively in absolute alcohol, five minutes; absolute alcohol and xylol (equal parts), 5 minutes; xylol, 5 minutes; and mount in Canada balsam or other similar medium.

14. Place slide in incubator overnight or longer to hasten hardening of the mounting medium.

Although specific diagnosis often can be made without resort to a hematoxylin-stained specimen, instances are not infrequent in which the most expert microscopist may experience more or less uncertainty without good stained smears. In Tables II and III the data to be gleaned from a microscopic study of hematoxylin-stained amebic stool smears are summarized, while in Figure 2 are shown typical forms of the trophozoite, precyst, and cyst stages of the five human intestinal amebae, respectively. For com-
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Comparison, trophozoites of Endamoeba gingivalis and Amoeba proteus are also included. It should be noted how closely Endamoeba gingivalis resembles Endamoeba histolytica, and how different Amoeba proteus is from either of these.

The five human intestinal amebae will be described in the order in which they appear in the columns in Figure 2 and in Tables I, II, and III; namely, Endamoeba histolytica, Endamoeba coli, Iodamoeba butschlii, Endolimax nana, and Dientamoeba fragilis, in their unstained and stained trophozoite, precystic, and cystic stages. In addition, the so-called coprozoic or free-living amebae occasionally found in the intestine of man will be briefly referred to.

PATHOGENIC AMEBAE

Endamoeba histolytica is the only ameba of the human intestine which is generally accepted to be pathogenic. However, it seems likely that at least one other species (namely, Dientamoeba fragilis) in certain individuals may be associated with recurring episodes of varying degrees of diarrhea which some attribute to its rapid multiplication. This organism will be described in connection with the nonpathogenic or commensal group to which it is generally assigned.

Endamoeba histolytica (Schaudinn, 1903) Hickson, 1909

Trophic Stage. The trophozoites of E. histolytica (Fig. 2) vary in size, depending on such factors as environment and rate of multiplication. The actual sizes range from 7 or 8 to 40 µ or more. Rounded up, the average diameter is usually from 18 to 25 µ. A conspicuous feature of this form is the differentiation of ectoplasm from endoplasm. When the organism is in motion this feature is not always noticeable. However, when it remains more or less in one place for a short time,* sending out one pseudopodium and then withdrawing it to send out another, the ectoplasm is conspicuously clear and the endoplasm is finely granular.

* Nonmigrating E. coli also sometimes throw out clear pseudopodia very similar in appearance to those of E. histolytica.
With proper illumination the organism usually has a slightly greenish hue.

In the animal body, the trophic stage of *E. histolytica* appears to be a true tissue parasite, which lives on body cells and fluids to the exclusion of practically all other types of food. The damage it causes varies quite widely. In fresh diarrheal stools, *E. histolytica* rarely contains ingested bacteria, but red corpuscles or leucocyte fragments are frequently observed. Except when it is undergoing degeneration, the only vacuoles to be seen in the endoplasm are food vacuoles.

In a favorable environment and when not dividing or nearing encystment, the trophozoites of *E. histolytica* frequently move rapidly, progressively, and more or less continuously. Their movement is sluglike and associated with ability to cling to the surface and make progress even against or athwart currents in the surrounding medium. The organism appears to progress by extending a pseudopod as far as possible, fixing the tip of this pseudopod on a solid surface, then pushing out a new pseudopod from a point near the fixed tip of the previous one. Thus it may be seen that the ameba, with one fixed point, is able to push out a second pseudopod and allow the rest of its body to flow into it until the tip of the first pseudopod becomes the rear end of the ameba. This process is repeated again and again. A striking characteristic of the moving ameba is the trail of debris which is carried along behind it.

The nucleus is disklike in appearance, with a diameter about one-sixth that of the ameba. Ordinarily it is rather difficult to discern in the living state, but when moving with the protoplasmic currents it may be occasionally seen, tilting slightly as it floats along. The nucleus is most likely to be visible contrasted against the ectoplasm, as the endoplasm flows into the ectoplasm of the pseudopod. When stained with hematoxylin, the chromatin of the nucleus is seen to be distributed in delicate beads along the inner surface of the nuclear membrane. The linin network is practically free from chromatin, while the karyosome consists of a fine centrally placed black granule, Figures 2 and 3 (from draw-
ings of trophozoites of *E. histolytica* stained with Heidenhain's hematoxylin, which should be compared with corresponding descriptions given in column 1 of Table II).

![Diagram of developmental cycle of *Endamoeba histolytica*](image)

Reproduction is by binary fission; this process may be observed on the warm stage, particularly in preparations made from rapidly growing cultures. It usually consists of an equal division of both nuclear and cytoplasmic elements. In rapidly growing cultures, however, there may be marked unequal divisions of the cytoplasm, giving rise to smaller and larger organisms. Correspondingly small cysts in stools are thought by some observers to belong to a different race from the larger sized ones encountered more frequently.

**Precystic Stage.** The term "precystic" connotes simply transition from the trophic to the cystic stage; it is characterized by relatively smaller size, greatly diminished motility, and absence of solid food particles. The chromatin granules in the nucleus become coarser, sometimes rendering it somewhat visible in the fresh state when observed in subdued light under high-dry lens. Rod-shaped *chromatoid bodies* occasionally appear in the endoplasm when the organism is in process of forming a cyst wall.
It is thought that adverse environmental conditions predispose to precyst and ultimately to cyst formation. When such changes are too sudden the amebae may be unable to survive; in this case they round up and die. Under optimal conditions, *E. histolytica* may continue a vegetative (trophic) existence for prolonged periods, particularly when the resistance of the host's tissue is low. However, this organism must sooner or later encyst in order to survive the ordeal of passage to a new host, a transition that occurs under natural conditions.

When treated with iodine the precysts of *E. histolytica* stain a diffuse light brown, indicating the presence of glycogen. Staining with hematoxylin brings out the characteristic *E. histolytica* morphology in cytoplasm and nucleus. Chromatoid bodies, if present, will be revealed by their smooth outline and intense bluish black color. Compare carefully the illustration of *E. histolytica* precyst (H.H. stain) in Figures 2 and 3 with the corresponding description given in Table III.

**Cystic Stage.** There is no real line of demarcation between the precystic and the cystic stages. When the cyst wall is completely formed, the cystic stage begins, but development is incomplete. Prior to this time, gaps in the cyst wall are indicated by the fact that small pseudopods at times may be seen protruding. Subsequent changes within the cyst (Fig. 3: 6, 7, and 8) consist principally of nuclear divisions and the gradual disappearance of stored glycogen and chromatoid material. The latter, however, is present visibly in only about 20 per cent of newly formed cysts. One or more chromatoid masses in the form of large refractile rods with rounded ends (sausage-like) may be seen. When present, chromatoid bodies may be detected readily in the fresh, unstained saline preparation by reducing the intensity of the illumination. They are invisible in iodine preparations but stain a deep bluish black with hematoxylin. The iodine stain may, on the other hand, reveal small to large glycogen vacuoles in newly formed cysts. These stain light mahogany brown.

The recently formed cyst (Fig. 3: 6) contains but one nucleus, which is of the same type as that of the trophic stage. This single
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nucleus soon divides, then each daughter nucleus divides again, resulting usually in four small nuclei similar morphologically to all their antecedents, beginning with the original trophozoite. Although mature *E. histolytica* cysts usually contain only four nuclei, a larger number may occasionally result from further nuclear division. These nuclei are difficult to discern in saline preparations even under the oil-immersion objective, but they stand out distinctly in properly made iodine preparations; whereas the chromatoid bodies, if present, become invisible. Good hematoxylin staining affords the most perfect picture obtainable of the morphology of the *E. histolytica* cyst as well as of the precyst and the trophozoite. The features to be looked for are depicted in the first column of Figure 2 and in Figure 3, and briefly described in Table III.

Cysts of *E. histolytica* are generally round, but they may be somewhat distorted in microscopic preparations. They vary in size from 5 to 20 μ or even more in diameter and their cytoplasm is clear, translucent, and finely granular in structure. In the fresh, unstained state, they present a slightly greenish sheen similar to that frequently observed in rapidly growing *Blastocystis hominis*, but the cytoplasm is less refractile than are the chromatoid bodies when they are present.

**NONPATHOGENIC OR COMMENSAL AMEBAE**

The importance of the presence of nonpathogenic amebae in stools lies in the fact that they may be confused with the pathogenic *Endamoeba histolytica*. One or more of these may be present in stools alone or in association with *E. histolytica*. They are generally considered to be harmless and ordinarily without clinical significance.

*Endamoeba coli* (Grassi, 1879) Casagrandi and Barbagallo, 1895

*Endamoeba coli* is a comparatively innocuous species, living in the lumen of the colon. It has been suspected of exerting harmful activities at times, but very little is known definitely regarding this.
Because this ameba occurs very frequently in the human colon it may be mistaken for *E. histolytica*. The average size of the trophozoite is often greater than that of the trophozoite of *E. histolytica* and the cysts also are usually considerably larger. The life history of *E. coli* (Fig. 2) is similar to that of *E. histolytica*, from which it differs in the following respects:

1. *E. coli* is not associated directly with tissue destruction.
2. Its pseudopods are not so prominent, less clear, and rarely explosive in character; it generally moves sluggishly, usually making little definite forward progress.
3. It rarely ingests red blood cells, but usually contains bacteria, mold spores, starch granules, and sometimes leucocytes.
4. Its cytoplasm is coarsely granular and is not usually so well differentiated into ectoplasm and endoplasm as is that of *E. histolytica*.
5. Its nucleus is more readily visible, more fixed in position, and contains coarse, irregular-sized grains of chromatin on the inner surface of the nuclear membrane.
6. The linin network may have a few grains of chromatin suspended in it and the karyosome is larger and usually eccentrically situated in the nucleus.
7. When first formed, *E. coli* cysts usually contain larger and more marked glycogen vacuoles.
8. The average mature *E. coli* cyst usually contains eight small, fairly typical nuclei similar morphologically to the parent nucleus.
9. The chromatoid bodies, when present, generally have sharp pointed acicular ends.

A study of columns 1 and 2 of Tables II and III in conjunction with the illustrations given in columns 1 and 2 of Figure 2, and in Figure 3 should serve to make clear the differences between *E. coli* and *E. histolytica*.

**Endamoeba gingivalis** (Gross, 1849) Smith and Barratt, 1915

*Endamoeba gingivalis*, the sole ameba of the mouth of man, may be found in pus pockets of pyorrhea alveolaris, cavities of carious teeth, tartar deposits, and even in buccal erosions or
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abscesses. Its presence may not necessarily be indicative of carelessness in the care of the mouth. Fortunately it manifests little if any direct invasive power. Since a definite cyst stage has not been proved, the range of its distribution is assumed to be human mouths. However, it has been stated that apparently similar amebae have been found in the mouths of dogs which have a condition of the gums resembling pyorrhea in man. The trophozoites (Fig. 2) are almost identical with those of *E. histolytica*, and visible ingested material, which is usually abundant, generally consists of pycnotic bodies and remnants of ingested cells other than red blood corpuscles.

According to reports in the literature, when animals susceptible to infection with *E. histolytica* have been inoculated with *E. gingivalis*, they have showed no positive evidence of pathogenicity in the colonic mucosa. Attempts to transplant this ameba direct from the human mouth to the gums of various normal laboratory animals have been unsuccessful. However, the fact that I have been able to produce amebic lesions in the colons of cats with *E. histolytica* by using accessory traumatizing agents, indicates that certain types of tissue injury may play a role in determining not only the original infection of gum tissues by *E. gingivalis*, but also in subsequent continuance of the infection. In support of this opinion, it may be pointed out that proper instrumental treatment and simple dental prophylaxis still give the best results in the treatment of amebic pyorrheic conditions. Kofoid (1929) was successful in implanting *E. gingivalis* only in the mouths of dogs already presenting abnormal conditions of the gums.

*Iodamoeba bütschlii* (williamsi) v. PROWAZEK, 1911;
DOBELL, 1919

*Iodamoeba bütschlii* is one of the rarer nonpathogenic amebae that inhabit the colon of man. It is most commonly found in persons residing or traveling in warm countries. The trophozoites (Fig. 2), which resemble those of *E. coli* in form and habit but not in size, vary from about 9 to 15 μ in diameter. The nucleus
possesses a large round or oval eccentric karyosome containing most of the chromatin. It is, however, the cyst form which is usually passed in stools. These cysts are generally somewhat irregular in shape and usually contain one nucleus (occasionally two nuclei) similar morphologically to that of the trophozoite.

The most characteristic feature of the cyst of *I. bütschlii* is its reaction when stained with Lugol’s iodine solution; one or more large, deeply staining darkish brown vacuoles containing glycogen are revealed. These vacuoles, which become smaller with age and may gradually disappear, are at times so large that they displace the nucleus to one side. This feature may cause confusion in differentiating these cysts from the binucleate, vacuolate cysts of *E. coli* and *Blastocystis hominis*, a yeastlike organism quite commonly found in feces, the differential characteristics of which are described on pages 23 and 24. The description of the trophozoite of *I. bütschlii* given in column 3 of Table I should be compared with the description of the stained organisms in column 3 of Tables II and III and with the figures in column 3 of Figure 2.

**Endolimax nana** (Wenyon and O’Connor, 1917) Brug, 1918

*Endolimax nana* is a small, nonpathogenic ameba which inhabits the lumen of the colon. In many surveys it appears to be a more common amebic infection of man than *E. coli* is. Its principal importance is due to the fact that it may be confused with small-sized *E. histolytica* trophozoites, measuring from 6 to 12 μ in diameter. The life cycle is similar to that of *E. histolytica* and of *E. coli*. Differentiation from other amebae may be made on the basis of the following characteristics:

1. *E. nana* is a slowly moving ameba showing changes in shape rather than in position.

2. Most of the chromatin of the nucleus is contracted into an irregularly shaped karyosome which is most often centrally located.

3. The thin layer of chromatin against the inner surface of the nuclear membrane described by Stabler (1932) is usually difficult to see without resort to special staining.
4. Cysts are usually oval or irregular in shape; when mature they contain four small nuclei similar to those of the trophozoite.

5. The cyst rarely contains chromatoid bodies and usually presents a strikingly clear appearance. It stains lightly and characteristically with iodine.

Other important characteristic and differential points will be found in column 4 of Tables I, II, and III and column 4, Figure 2.

**Dientamoeba fragilis (Jepps and Dobell, 1918)**

*Dientamoeba fragilis* was formerly thought to be the rarest intestinal ameba occurring in man. However, it was later found to be present more frequently, especially in temperate climates.

*D. fragilis* ranges from 3 to 12 μ or larger in diameter. The endoplasm is finely granular and the ectoplasm is clearly demarcated. The organism rarely moves; it merely changes shape by throwing out pseudopods which, when fully extended, spread laterally and then retract. The majority of individual organisms have two nuclei, hence the generic name *Dientamoeba*. The uninucleate forms, it is said, are generally outnumbered about four to one by the binucleate ones. The chromatin of the nucleus is arranged somewhat in the shape of a small ring of four to six granules, situated about half way between the central point and the nuclear membrane. Cysts have never been satisfactorily demonstrated in this organism. Further detail may be obtained by comparing the descriptions in column 5 of Tables I, II, and III with the illustrations in column 5, Figure 2.

The specific name “fragilis” was given because of the belief that *D. fragilis* is extremely sensitive to changes in environment. This, however, is probably only partially true, according to the independent observations of Wenrich (1936) and Hakansson (1935). The latter reported that in wet smears *D. fragilis* presents three extraordinarily distinctive features, two of which are displayed in normal saline and one in aqueous smear preparations. In the normal saline smear, the characteristic features are the faultless circular outline when the organism is at rest, and the
filmlike pseudopodia with sharp points when it is active. In the aqueous smear an explosive rupture of the ectoplasm with complete evacuation of the endoplasm occurs, followed by restoration of the spherical ectoplasmic shell.

Hakansson (1935) points out that changes in the suspending medium affecting osmotic pressure within the cell apparently account for the latter phenomenon. He states further that, in contrast, the other intestinal amebae of man and Blastocystis hominis tend to manifest in the aqueous smear progressively, distention, increased visibility of nuclei, and disintegration. Morphological manifestations which may occur in the aqueous smear have been summarized by Hakansson as follows:

(a) Distensibility of the ectoplasm of *E. histolytica*
(b) relative fragility of the ectoplasm of *E. coli* and *E. nana*
(c) resiliency of the ectoplasm of *D. fragilis*
(d) remarkable strength of the nuclear membrane of *E. histolytica* and *E. coli*

In *B. hominis*, distention is rapid, becoming complete in from one minute to one hour. Frequently it is completed by the time the preparation is examined. In aqueous smear, *E. nana* and *D. fragilis* may at some stages of distention closely resemble, superficially, *B. hominis*, but the endoplasm is granular, whereas the central vacuole of *B. hominis* has a clear or ground-glass appearance. The ruptured capsule of *D. fragilis*, although it may resemble a distended blastocyst, nearly always contains suspended granules in brownian movement.

The typical *D. fragilis* stool is usually mushy in consistency and often contains the organism in abundance. According to Hakansson (1935) the trophozoite can be readily identified in fresh smear preparations, particularly in the aqueous smears, and still more certainly in wet-fixed, hematoxylin-stained preparations. The nuclear characteristics already described are very distinctive in smears so stained.

**Coprozoic or Free-Living Amebae**

Coprozoic amebae may be found in the feces and other body excretions or secretions after these have been passed. They may
easily be confused with amebae which inhabit the gastro-intestinal tract of man. The presence of at least one “contractile vacuole” (as in *Amoeba proteus*, Fig. 2) characterizes these free-living amebae. Such a structure is never present in the amebae normally infecting man. Common genera are *Dimastigamoeba* and *Hartmanella*, the former assuming a biflagellate state under certain conditions. *D. gruberi* is one of the most prevalent small free-living amebae; it occurs naturally in soil and water.

**Differential Diagnosis**

In the differential diagnosis of human intestinal amebae, the probable presence of *B. hominis*—a vegetable, yeastlike organism—should always be kept in mind. Common forms of this very frequent inhabitant of the intestine of man and other animals are shown in Figure 4, stained with hematoxylin. There are many variations other than those shown in the illustration, and at times

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**Figure 4.** *Blastocystis hominis* from human feces fixed in Schaudinn’s fluid and stained with iron hematoxylin, X 2000. In addition to the nuclei, the thin layer of cytoplasm surrounding the large central vacuole contains dark-staining granules of volutin. 1–7, Ordinary forms; 8, dividing form. (After Wenyon. Protozoology, 1926. Courtesy of Baillière, Tindall & Cox.)
most bizarre forms are seen. This organism may often simulate
the cysts of practically all intestinal protozoa. Familiarity with the
various forms of B. hominis is invaluable in making the differentia-
tion, which is particularly important since proper treatment
of the patient depends on a correct laboratory diagnosis. Lacking
this familiarity, a knowledge of the general appearance and mor-
phology of these organisms such as I have endeavored to present
briefly, should enable the student to acquire gradually some de-
gree of competence in differentiating the most important forms.
From a purely clinical standpoint, the ability to differentiate the
forms of E. histolytica from other organisms is the most impor-
tant consideration.

In Appendix III may be found a discussion of the common
constituents of stools, other than protozoa, and worm parasites
and their eggs. The data in this appendix should be of value to
the student while he is pursuing the subject of parasitology in his
medical course and it may prove useful in his subsequent career.

Culture of the stool as a practical means of differentiating
E. histolytica from the other intestinal amebae, is of considerable
value, but it should be relied upon only as an accessory to direct
microscopic examination of the organism in saline and iodine
preparations and in hematoxylin stained stool smears. At times,
culture may reveal the presence of E. histolytica in stools in
which direct microscopic examination failed to show this organ-
ism; on the other hand, it may not be found in culture, even
when known to be present in the stool. The frequent presence of
B. hominis complicates somewhat the successful use of culture in
differential diagnosis of E. histolytica. Also it should be borne in
mind that occasionally there is a growth in culture of E. coli that
manifests unusual activity. Among the other intestinal amebae,
D. fragilis often grows well in culture for a short time. Methods
suitable for culturing amebae and other intestinal protozoa are
described in Appendix V.
CHAPTER II

AMEBIASIS

Amebiasis and amebic dysentery are clinical terms which have been rather loosely employed to designate the same clinical condition, namely, dysentery caused by the protozoan parasite, *Entamoeba histolytica*. By "amebiasis" is meant infection by *E. histolytica* of the colon and, at times, of the lower ileum and other tissues, particularly the liver. The term "amebic dysentery" applies to a single manifestation of amebiasis that is characterized by a bloody mucoid diarrhea which is traceable primarily to the presence of the vegetating trophozoites in the wall of the colon, extending sometimes into the ileum of man and other susceptible animals.

Recognition of the fact that amebic dysentery comprises but a part of the picture of amebiasis is essential to a proper understanding of infection by *E. histolytica*. The name "tropical dysentery" has been similarly applied to amebiasis, thus limiting not only the pathogenesis and symptomatology of the disease to a specific diarrhea, but also creating the impression that it is confined to warm climates. Actually neither amebiasis nor amebic dysentery is exclusively tropical, although in warm countries the relative number of cases in which the symptoms of dysentery appear, is likely to be greater in proportion to the number of individuals harboring *E. histolytica* in the intestine than it is in temperate climates. This may be due in part to the influence of certain accessory factors which are afforded greater opportunity for effective play in warm climates. These factors will be discussed under Pathogenesis.
E. histolytica cysts as passed in the feces are moderately resistant to mild desiccation and ordinary disinfectants. They are believed also to be immune to chlorination in the concentrations allowable in the treatment of water supplies. In the feces, E. histolytica cysts will probably live not longer than from four to ten days. If the cysts are washed free of fecal matter or diluted with water, as in sewage, and the temperature does not exceed 20° C., they may remain viable for considerable periods. Heating to 50° C. for five minutes will kill them.

Since the cyst is usually the only form in which E. histolytica remains viable after passing through the normal acid stomach, fecal pollution of food and water is significant epidemiologically. Such contamination of food or water or of the mouth directly through the medium of accessory utensils infected from feces-soiled hands, constitute common ways in which amebic infection may originate in so far as the etiologic agent is concerned. However, as already stated, there are certain other factors which appear, according to recent experimental evidence, to play a very important determinant role in tissue invasion by E. histolytica. Such factors have a significant implication in a correct understanding of the epidemiology of amebiasis and amebic dysentery.

In addition to the means of carriage of cysts of E. histolytica already enumerated, flies and cockroaches are an important secondary agency of transmission. They may ingest feces containing viable cysts and carry them in their vomitus or excrement or they may serve as simple carriers of cyst-laden feces adhering to their bodies.

Pathogenesis

After being ingested, E. histolytica cysts pass through the stomach and intestines until they reach the ileum or the colon. There, excystation takes place, a 4-nucleate ameba emerging from each mature cyst. Then, by nuclear and cytoplasmic divisions, 8-meta-cystic amebulae are formed. These may invade the intestinal wall under favorable conditions (Fig. 5). It was formerly thought that
Fig. 5. Pathogenesis of amebiasis in colon (at various magnifications).
the amebae centered their attack, so to speak, on the tubular glands of the large intestine (crypts of Lieberkühn). Here, it was explained, they propagate, and destroy the gland cells, thus plugging the exit into the lumen of the colon. With the evacuation of these “pinpoint” abscesses containing bacteria as well as amebae, it was conjectured, amebae continue to digest away the surface cells and undermine the mucosa.

While the crypts of the Lieberkühn undoubtedly are involved in the initial phases of tissue invasion by *E. histolytica*, it seems much more probable that the main points of attack are the mucosal epithelial areas between the glands. Critical histopathologic studies of the earliest detectable lesions in amebiasis have shown, according to James and Getz (1928), that “the presence of *E. histolytica* in the tissues is associated with a lysis or disintegration of their cells in the beginning not unlike that seen in other pathological conditions.” In fact, it appears, according to these and other investigators, that the very earliest lesion is characterized by some dissolution and destruction of the mucosa before any appreciable degree of invasions has begun—an early breaking down out of all proportion to the number of amebae present. Experimental work by Nauss and Rappaport (1940), and French and German workers contemporaneously, supplies abundant evidence in support of the belief that suitable toxic traumatizing agents play an important role in the pathogenesis of the early amebic lesion.

For sixty years there has been much controversy as to how and under what circumstances and conditions *E. histolytica* breaks through the usually formidable initial tissue barrier offered by the colonic mucosa. With a point of entry available, the evolution of the pathologic process involves a lysis of tissue cells, with undermining of the mucosa, invasion of lymph spaces, dissection of connective tissue planes, and thrombosis of nearby capillaries. The spreading necrotic area thus formed soon sloughs, leaving a small ulcer with undermined edges. Amebae on the surface of this ulcer are swept out by fecal movements into the lumen of the colon whence they may invade fresh tissue, become encysted, or
AMEBIASIS

be passed out as trophozoites without becoming encysted.

If the host's resistance is low, new ulcers are likely to develop in increasing numbers and the individual will become aware of gaseous distention and vague abdominal discomfort, followed by symptoms of dysentery. On the other hand, if the host's resistance to invasion is good, only a limited number of ulcers will be formed and a chronic course usually ensues, known as the "carrier state." However, with the lowering of the carrier's resistance, this equilibrium may be disturbed and acute symptoms of dysentery develop.

The muscularis mucosae usually constitutes a formidable barrier to further penetration of the intestinal wall by *E. histolytica*. In severe infections when the resistance of the host dwindles sufficiently, amebae may pass through this barrier into the submucosa and deeper muscular layers. Opinions differ as to the mechanism of penetration of the submucosa; possibly the path of invasion is through the lymph channels. Liver abscess occurs comparatively rarely—usually in untreated cases of *E. histolytica* infection.

Nauss and Rappaport (1940) have shown that mucosal traumas play an important role in the successful experimental inoculation of kittens and cats with *E. histolytica* cultures. Bacteria associated with intestinal proteolytic floras appear to be effective accessories in determining tissue invasion by cultures of *E. histolytica* of very low virulence. Chemical trauma of the type that may be induced by Croton oil exerts a similar favorable influence. In fact, such trauma, produced in a special manner, enabled us to infect adult cats with ease. Recent independent work by Deschien (1938) and Westphal (1937) has substantiated our results concerning the accessory action of both bacterial and chemical traumatizing agents. I feel, therefore, that specific damage to the colonic mucosa may play a very important part in determining the occurrence of tissue invasion by apparently lumen-dwelling *E. histolytica* in man as well as in cats of various ages and weights.

The traumas produced experimentally by some bacterial and chemical agents are highly significant in relation to the wide distribution of the cyst-carrier state, and the relative paucity of clin-
ical cases of amebic dysentery, particularly in the temperate zone. Frequently the insidious onset and chronicity of amebiasis tend to obscure its true nature. Craig (1934) states that “E. histolytica is responsible not only for amebic dysentery but also for a host of other symptoms referred to the intestinal tract.” Although it is not definitely known that the proportion of frank cases of amebic dysentery to cyst-carrier prevalence in warm countries exceeds that in more temperate zones, this seems to be the case. At least, gastro-intestinal upsets which might tend to favor tissue invasion by E. histolytica are certainly more frequent in warm climates than in temperate regions, according to many observers. One may not, therefore, justifiably minimize the potential hazard to physical well-being presented by E. histolytica cysts in the colon of so-called “healthy carriers.”

Repeated careful microscopic stool examination for E. histolytica may reveal the role which this organism plays in the etiology of a multitude of obscure gastro-intestinal symptom-producing conditions. Unfortunately such stool examinations by adequately trained observers are almost the last diagnostic aid summoned by the internist or surgeon. Even when an examination is requested, a single negative report is often considered adequate in evaluating parasitic factors in the etiology and pathogenesis of disease. At least three microscopic examinations are necessary to insure a positive finding in 90 per cent of cases of amebiasis, about 90 per cent or more being missed during the first examination.

Symptomatology, Complications, and Sequelae

Clinically, amebiasis is essentially a chronic disease, periods of improvement alternating with recurrences of pain, diarrheic or dysenteric stools, and related symptoms. The onset is usually insidious and the patient is much more likely to complain of diarrheic than of dysenteric symptoms. A history of passing three or four pultaceous stools daily is frequently given. The patient often complains of tenderness in the region of the cecum or along the course of the large intestine. In thin persons thickening of the colon is sometimes noted. According to the experiments of
AMEBIASIS

Walker and Sellards (1913) on human volunteers in the Philippines, the incubation period ordinarily varies from one to three months. The fact that a significant number of persons with apparently average normal large intestines harbor the cysts of E. histolytica suggests that the development of clinical disease is brought about by the co-operative action of supplementary factors which determine or open the way for active tissue invasion.

According to Manson-Bahr (1940), the symptoms of amebic dysentery, both subjective and objective, are similar to those of bacillary dysentery. In amebic dysentery, however, abdominal tenderness is much less acute and is usually confined to localized areas such as the cecum, the transverse colon, the sigmoid flexure, or the rectum. Ulceration in these areas may give rise to signs and symptoms suggesting appendicitis, various forms of colitis and symptoms of ulceration due to other causes. The stools often number no more than three or four in twenty-four hours (seldom more than twelve) and are larger than those of bacillary dysentery. They generally contain considerable dark altered blood which gives them a fetid odor. Their resemblance to “anchovy sauce” in appearance and consistency has been noted. When mucus is passed, it usually occurs in blobs and flecks disseminated through the fecal mass, and is frequently also streaked with blood. Gangrenous sloughs may be passed. Formed stools are the exception. During exacerbations, grayish green or grayish brown mucoid masses of various sizes should be sought for and examined microscopically.

The symptoms of amebiasis vary so widely that the disease may simulate almost any intestinal disorder. It is not always associated with diarrhea or dysentery; it may be marked by constipation and associated symptoms. The pathologic changes resulting from the ulceration may lead to various types of dilatation on the one hand, or to hypertrophic changes on the other. The resultant signs and symptoms frequently challenge the differential diagnostic acumen of experienced clinicians. Very often the cecum particularly is affected and it may become grossly distended with gas, causing much discomfort. Sometimes a result-
ing amebic granulomatous condition of the rectum may simulate carcinoma or tuberculosis.

Manson-Bahr (1940) has summed up the clinical history of the average case of amebic infection: "Often, without treatment, the condition may subside, and the patient may be apparently cured, only to relapse after an interval of weeks, months, or it may be of several years or even longer. More often the patient continues to pass loose, semiformalized stools, attacks of diarrhea alternating with constipation. After any physical exhaustion, chill, alcoholic or dietetic indiscretion, a fresh exacerbation may supervene. On account of the variable nature of the symptoms, the shifting character of the pain, and the occasional appearance of melanic stools, it is clear that the condition has to be differentiated from duodenal ulcer, gall-bladder disease, pancreatitis, and intestinal neoplasm."

Acute hepatitis may appear at any time during the course of amebic infection. The patient usually complains of severe pain over the hepatic area, together with symptoms of toxemia and considerable fever. The liver is enlarged, the lower border extending below the costal arch to varying degrees. Pain referred to the right shoulder is frequently a characteristic symptom. The associated leucocytosis may show a white blood cell count of 20,000 or even higher. This condition sometimes subsides without treatment.

Among the graver complications of amebiasis, abscess of the liver is by far the most common and the most important. Statistics regarding its incidence vary rather widely. The greatest number of reported cases have been associated with frank amebic dysentery. The percentage, if based on the total amount of amebic infection, may not be relatively high, but this complication has not infrequently been found when least expected. The diagnosis of the presuppurative stage of amebic abscess of the liver is most difficult. If *E. histolytica* is found in the feces, a diagnosis of amebic hepatitis is justified when the patient has a daily intermittent or remittent fever of low degree, pain or tenderness over the hepatic area, and mild leucocytosis. The physical signs de-
pend largely on the duration of the disease. Enlargement of the liver is most frequently upward, the dullness extending over a dome-shaped area with the highest portion situated in the region of the right lobe (Fig. 6). Edema or bulging may be noted in this area. If the abscess is in the left lobe, there may be bulging in the epigastric region. Tenderness on deep pressure is a constant sign and the upper portion of the right rectus muscle may be rigid. Multiple abscesses not infrequently cause downward enlargement.

The lung and the brain may present conditions favoring local development of amebic abscesses. In most cases of pulmonary abscess, infection usually results from the rupture of an abscess of the liver into the thorax through the diaphragm. The symptoms produced in such secondary amebic abscess of the lung are similar to those of primary abscess of the lung; symptoms resulting from primary invasion of the lung, a much rarer condition, are very similar to those of pulmonary tuberculosis. Amebic abscess of the brain is usually secondary to amebic abscess of the liver or the lung; fifty or more cases have been reported in the literature.

*E. histolytica* infection of the skin sometimes occurs, producing extensive destruction and sloughing. The invasion may follow surgical drainage of a liver abscess, the appendix, or other area of the lower portion of the intestine. Extension from lesions in the rectum to the tissues about the anus, and direct parasitic invasion of the skin have also been reported.

Amebic appendicitis is by no means an uncommon complication of amebic infection of the colon. Many of the fatal cases of amebiasis originating in Chicago during the 1933 epidemic as reported by the National Institute of Health (1936) had been operated on for what appeared to be acute or chronic appendicitis. The symptoms generally resemble the chronic rather than the acute stage of appendicitis. Perforation of the colon and local peritonitis are possible complications if the ulcers are deep seated.

The sequela of amebiasis are associated more or less directly with the chronic phases of this condition. Patients who have suffered recurrent attacks of amebic dysentery frequently develop
FIG. 6. Results of treatment of case of long-standing chronic amebiasis as indicated by series of x-ray films. (Courtesy, Dr. Douglas Palmer and New York Hospital.)

A, X-ray film (1932) showing lesion of cecum interpreted as healed tuberculosis. X-ray film of liver, also taken in 1932, showed some elevation of right diaphragm. B, X-ray film of liver, taken in 1935, shows high right diaphragm, patient complaining of acute abdominal symptoms with cysts of Endamoeba histolytica in feces.
Fig. 6. 

C. X-ray film of cecum, taken after active intensive treatment, shows marked improvement of the lesion over that indicated by film A and another taken in 1934. (The x-ray film of the liver just prior to this time showed a high right diaphragm still present, and in addition an adjacent pleural abscess.)

D. X-ray film of liver, taken in 1937, shows marked recession of abnormally high upper border of liver as revealed in B, prior to beginning anti-amebic treatment.
a condition of chronic diarrhea resulting from the extensive de-
struction of mucosa of the large intestine and its replacement
by fibrous tissue. Those who suffer less severely from these after-
effects of chronic amebiasis are very apt to experience diarrheal
attacks during the warmer months of the year when large amounts
of fluid are taken. During the colder months, on the other hand,
the bowel movements may be almost normal. Various degrees of
invalidism result from this tendency to diarrheal attacks, de-
pending largely upon the actual amount of permanent damage to
the bowel wall. In some individuals a normally formed stool seldom,
if ever, occurs. If repeated stool examinations for amebae are
negative, the attacks of diarrhea may be attributed to the func-
tional inadequacy of an impaired intestinal mucosa. It is im-
portant to differentiate clearly nonspecific colitis from the colitis
caused by amebic infection, since the correct diagnosis may spare
the patient much inconvenience and probable harm from the
useless administration of amebicide, and the practitioner the
ultimate realization of failure from drugging for a condition not
amenable to such a method of attack.

Sprue or "psilosis" is a chronic diarrheal condition which was
formerly believed by many physicians to be a sequel of amebic
dysentery. The symptoms of sprue and of chronic amebic dysentery
are frequently very similar. In amebic dysentery, as in sprue,
atrophy of the mucous membrane of the large intestine occurs,
followed by the formation of scar tissues with diarrhea in which
the stools are always semifluid or fluid in consistency, light yellow
in color, and frothy from the presence of much gas. It has been
noted that some patients with amebiasis develop the sore tongue
and throat which are striking features of sprue.

Finally, adhesions and contractures of the intestines resulting
from localized peritonitis, intestinal perforations, and the heal-
ing of large amebic ulcers must not be overlooked. Fortunately,
these contractures usually produce only partial obstruction, caus-
ing obstinate constipation. The amount and extent of the thicken-
ing of the coats of the colon in some of these cases, as revealed
at autopsy, is astonishing.
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The occurrence of \textit{E. histolytica} in the glands in Hodgkins' disease and in the bone marrow in chronic arthritis, as reported by Kofoid and others (1922), appears to be doubtful. In fact, satisfactory unbiased confirmation of their findings has not thus far appeared in the literature.

\textbf{Diagnosis}

The diagnosis of amebiasis rests primarily upon the demonstration of \textit{E. histolytica} in the feces, exudate, or tissues of the patient. Although the clinical symptoms may appear to justify a diagnosis of amebic diarrhea or dysentery, confirmation should be obtained by the demonstration of \textit{E. histolytica} in the stools. It is practically impossible to make a clinical diagnosis in the case of carriers without symptoms.

Differentiation must be made from many conditions in which blood and mucus are passed in the stools, such as other forms of diarrhea and dysentery, colitis, tuberculosis, and neoplasms of the bowel. Numerous observers have described a hypertrophic form of amebiasis which may affect different parts of the colon. Growths up to 10 cm. in diameter, or larger, have been reported in this condition; they may closely resemble carcinoma and tubercular granuloma of the colon. These processes, according to the opinion of Manson-Bahr (1940'), develop from isolated ulcers associated with progressive erosion of the bowel wall in response to long continued secondary infection. Large amounts of edematous granulation tissue appear, sometimes involving the entire bowel wall and the neighboring mesocolic fat.

\textbf{Differential Diagnosis}

Bacillary dysentery is commonly confused with amebic dysentery. Early attempts to distinguish between these two conditions clinically led to many erroneous ideas regarding their principal characteristics. The chief differences are briefly contrasted in Table IV.

Other much less frequent parasitic causes of dysentery are the ciliate, \textit{Balantidium coli}, and the eggs of \textit{Schistosoma} (blood...
TABLE IV
DIAGNOSTIC DIFFERENCES BETWEEN AMEBIC AND BACILLARY DYSENTERY
(From C. F. Craig, 1934)

<table>
<thead>
<tr>
<th></th>
<th>Amebic Dyentery</th>
<th>Bacillary Dyentery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Usually a chronic endemic disease</td>
<td>Usually an acute epidemic disease.</td>
</tr>
<tr>
<td></td>
<td>(May occur in epidemics)</td>
<td>(May be endemic)</td>
</tr>
<tr>
<td>Incubation period</td>
<td>uncertain. From a few days to months or years</td>
<td>Incubation period a week or less</td>
</tr>
<tr>
<td>Onset</td>
<td>slow and insidious but may be acute. History of poor</td>
<td>Onset acute. Good health previously</td>
</tr>
<tr>
<td></td>
<td>health previously</td>
<td></td>
</tr>
<tr>
<td>Course</td>
<td>usually chronic with acute exacerbations</td>
<td>Course usually a few days but may be chronic</td>
</tr>
<tr>
<td>Liver abscess</td>
<td>a complication</td>
<td>Liver abscess does not occur</td>
</tr>
<tr>
<td>Physical signs</td>
<td>local thickening and tenderness over ascending, transverse colon, cecum, or sigmoid flexure</td>
<td>Arthritis a frequent complication. General abdominal tenderness without thickening of gut</td>
</tr>
<tr>
<td>Tenesmus</td>
<td>usually moderate in character</td>
<td>Tenesmus usually very severe</td>
</tr>
<tr>
<td>Death</td>
<td>due to exhaustion, liver abscess, intestinal hemorrhage, or peritonitis due to perforation of amebic ulcer</td>
<td>Death due to toxemia and exhaustion</td>
</tr>
</tbody>
</table>

In addition to suggestive laboratory findings, the previous residential history of the patient is of the greatest importance in the diagnosis of conditions due to these parasites. This point is frequently ignored or overlooked by physicians in temperate climates. Symptoms produced by the presence of intestinal worms, coccidiosis, kala-azar, malarial dysentery, syphilis, tuberculosis, and hemorrhoids may be confused clinically with those of amebiasis. Differentiation of these conditions need not be detailed here.

Finally, the more common chronic intestinal conditions known as mucous colitis, chronic ulcerative colitis, and nonspecific colitis are possible sources of error or confusion in the diagnosis of amebiasis. When one of these conditions coexists with amebic infection, treatment of the latter almost always results in improvement of the associated intestinal condition. Therefore careful, reliable microscopic examination of fresh stool specimens is important in all such cases.
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The value of sigmoidoscopic examination should be emphasized as an important aid in the diagnosis of the various disease conditions of the colon. Usually in amebiasis the ulcers are small, yellow, and surrounded by distinct hyperemic areas. Scrapings from these ulcers frequently contain actively motile forms of *E. histolytica*, when the fecal specimens are reported negative for amebae. In chronic bacillary disease, sigmoidoscopic examination causes considerable pain; in uncomplicated amebic ulceration the patient complains of only moderate discomfort.

Differentially the exudates in amebic and bacillary dysentery are briefly presented in Table V. In Figures 7 and 8 are shown the microscopical appearances of these exudates. These illustrations supplement the tabulated data contrasting these two characteristic types of dysentery exudates.

Fig. 7. Cell picture in the feces of acute amebic dysentery, × 1100 app. (After Thomson and Robertson. Protozoology, 1929. Courtesy of Bailliére, Tindall & Cox.)
Culture of stools and dysentery exudates for amebae affords a very useful adjunct in the diagnosis of amebiasis, but it must not be relied upon to the exclusion of the various methods of direct microscopic examination described in Chapter I. It serves, however, to confirm other findings and increases the chances of finding the parasite if only small numbers are present. Suitable methods for culturing amebae are given in Appendix V.

The complement fixation test developed by Craig (1934) and employed by the Army Medical School at Walter Reed Hospital in Washington has been reported to give positive results in 90 percent of the cases. Others have reported less satisfactory results. This test may be used in the diagnosis of atypical cases, carriers, amebic abscess, and in the control of treatment. The reaction
### Table V

**DIFFERENTIAL FEATURES OF EXUDATE IN AMEBIC AND BACILLARY DYSENTERY**

(Adapted from Haugwout and Callender)

<table>
<thead>
<tr>
<th>Exudate</th>
<th>Bacillary Dysentery</th>
<th>Amebic Dysentery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount (definite hemorrhage excluded)</td>
<td>Frequently massive, making up a large part of stool</td>
<td>Usually comparatively small</td>
</tr>
<tr>
<td>Character: Acute stage</td>
<td>Glary white or yellowish white mucus flecked or streaked with blood little altered in color</td>
<td>Stool often entirely fluid with little fecal matter, containing flecks, streaks, or blobs of mucus (sago grains), frequently grayish green to reddish brown</td>
</tr>
<tr>
<td>Subacute or chronic stage</td>
<td>Brown, liquid stools resembling those resulting from saline purge, mucus being much less (relatively) in quantity</td>
<td>Stools pultaceous from time to time, containing grayish green or brown mucoid masses of various sizes</td>
</tr>
<tr>
<td>Blood</td>
<td>Varying amounts</td>
<td>Small quantity to actual hemorrhage</td>
</tr>
<tr>
<td>Cell content:</td>
<td>Low</td>
<td>Usually plentiful</td>
</tr>
<tr>
<td>Bacteria</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>Often 90 per cent of exudate. Many show degenerative nuclear changes (ringing) and cytoplasm frequently contains fat</td>
<td>Few generally. Cytoplasm of some show degenerative changes and the nuclei may appear “pycnotic”</td>
</tr>
<tr>
<td>Polymorphonuclears</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endothelial macrophages</td>
<td>Present in variable numbers; frequently phagocytic, containing erythrocytes and leukocytes. Undergo toxic degeneration becoming so-called “ghosts cells”</td>
<td>Rarely seen except in cases with coexisting bacterial dysentery</td>
</tr>
<tr>
<td>Plasma cells</td>
<td>Present, but relatively more abundant early</td>
<td>Present in small numbers</td>
</tr>
<tr>
<td>Pycnotic bodies</td>
<td>Occur, but are comparatively insignificant</td>
<td>May constitute as much as 80 per cent of cellular elements</td>
</tr>
<tr>
<td>Amebae (trophozoite)</td>
<td>E. histolytica absent unless both diseases are present. Other amebae may be present</td>
<td>Present and must be found to complete diagnosis</td>
</tr>
</tbody>
</table>
becomes negative after the elimination of infection. However, this test, as well as the culture method, should be employed only as additional and confirmatory means of diagnosis rather than as a substitute for direct microscopic examination.

**TREATMENT**

Drug therapy in amebiasis should be determined largely by the clinical status and the history of the individual patient. When diarrhea is prominent, or dysenteric symptoms are present, they may be controlled by injections of emetine hydrochloride; rest in bed is essential until the acute symptoms have subsided. Emetine hydrochloride is usually given in 1 grain (0.065 gm.) doses, once a day, the total amount to be given in one course of treatment not to exceed 10 or 12 grains, because of its high cumulative toxicity. Symptoms which may be attributed to the effect of emetine itself are severe diarrhea, muscular weakness, neuritis, myocarditis, and cardiac paralysis from degeneration of heart muscle.

The practical usefulness of emetine ceases with the disappearance of the acute bowel symptoms, since this drug is rarely curative alone. It should be followed by amebicidal drugs such as the halogen quinoline derivatives—chiniofon, anayodin, yatren, and vioform, or carbarsone (an arsenical). The maximum dose of chiniofon, anayodin, and yatren is 3 or 4 enteric-coated 4 grain (0.25 gm.) pills or tablets daily, in divided doses, for ten days. Vioform and carbarsone are also given in single doses of 4 grains (0.25 gm.), in hard gelatine capsules, the former being given thrice and the latter twice daily for a period of ten days. All these drugs, while somewhat toxic, are considered comparatively safe when administered according to directions.

Another very useful and well tried amebicidal drug is emetine-bismuth iodide (E.B.I.), the maximum dose of which is 3 grains daily, given in one dose for from ten to twelve consecutive days. However, Manson-Bahr (1940*), who has had extensive experience with the use of this drug, prefers to give it in daily doses not exceeding 2 grains, combined with daily use of yatren in rectal
enema. These two amebicidal agents administered according to his scheme can be highly recommended.

To check the effectiveness of each course of treatment a microscopic stool examination should be made shortly after completion of treatment and subsequently at intervals. Craig (1934) recommends the examination of at least three stool specimens, collected at intervals of one week (one of which should be secured after a saline laxative), and thereafter once a month for at least three months. In the case of carriers, the intervals between collections may be shortened. To be permitted to serve as food-handlers, all infected persons should have, in addition to three successive negative stool examinations, at least three negative reports on stool specimens which are collected at monthly intervals. Regarding amebic therapy in general, Craig (1934) aptly remarked that "no method of treatment or drug will cure every infection with this parasite and it is a great mistake (as has often been the case in the past) to adhere to any one drug or method where it has proven a failure after a careful trial." In such instances it is much better to employ some other approved remedy than to subject the patient to the continued administration of a drug which is ineffective and may become toxic.

If amebic hepatitis or amebic abscess of the liver is detected early enough, treatment consists in the use of emetine hydrochloride injections (1 grain a day) for from ten to twelve days in succession. If this procedure fails to arrest the symptoms, surgical intervention is necessary, but operation should not be attempted until the beneficial effects of emetine have been faithfully exploited. This applies also to amebic abscesses in other organs. Such conservatism is well illustrated in Figure 6 with reference to amebic infection of the colon as well as the liver and lungs.

Prognosis

When cases of acute amebiasis are recognized early, and prompt and adequate treatment is instituted, the prognosis is very good. However, the longer the infection has existed, the less favorable is likely to be the progress toward cure. Delay in treatment with
recurrences tends greatly to diminish a favorable prognostic out­look. Death seldom occurs during an initial attack except in ful­minating cases. In amebic hepatitis and amebic abscess of the liver and the lungs, prognosis is often good; but in amebic abscess of the brain the extent of recovery, if it occurs at all, will depend on the centers and tracts destroyed.

**Prophylaxis**

Since the infective cysts of *E. histolytica* are transported by human feces from points of origin in the colon and sometimes the lower ileum, the control of amebiasis and the prevention of its spread must be based primarily on the personal and community aspects of the problem of disposal of human feces. As reported by Craig (1934) the general recommendations drawn up by a special committee appointed by the Chicago Board of Health and adopted in 1933 cover the essential prophylactic procedures:

1. Cases of amebiasis (including amebic dysentery) should be promptly reported as such to the lawfully constituted health authorities of the community.
2. Carriers not engaged in food handling should be treated but not required to vacate their occupations.
3. The feces of untreated and insufficiently treated carriers and sometimes those of active cases contain the infective (cyst) forms of the parasite. Therefore, the stools of such persons should be disposed of according to good, modern sanitary regulations. Those infected should be informed of the manner of transmission of the disease, treated, and cautioned to
   1. Wash their hands thoroughly after using the toilet.
   2. Avoid depositing their feces where it may be exposed to flies and other insects, or where it may contaminate food, water or articles handled by other persons.
   3. Avoid preparing and handling food to be eaten by other persons until considered incapable of spreading the disease. Laboratory examination of the feces of food handlers should be required if circumstances point to a particular individual or group of individuals as a possible source of infection. The general examination of all food handlers for *E. histolytica* is considered impractical.
4. A patient or carrier should not be allowed to become a food handler unless, after at least seven days since completion of treatment,
three successive negative stool examinations at suitable intervals are obtained. In addition thereafter, in order to be allowed to continue as a food handler, such a person should have at least four negative stool examinations at intervals of one month.

The community aspects of this problem depend essentially upon prevention of contamination of food and drink by fecal-borne *E. histolytica* cysts. In summary, the important points in this connection are as follows:

(a) Proper sanitary disposal of sewage
(b) Prohibition of use of human excrement for fertilizing truck gardens
(c) The prevention of fly-breeding and protection of food from flies, cockroaches, etc.
(d) A sanitary water supply, preferably filtered, chlorination alone being insufficient
(e) Sanitary plumbing, particularly of hotels, institutions, and public buildings
(f) Examination and treatment of food-handlers in public eating places.
CHAPTER III

FLAGELLATA (CLASS MASTIGOPHORA)

Flagellata are characterized during the vegetative stage by the possession of flagella. These hairlike processes greatly facilitate locomotion in liquid media. For practical purposes they may be considered in groups according to the type of tissue involved.

FLAGELLATES OF MUCOUS SURFACES

The Trichomonads

The three trichomonads of man, although generally considered to be distinctive species, will be discussed here as closely similar organisms which have adapted themselves to different environmental conditions. From the sites of occurrence, they are referred to as Trichomonas buccalis* (Goodey, 1917) Kofoid, 1920, Trichomonas intestinalis (Leuchart, 1879), and Trichomonas vaginalis (Donne, 1837). Although heretofore these organisms have been believed to be harmless commensals, recent studies of T. vaginalis seem to indicate that this may not always be true. They appear to feed freely on bacteria and cellular debris, being found abundantly in conditions associated with excessive mucous secretion and the presence of large numbers of white blood cells. In general, however, there is little evidence to indicate that they are able, alone, to penetrate normal mucosa.

Formerly it was thought that T. buccalis is related to T. vaginalis, possibly being a derivative of it. On the other hand, some workers believed that T. vaginalis and T. intestinalis are related. There appears, however, to be little evidence pointing to any direct relationship between T. buccalis and T. intestinalis.

*Dobell (1934) has called attention to the priority of tenax as the specific name of this trichomonad living in the mouth, the revised name being Trichomonas tenax (Müller, 1786).
Lynch (1922), Wenyon (1926), and Dobell (1934) were of the opinion that all three organisms are the same under similar conditions, as in culture; but Kessel (1939) suggested that *T. intestinalis* and *T. vaginalis* are probably physiologically different under natural conditions. The consensus of present opinion is that these three trichomonads are sufficiently different in both their detailed morphologic and physiologic characteristics to discredit previous ideas or beliefs of a close relationship between any of them.

**Morphology.** These trichomonads (Fig. 9, A, B, C, D, and Table VI) vary in length from about 5 to 20 \( \mu \). Their shape is usually pyriform. The nucleus is located in the anterior end, commonly behind the blepharoplast whence the flagella originate. There are usually four or five flagella; one is directed backward and attached marginally to another external structure, which extends diagonally posteriad for a variable distance and is known as the "undulating membrane." The undulating membrane flagellum of *T. intestinalis* has a free terminal extension, which lashes about, as do the anteriorly directed flagella. The undulating membrane with its attached backwardly directed flagellum waves from side to side. This movement evidently originates in the blepharoplast and terminates with the lashing of the free end of the attached flagellum. Beneath the line of attachment of the undulating membrane to the body is a slender structure known as the "basal fiber."

The axostyle—a clear, elastic, rodlike supporting structure—runs longitudinally through the middle of the organism and often extends posteriorly in the form of a spine. The "cytostome" or mouth is inconspicuous, appearing as an indistinct slit at the anterior end near the origin of the flagella, and opposite the undulating membrane. When the flagellates become sluggish they present false surface pseudopods. The movement of the undulating membrane is best seen at such times.

**Movements.** They have a jerky, nervous type of movement and appear to be constantly darting aimlessly about. The body rotates rapidly, and under the oil-immersion objective with subdued
Fig. 9. Human intestinal flagellates. (Courtesy of D. H. Wenrich.) Large scale divisions = 5 μ; magnification, X 1750 app.

light or darkfield illumination, the undulating membrane is clearly visible. By the use of procedures designed to slow up their movements, the flagella may be counted, if one has the time and patience to do so.

**Multiplication and Nonencystment.** Multiplication is by longitudinal division. The width of the body becomes much greater than the length, the nucleus and blepharoplast divide, the axostyle degenerates, and the undulating membrane of the parent is retained by one daughter, a new membrane growing out for the other daughter. A new axostyle grows out from the blepharoplast of each daughter individual. The flagella are divided between the two daughter organisms. This phenomenon of cleavage, division of organelles, and completion of development proceeds until the process is completed. The posterior extremities adhere for a time before they finally separate and then each swims away independently. Such division may be observed best in coverglass preparations from culture.

A cyst form of these organisms is not definitely known. Although this apparent absence of encystment would seem to make control, prevention, and treatment of the trichomoniasis a comparatively simple problem, this is by no means the case. Once established, infections with the intestinal and vaginal forms are persistent and not readily amenable to drug therapy in concentrations tolerated by the mucosa. Recent studies indicate that transmission of *T. vaginalis* may take place through coitus. It is significant here that the incidence of urethral trichomoniasis among males is high.

**Culture.** All three human trichomonads have been successfully cultured on suitable media. Under such artificial conditions, they vary considerably from the original flagellates, tending toward more rotundity in form and variability in size. The nature of the environment and the character of the medium evidently influence the size and other relatively minor differences between these trichomonads.

**Treatment of Trichomoniasis.** Recent reports on the treatment of trichomonas vaginitis infections offer encouragement.
<table>
<thead>
<tr>
<th></th>
<th>TROPHOZOITES</th>
<th>CYSTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SHAPE</td>
<td>MOTILITY</td>
</tr>
<tr>
<td><em>Helicobacter pylori</em></td>
<td>Pear-shaped, somewhat inflexible and nonplastic</td>
<td>Forward, jerky, rotating spirally at same time</td>
</tr>
<tr>
<td><em>Trichomonas vaginalis</em></td>
<td>Roughly pyriform but quite flexible and plastic</td>
<td>Combination of darting, swimming, revolving, crawling and twisting. Axostyle may be adherent to debris.</td>
</tr>
<tr>
<td><em>Trichomonas buccalis</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Giardia lamblia</em></td>
<td>Partially hollowed out on the flat side at broad part</td>
<td>Vibrates and rolls as it swims and frequently doubles upon itself</td>
</tr>
<tr>
<td>Genus</td>
<td>Description</td>
<td>Length</td>
</tr>
<tr>
<td>---------------</td>
<td>--------------------------------------------------</td>
<td>----------------------</td>
</tr>
<tr>
<td>Relotamonas</td>
<td>Variable with cytoplasm lateral, generally pyriform</td>
<td>4-9 µm by 3-4 µm</td>
</tr>
<tr>
<td>(Embadomonas)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>intestinalis</td>
<td>Jerky, like that of <em>Chilomastix</em></td>
<td></td>
</tr>
<tr>
<td>Enteromonas</td>
<td>Pyriform but very plastic, often flattened on one side</td>
<td>4-10 µm by 5-6 µm</td>
</tr>
<tr>
<td>(Triceromonas)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>intestinalis</td>
<td>Jerky, rotating at same time</td>
<td></td>
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One method, said to be very efficient in eliminating *T. vaginalis*, is application to the vagina by insufflation of a powder containing twelve parts of acetarsone (stovarsol), two parts of salicylic acid, and equal parts of kaolin and sodium bicarbonate sufficient to bring the total amount to one hundred parts of the mixture. The powder is best applied by means of a blowing device (like that used in dusting insect powders upon small animals and plants), after the vagina has been thoroughly cleansed with tincture of green soap diluted with an equal quantity of warm water. This treatment may be given twice a week but its effects should be carefully watched.

According to Buxton and Shelanski (1937), the application of silver picrate by insufflation produces effective results. The preparations employed are a powder, consisting of one part of silver picrate dispersed upon ninety-nine parts of kaolin, and horoglyceride-gelatine suppositories containing 2 grains of silver picrate each. Five grams of the powder are insufflated as described in the preceding paragraph and the patient is given six of the suppositories with instructions to use one each night and report to the physician one week later.

Thus far the results of treatment for intestinal trichomoniasis have been rather unsatisfactory. Infections with *T. buccalis*, on the other hand, appear to clear up promptly with successful treatment of the associated abnormal conditions of the mouth, such as caries, gingivitis, and pyorrhea.

**Prophylaxis.** Control and prevention of these flagellates depend on their location. If the site is the mouth, avoidance of kissing, and proper hygiene of mouth and hands is requisite. Prophylaxis of trichomonas vaginitis, and urethritis in the male as well as in the female, presents a difficult problem. Since the exact mechanism of infection is not known, the only measures to be suggested are general hygienic and sanitary precautions. Measures directed against vaginal irritation and the various bacterial infections of the sexual mechanism undoubtedly constitute fundamental and valuable aids in both prophylaxis and treatment of flagellate infection of the vagina.
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Little can be said regarding the control and prevention of intestinal trichomoniasis since less is known about its mode of transmission than about that of vaginal trichomoniasis. In general, measures similar to those employed against fecal contamination of the hands and of food and water by human ameba-infected material, should prove of value. Finally, prophylactic measures directed against intertransfers of trichomonads from vagina, mouth, and rectum should be adopted on general principles in so far as is practicable.

Giardia lamblia (STILES, 1915)

Giardia lamblia (Fig. 9, E, F, and Table VI) is a fairly common protozoan parasite of human beings, particularly of children. The vegetative forms inhabit the lumen of the upper part of the small intestine and are usually found only in fluid or semifluid stools. The cysts (Fig. 9, G, and Table VI) are found in the lower ileum and the large intestine, and occur principally in formed stools. The outstanding point of difference from other intestinal flagellates—the possession of a double suctorial disk—is of considerable significance. By means of this G. lamblia is able to attach itself firmly to the duodenal mucosa.

Pathogenicity. There has been much controversy regarding the pathogenicity of this flagellate, but in view of the accumulating clinical evidence of the past ten or fifteen years, it seems logical to accept claims of its tendency to produce fairly definite gastrointestinal symptoms, notably in children. The constancy of the association of G. lamblia with a certain type of recurring diarrhea of childhood should not be ignored by the physician in diagnosing such conditions.

Morphology. G. lamblia is distinctly pear-shaped, tapering to a point at the posterior extremity. It measures from 8 to 15 μ or more in length and 5 to 12 μ in width. The anterior two-fifths of the ventral surface is depressed and the major portion of this concavity constitutes the so-called “sucking disk” already referred to. This organism is more highly differentiated than are the trichomonads or chilomastix, being distinctly symmetrical bi-
laterally, and having eight flagella in pairs. There are two oval vesicular nuclei with central karyosomes which may be rod-shaped masses or separate granules. These nuclei lie in the anterior rounded part of the organism, dorsal to the sucking disk and opposite to each other on either side of the midline. Between the two nuclei, and approximately on a level with their anterior borders, are two blepharoplasts, and more laterally, points of origin of the two axostyles which pass directly backward to the tip of the caudal extremity. The four pairs of flagella arise at various points near the midline of the ventral surface; the most anterior cross each other before emerging from the cytoplasm. The other three pairs of flagella also have relatively symmetrical points of origin, as shown in E and F of Figure 9. One or two dark-staining, comma-shaped rods (parabasal body) may usually be seen lying across the axostyles somewhat posterior to the middle of the organism. The exact function of this structure is not known.

Movements. The body of the trophozoite vibrates rapidly under the action of its flagella, frequently doubling back upon itself. This behavior is so characteristic that identification often may be made from it alone. When the trophozoite becomes moribund and motion has ceased, the flagella, which then usually extend backward along the axial line, may continue to undulate for some time.

Encystment. *G. lamblia* encysts readily. This is the form often seen in semisolid stools and practically always in formed stools. The cysts are usually elongate ovals and contain from two to four nuclei, depending on their stage of development. A pair of rudimentary axostyles and parabasal bodies are to be seen in the cytoplasm. However, there may be considerable diversity, in the relationship of these structures. The cysts vary from 8 to 15 \( \mu \) in length. The nuclei are nearly always at the ends of the cyst and with proper lighting may be seen without staining; their appearance suggesting eyes. In iodine preparations, considerable detail of the nuclei and the flagellar apparatus may be observed, but iron-hematoxylin staining is essential to bring out clearly the finer details.
Multiplication. Binary longitudinal fission of this organism takes place within the cyst, when ingested by man; two offspring are liberated in excystment. A similar longitudinal division in the active form has been described; if this does occur, however, it is rarely observed under ordinary conditions in either duodenal contents or liquid stools.

Treatment and Prophylaxis. In the treatment, control, and prevention of giardial infection, the same general rules applicable to *E. histolytica* and *C. mesnili* should be followed. Such amebicidal drugs as chiniofon, carbarsone, acetarsol, and treparsol have been employed in the treatment of giardial infection but these frequently fail, as do many other drugs which have been tried. In most cases, repeated courses of medication are necessary to effect a cure, and some infections resist all known methods of treatment. Recently, however, claims have been made of highly satisfactory results with atabrin in the same doses and for the same period as employed in treating malaria. In communities in which good hygiene and sanitation are practiced, and yet giardiasis is of common occurrence, it is believed that direct person to person transmission constitutes the most important epidemiologic factor. Organisms of almost identical characters commonly occur in the lower animals, such as the domestic cat, mice, and rats, but their identity with *G. lamblia* of man has not been established.

**Chilomastix mesnili** (Wenyon, 1910) Alexeieff, 1912

Distribution and General Morphology. This intestinal mucosal commensal flagellate (Fig. 9, J, and Table VI) is widely distributed throughout the world. It may be found frequently in microscopic smears and protozoal stool cultures in temperate as well as warm climates. It is a somewhat inflexible, carrot-shaped organism, usually larger than *T. intestinalis*, averaging from 10 to 15 μ in length, although forms much smaller and larger may occur. The anterior end is comparatively large, rounded, and is indented by a spiral groove which extends backward over the tapering body toward the narrow tail-like posterior tip. An axostyle is conspicuously lacking. Beginning anteriorly and extending
backward for about one-third to one-half the body length is the oral pouch or “cytostome.” One of the four flagella is directed posteriorly and undulates in the oral pouch, sweeping food particles through it into the organism. The other three anteriorly directed flagella (together with the cytostomal flagellum) originate in the “blepharoplast” situated just anterior to the anteriorly located nucleus and serve for locomotion and direction of suspended particles toward the cytostome.

**Differential Microscopic Features.** The nucleus, which is detectable with difficulty when unstained, is round or somewhat oval in shape, and is fixed in a relatively far anterior position. Its chromatin is often clumped and is located near one section of the inner surface of the nuclear membrane. The “granula” (or granules) composing the blepharoplast are in apposition to the most anterior portion of the nuclear membrane. Most of these morphologic features may be imperfectly observed in saline and iodine preparations but are brought out most clearly by the iron-alum hematoxylin stain.

**Movements.** Motility of this flagellate is quite characteristic. As compared with other trichomonads, it appears to glide deliberately forward, the body rotating slowly and at the same time oscillating somewhat. There is a notable retention of form and rigidity. These characteristics of the living organism are obvious and provide an important aid in its identification.

**Cyst Form.** Although usually present in the trophozoite form in fresh, liquid stools, *C. mesnili* often produces resistant cysts. These cysts (Fig. 9, K) are considerably smaller than the trophozoite, being 7 to 10 \( \mu \) in length. They are pear-shaped with a characteristic flat, knoblike extension at one end. Sometimes cytostome and nucleus may be seen in the unstained cyst but they are much more readily discernible when stained with iodine or hematoxylin.

**Transmission, Prophylaxis, and Treatment.** Transmission takes place through fecal contamination of hands, food, water, flies, and the like, as in the case of other intestinal protozoa. Measures for control and prevention of infection with this commensal are
similar to those already described for cyst-forming members of this group of organisms. Since *C. mesnili* thrives in spite of modern sanitary precautions against the spread of fecal-borne infections, it is suspected that direct person to person transmission may be a common means of transfer of trophozoites, as well as of cysts. The methods of treatment thus far employed have not given very encouraging results. When the organism is present in large numbers, it seems not at all unlikely that symptoms associated with concurrent diarrhea may be due to their presence. Such a positive correlation appears to be borne out by clinical findings in cases in which no other plausible or probable etiologic factors are apparent or demonstrable.

*Enteromonas hominis* (da Fonseka, 1915) and *Retortamonas intestinalis* (Grassi, 1879) Wenrich, 1932

These two rarer types of intestinal flagellates of man (Table VI) have been reported under different names. Organisms quite similar in appearance to *Enteromonas hominis* and *Retortamonas intestinalis* have been described by Wenyon (1926) employing the generic names *Tricercomonas* and *Embadomonas* (Waskia), respectively. The exact geographical distribution of these types is not known. One or both of them have been found in Europe, Egypt, China, Malaya, the Philippines, Brazil, and the United States. They measure from 4 to 10 μ in length and 3 to 5 or 6 μ in width. In the living state they are colorless, pear-shaped, and actively motile, the movements being jerky and progressive in character.

*Enteromonas hominis* is a very active organism, somewhat flattened on one side along which one of the four flagella is attached, terminating in a free end posteriorly. All four flagella arise near the single anteriorly situated nucleus. The cysts measure from 6 to 8 μ in length, are colorless and appear to have a double wall. Without staining, no structure within can be seen.

In preparations stained with hematoxylin, both trophozoite and cyst of *Ent. hominis* show a characteristic morphology as depicted in *H, H₁*, and *I* of Figure 9. The cytoplasm of the tropho-
zoite appears to be alveolar in structure and the nucleus is sometimes pear-shaped with flagella arising from the smaller anteriorly directed pole. The cysts are ovoidal to round in shape and have from two to four nuclei arranged at opposite ends. The cyst wall is well defined, presenting a double outline.

The cytoplasm of *Retortamonas intestinalis* is finely granular and vacuolated. There is a well-marked cytostomal cleft, a single nucleus and two flagella situated near the anterior end of the somewhat spirally twisted body. The cysts are pear-shaped and measure from 4.5 to 6 μ in length. When stained, both trypanozoa and cyst show a characteristic morphology as depicted in *L* and *M* of Figure 9.

Both of these flagellates have been cultivated by various investigators. Little is known regarding their life cycles and methods of reproduction. The trophozoites increase by binary fission. Transmission is probably associated with ingestion of food or water contaminated with the specific organisms. Infection may be prevented by prophylactic measures similar to those described for intestinal amebae and other flagellate infections. There is no evidence to show that either is pathogenic. The organisms present in feces disappear spontaneously within a comparatively short time.

**Coprozoic or Free-Living Flagellates**

Coprozoic flagellates are accidental contaminants of feces, often derived from cysts ingested with food or water, as in the case of coprozoic amebae. These extrinsic flagellates, similar to coprozoic amebae, usually have contractile vacuoles and nuclei with a relatively large karysome containing practically all the chromatin. *Copromonas subtilis* and *Bodo caudatus* are the two most common forms of this group encountered in feces. Their general morphology is shown in Figure 10, *A* and *B*.

**Blood and Tissue Flagellates**

Blood and tissue flagellates are all pathogenic. They belong to a single family—the Trypanosomidae (Doflein, 1901). They un-
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dergo morphologic changes during certain phases of their life cycles, a correct understanding of which necessitates a clear idea of the relationship of the various genera to the host. Probably this

may be achieved most readily from brief definitions correlated with the diagrammatic table comprising Figure 11. Beginning with the simplest, the differences between the genera in relation to host are as follows:

Genus Leptomonas: Forms which have only leishmania and leptomonas stages in the life cycle and occur in the intestine of invertebrates, transmission being through the leishmania or cyst-like bodies passed in the feces.

Genus Leishmania: Forms which also have only leishmania and leptomonas stages in the life cycle but differ from Leptomonas in that they have both a vertebrate and an invertebrate host, the infective stage for the vertebrate host ordinarily being the lepto-
Gomontias stage in the feces of the insect host. Both the leishmania and leptomonas forms may occur in either host.

Genus *Crithidia*: Forms which have leptomonas, leishmania, and crithidia stages in the life cycle, but occur only in invertebrate hosts, and are transmitted by leishmania or cystlike bodies in the feces, as in *Leptomonas*.

Genus *Herpetomonas*: Forms which have leptomonas, leishmania, crithidia, and trypanosome stages in the life cycle, which occur only in invertebrate hosts, and are transmitted by means of leishmania or cystlike bodies in the same manner as *Leptomonas* and *Crithidia*.

The genus *Phytomonas* is used by some authors to include those forms which have a plant and an insect stage in the life cycle. Other authors prefer to classify these plant-insect forms as *Herpetomonads*.

Genus *Trypanosoma*: Forms which have all the stages or flagellate types in the life cycle listed in Figure 11, requiring both vertebrate and invertebrate hosts. Development in the insect ends with the formation of so-called “metacyclical” trypanosomes which are infective to the vertebrate; in the vertebrate, trypanosomal forms are produced which are infective to the specific insect host.

In so far as man is concerned, “leishmaniasis” is the clinical term applied to those diseases in which the leptomonas form of the parasite is transferred, following the bite, by rubbing in the excretions or crushed body of the infected insect vector. Only the leishmania stage of this genus is ordinarily seen in man. Development in the invertebrate host requires about ten days before the organism becomes infective for the vertebrate host, man. Leishmania forms develop in the intestinal tract of the invertebrate to infective leptomonas forms which reach the pharynx and buccal cavity in from six to nine days. The exact modus operandi of transmission of *L. donovani* thus far has not been satisfactorily determined. However, it is believed, that infection is caused by rubbing of the infective material into the skin puncture produced by the insect or other injury to the epithelium.

The term “trypanosomiasis” is applied clinically to those dis-
cases of man and other vertebrates in which the trypanosome form of the parasite is transferred principally through the bite of the insect vector to the susceptible vertebrate victim. The exact mechanism of transfer of the infective inoculum differs considerably in the African and the Brazilian forms of human trypanosome disease. In Africa the insect vector of trypanosomiasis is generally a "tsetse" fly, and transmission is directly through the bites of these insects; in Brazil a large bug (Triatoma megistá) appears to be the principal transmitting insect agent, transfer of infection being indirect through fecal contamination of the insect bite or other small wound. Trypanosomes occur in various circulating body fluids such as the blood, lymph, and spinal fluid. In Brazilian trypanosomiasis, leishmania forms also occur elsewhere in small nests, particularly in voluntary and heart muscle.

Further details regarding the blood and tissue flagellates and discussion of the diseases which they cause are given in the following chapter.
CHAPTER IV

DISEASES CAUSED BY BLOOD AND TISSUE FLAGELLATES

THE LEISHMANIASIS

Leishmania infections may be grouped in two categories, depending on whether the disease occurs in the endothelial tissues of the internal organs, such as the spleen, the liver, and bone marrow; or in the tissues of the skin or adjacent mucous surfaces, such as the nasopharyngeal mucosa.

VISCERAL LEISHMANIASIS OR KALA-AZAR

Distribution. Kala-azar occurs in the warmer portions of the Eastern Hemisphere (Fig. 12) and is especially prevalent among adults in southern Asia and among children in the Mediterranean region. In the latter and to some extent elsewhere, the dog has been long considered a reservoir of infection for man. Sandflies fed on leishmania-infected dogs showed the presence of this flagellate in 60 per cent of instances. The existence of such a reservoir of infection in close association with children may, in part at least, account for the unusual prevalence of infantile kala-azar in the Mediterranean countries.

Specific Etiologic Agent. Prior to the discovery of the causative agent by Leishman in 1900, kala-azar was known as dum-dum fever in southern Asia, notably in India. The specific organism, designated Leishmania donovani, occurs in large numbers in the endothelium, particularly of the liver, bone marrow, intestinal mucosa, mesenteric glands, and spleen. These cells are often literally packed with leishmania forms (Fig. 13). They multiply
rapidly by simple binary fission until the cell is eventually destroyed and new cells are invaded.

Although the neutrophilic leucocytes and perhaps the mono-

![Figure 12: Distribution of leishmaniasis. (After Manson-Bahr, Manson's Tropical Diseases. Edition 11. Courtesy of Cassell & Company.)](image)

...cytes phagocytize the freed leishmania bodies, the invasion of fresh endothelial cells occur so rapidly that ultimately the host may succumb from the parasitic invasion. Studies of this parasite in certain potential insect vectors and in culture, reveal the presence of the leptomonad stage, in which form transmission is believed to occur. However, there still exists considerable confusion as to the mechanism of transmission in nature. According to Craig and Faust (1940) the following facts have been established by the Indian Kala-azar Commission:

1. *L. donovani* develops without difficulty in the intestinal tract of *Phlebotomus argentipes* into the leptomonas form and its buccal cavity becomes infected with these flagellates.
2. Naturally infected *P. argentipes* have been found by several observers.
3. It is possible to infect susceptible animals by intraperitoneal injection of the contents of the intestine and buccal cavity of *P. argentipes* infected with *L. donovani*. 
4. The experimental production of kala-azar in the hamster, a far-eastern rodent very susceptible to this infection, has been successful only in a few instances out of many attempts by competent workers.

Transmission. In China, as reported by Craig and Faust (1940b), dissection of locally prevalent sandflies (genus *Phlebotomus*).
showed a natural infection rate of 1.66 per cent, and about seventy laboratory-bred flies fed on kala-azar patients showed 6.85 per cent infection. A logical deduction from these data is that the local sandfly probably plays an important role in the transmission of kala-azar in China, and also in other regions, but it may not be the sole agency of transmission.

*L. donovani* may be found at times in the feces and urine of patients with kala-azar; and susceptible animals, such as the hamster, may be infected by feeding on material derived from the diseased tissues. When cages containing normal hamsters were placed in contact with cages of artificially infected hamsters, one-third of the animals thus exposed became infected. The more recent discovery that *L. donovani* occurs in the nasal secretions of patients with kala-azar, suggests the possible importance of droplet transmission.

**Symptomatology.** Kala-azar is characterized by an irregular, undulant type of fever, enlargement of the spleen and liver, anemia with leukemia, edema of the skin, emaciation, diarrhea or dysentery, and cachexia. Unlike Banti’s disease, there is no ascites in kala-azar. It is often confused with typhoid and paratyphoid fevers, relapsing fever, malaria, undulant fevers, and tuberculosis.

**Diagnosis.** The diagnosis of kala-azar can be definitely established only by demonstration of the presence of *L. donovani* in the peripheral blood, splenic or liver pulp, or glandular fluid; in culture; or by successful animal inoculation with infected material, and subsequent identification of the parasite. The aldehyde test of Napier, the antimony and the finger-prick blood tests of Chopra, and Sia’s euglobulin precipitation test are valuable diagnostic aids.* None of these tests should be relied upon to the exclusion of direct demonstration of the presence of the causative organism.†

**Treatment.** Among the many therapeutic agents tried in the treatment of kala-azar, antimony in the form of the double tartrate

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* These tests are described in Manson-Bahr, Manson’s Tropical Diseases, Ed. 11, 1940; Craig and Faust, Clinical Parasitology, Ed. 2, 1940.
† Directions relative to suitable methods will be found in Appendix III.
of sodium and potassium (tartar emetic) and certain of the pentavalent antimony preparations have been found to be almost specific. The former is the least expensive. It is given intravenously in 2 per cent sterile freshly prepared solution. For an adult the initial dose is 2 cc. of the solution (0.04 gm. of drug); this should be increased 1 cc. for each subsequent administration at two day intervals until 5 cc. have been injected in a single dose. As much as 4 gm. of the drug may be administered in this manner. Negative aldehyde and antimony tests indicate elimination of the parasitic agent from the host.

Control Measures. In view of the lack of exact knowledge of the method of transmission of \textit{L. donovani}, it is impracticable to recommend any definite control measures other than the following presumptive prophylactic procedures: (a) control of breeding of sandflies and avoidance of their bite; (b) prevention of contamination of food and drink by dejecta of patients with kala-azar; (c) measures to control the spread of the disease through nasal secretions.

\textbf{Cutaneous Leishmaniases}

Clinically there appear to be two fairly distinct types of cutaneous leishmaniases: “oriental sore,” occurring in southern Asia, the Mediterranean region, and the northern half of Africa; and “espundia” or “forest yaws,” occurring in Central and South America (Fig. 12). Biologically and morphologically the causative organisms are strikingly similar to one another and to \textit{L. donovani} of kala-azar. Recently, however, certain investigators claim to have demonstrated serologic differences. \textit{Leishmania tropica} is the name given to the causative agent of oriental sore, and \textit{Leishmania braziliensis}, to the causative agent of espundia or forest yaws.

Nature of Lesions. In both diseases the lesions usually appear first as reddish papules which gradually develop a covering of fine, dry scales. This covering later becomes moist and forms a crust which, on removal, reveals a shallow ulcer. The lesion which gradually enlarges, has sharply cut, raised edges surrounded by an area of induration. Coalescence of adjacent ulcers and second-
ary bacterial infection result in further extension of the process and the development of a variety of local and general symptoms. In many cases the papules do not become ulcers but disappear after weeks or months, leaving a depigmented depressed scar.

The original skin lesion of espundia, is followed in a certain proportion of cases by apparently independent lesions that occur in the mucous membrane of the mouth, nasopharynx, and pharynx. They may be ulcerative or indurative in character and are correlated with corresponding histopathologic pictures. The characteristic lesions of the mucous membrane may appear either before the primary skin sore has healed, or weeks or months afterward. The first sign is a thickening of the mucosa in the involved area, followed by the development of nodules; these necrose and form ulcers which may spread to the mouth, the hard and soft palates, the pharynx, and even the larynx.

**Transmission.** In endemic regions infection is probably transmitted by contact, as from mother to child, since the etiologic agent (*L. tropica*) in oriental sore is directly inoculable experimentally. However, it is believed that the most common method of transmission in oriental sore is through the bite of the sandfly (*P. papatasii*). Adler and Theodor (1925–1929) have reported that they were able to transfer the infection from man to man by inoculation of material from both artificially and naturally infected *P. papatasii*. It was found that the artificially infected flies contained infective material from the eighth to the twenty-first days, but attempts to transfer the infection experimentally through the bite of the fly resulted negatively. These experiments prove that *L. tropica* may develop in *P. papatasii*, and that artificial inoculation of material containing the parasites which have developed in the fly, will produce typical lesions (both in experimental animals and man) containing the specific parasite.

An insect-transmitting host of *L. braziliensis* in nature is not definitely known. Experimentally it has been found that this species of *Leishmania* developed into the leptomonas form in the sandfly, *Phlebotomus intermedius*, and produced an infection in a dog when the macerated bodies of such infected sandflies were
inoculated subcutaneously. Prior to this, Pedrosa (1913) reported that he found this organism in a dog in the endemic area. In view of these reports, it has been suggested that P. intermedius may be a carrier of L. braziliensis of espundia or forest yaws, and that the dog may serve as a convenient reservoir for the parasite. The infection is inoculable and auto-inoculable, so that direct contact may also play an important role in the epidemiology.

Diagnosis. Diagnosis of both cutaneous and mucosal lesions of leishmaniasis is based primarily upon finding the causative organism in scrapings from the borders or base of the ulcer. Culture methods may also be advantageously employed.

Treatment. Ordinary discrete skin lesions respond readily to local injections of a 1 per cent solution of berberine sulphate—1 to 3 injections of 2 cc. each. More general and severe cutaneous lesions may require intravenous injections of suitable antimony preparations. The latter is essential for the effective treatment of mucosal lesions. It was in the treatment of this condition that antimony compounds were first employed, and following its use intravenously, the prognosis is excellent.

THE TRYPANOSOMIASES

As mentioned in the previous chapter, there are two types of trypanosome infections of man: (1) the trypanosomiases of Africa which are transmitted by flies belonging to the genus Glossina, limited geographically almost exclusively to Africa (Fig. 14), and (2) the trypanosome infection of the Western Hemisphere, known as Chagas' disease, which occurs principally in Brazil but has also been reported from Venezuela, the Argentine, Panama, Chile, Peru, and Guatemala. In so far as the principal insect vector of Chagas' disease is concerned, trypanosomes identical with those found in Brazil have been reported in other species of the genus Triatoma occurring in California and Texas. Thus far, however, no cases of American trypanosomiasis or Chagas' disease in man have been reported in the United States. Species of Triatoma may be found along railways, and similar locations, in various parts of the United States.
African Trypanosomiases (African Lethargy, or Sleeping Sickness)

Two distinct forms of this disease in man are known in Africa: one, widely distributed, is caused by *Trypanosoma gambiense* and transmitted by the tsetse fly, *Glossina palpalis*; the other, much more restricted geographically, is caused by *Trypanosoma rhodesiense* and transmitted largely by the tsetse fly, *Glossina morsitans*. The latter species is the principal insect vector of the trypanosomiasis of cattle and horses known as "nagana" (caused by *T. brucei*) in Central and South Africa. Game animals are believed to serve as reservoirs of infection for man as well as for animals. Ordinarily, trypanocidal substances present in human blood are lethal to this and other trypanosomes of lower animals, but in certain pathologic states it is thought that these substances may not be produced. Under such conditions it is believed by some workers that Rhodesian trypanosomiasis arose in Rhodesia and adjoining parts of Africa. It is even suspected that such animal to man adaptations may still be taking place when favorable circumstances exist.
GAMBIAN FORM

Causative Organism. The gambian form of trypanosome infection is caused by *Trypanosoma gambiense* (Dutton, 1902). This organism occurs in three forms in the blood; namely, long and slender trypanosomes with flagellum, short and broad ones without flagellum, and intermediate forms. In freshly drawn blood or in glandular or spinal fluid, trypanosomes (Fig. 15) appear as rapidly swimming and twisting, slender, double-pointed bodies. Slowing of motility reveals the undulating membrane and the free end of the flagellum, when this exists. For studying the morphology, stained preparations are essential. Blood films on slides prepared as for a differential blood count, dyed with any of the so-called Romanowsky stains, such as Leishman’s, Wright’s or Giemsa’s, are most satisfactory. This trypanosome has a length of about 15 to 30 μ, and a width of 1.5 to 3.5 μ. The slender forms are those most numerous in the peripheral blood. Near the middle of the organism is situated the nucleus, which stains purplish red. Close to the nonflagellar or posterior end lies the “kinetoplast,” made up of blepharoplast and the parabasal body, both similar in staining reactions to the nucleus. The cytoplasm is finely granular, stains a light blue, and may be vacuolated. A number of very dark bluish (sometimes reddish) staining, “volutin-like” granules are also present in the cytoplasm. The undulat-
ing membrane, staining light blue, appears to begin near the blepharoplast and ends at the anterior or flagellar end. The characteristic binary longitudinal division may be seen in blood and lymph in its various phases.

Formerly it was thought that the slender and stumpy forms constitute a sex differentiation, but this is very doubtful. Round or pear-shaped so-called “involutional” forms have been observed frequently in spinal fluid. Figure 16, comprising a differential chart of trypanosomes of importance in medical practice, shows \( T. \) gambiense, \( T. \) rhodesiense, \( T. \) cruzi, and \( T. \) lewisi (of the common rat) for comparison. This figure will be referred to subsequently in order to epitomize graphically various fundamental details regarding the two human trypanosomes.

**Transmission.** As has been stated already, \( T. \) gambiense is transmitted to a new host through the agency of the tsetse fly; \( Glos-sina palpalis \) (Fig. 71) is the species most frequently concerned. Although the transmission may be mechanical, the usual method requires an incubation or latent period of at least eighteen days in the fly; during this period the infective organisms undergo morphologic changes (cyclic development) and increase greatly in numbers. In mechanical transmission the time interval between the ingesting bite and the infective bite must be short.

The developmental changes in the tsetse fly (Fig. 17) terminate with the “metacyclic” or infective forms. Trypanosomes \( (A,D) \) ingested with the blood undergo rapid multiplication into a great variety of forms in the sucking stomach of the fly until the seventh to tenth day. From this time on, long, slender forms \( (E,I) \) appear in great numbers. They gradually migrate to the proventriculus of the fly, thence to the salivary glands \( (S) \) where they assume the crithidial shape \( (B) \) and attach themselves to the wall of the gland. After from two to five days of development there, metacyclic forms \( (C) \) are finally produced. Under optimum conditions, the complete cycle of development in the tsetse fly requires about eighteen days. Experimentally, the total number of flies becoming infected has been reported to be considerably less than 8 per cent.
<table>
<thead>
<tr>
<th>Species</th>
<th>Geographic Distribution</th>
<th>Length</th>
<th>Type</th>
<th>Red Blood Cell Damage</th>
<th>Tissue Damages</th>
<th>Drawing from Blood, Serum and Tissue</th>
<th>Coloring</th>
<th>Lactate Production</th>
<th>Culture</th>
<th>Disease Produced</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. gambiense</td>
<td>Tropical Africa</td>
<td>15-25</td>
<td>Elliptical</td>
<td>Small</td>
<td>N/A</td>
<td>Parasites seen as T. gambiense except in subcutaneous masses in suckers, lesions in vacuoles within macrophages</td>
<td>N/A</td>
<td>N/A</td>
<td>African</td>
<td>Sleeping sickness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60-80</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T. rhodesiense</td>
<td>East Africa, Sudan</td>
<td>16-18</td>
<td>Polygonal</td>
<td>Small</td>
<td>N/A</td>
<td>Parasites seen as T. rhodesiense except in subcutaneous masses in suckers, lesions in vacuoles within macrophages</td>
<td>N/A</td>
<td>N/A</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>35-55</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T. cruzi</td>
<td>Central and South America</td>
<td>13-16</td>
<td>Spirochete</td>
<td>Large</td>
<td>N/A</td>
<td>Parasites seen as T. cruzi, except in subcutaneous masses in suckers, lesions in vacuoles within macrophages</td>
<td>N/A</td>
<td>N/A</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>25-40</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
<td></td>
<td>N/A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T. lewisi</td>
<td>Cosmopolitan</td>
<td>20-30</td>
<td>Mannan</td>
<td>Large</td>
<td>Yes</td>
<td>Parasites seen as T. lewisi, except in subcutaneous masses in suckers, lesions in vacuoles within macrophages</td>
<td>N/A</td>
<td>N/A</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fig. 16. Differential chart of trypanosomes of medical importance, × 1000 app. (Courtesy of the Army Medical School and Museum.)
T. gambiense may be transmitted through coitus, as is T. equiperdum of horses. Evidences of transfer of the parasite from mother to offspring through the placenta and the milk have been reported. Such instances, however, are apparently rare.

Pathology and Symptomatology. The lymphatic and nervous systems are affected most by T. gambiense infection. The glands become enlarged; involvement of the postcervical, submaxillary, inguinal, and femoral glands is frequently suspected early in the disease by their characteristic rubbery feeling upon palpation. Later in the disease, when the glands become fibrosed, there may be little enlargement.

During the early, acute febrile phase, it is often difficult to differentiate this infection from various other tropical fevers. How-
ever, if the patient's history contains a record of previous contact with tsetse flies in known endemic regions, repeated careful scrutiny of the more accessible lymphatic glands should indicate to the informed clinician the probable nature of the disease.

The "sleeping-sickness phase" of the disease is a more or less chronic one. It begins with invasion of the central nervous system by trypanosomes from the circulatory, and possibly also the lymphatic, system. As the invasion advances, the brain shows, macroscopically, the usual lesions of leptomeningitis; the cerebrospinal fluid is increased in amount, turbidity and trypanosomes are often present. The most characteristic microscopic lesion is perivascular, cellular infiltration, involving particularly the capillaries and smaller blood vessels. This is often indistinguishable from similar infiltration in other forms of encephalitis.

The symptomatology during this period is a logical outcome of the evolving pathology and is a subject too remote and varied to be discussed here. Complete discussions may be found in books on tropical diseases, some of which are listed on pages 448-495.

**Diagnosis.** In human trypanosomiasis, an authoritative diagnosis depends upon demonstration of the presence of the parasite in the blood, or in the glandular or cerebrospinal fluid. Direct microscopic examination of one or more of these should be made at once if practicable, and a smear for staining prepared at the same time. Other available methods of examination are culture, and animal inoculation, with or without previous employment of suitable concentration methods.

The earlier the diagnosis is established, the better will be the prognosis after prompt appropriate treatment. In the early stages, symptoms of diagnostic importance are irregular fever, enlargement of lymphatic glands, particularly of the posterior triangle of the neck (Winterbottom's sign), erythematous skin eruptions, and delayed pain sensation (Kerandel's sign). Differential diagnosis from malaria, relapsing and undulant fevers, ancylostomiasis, and syphilis is important.

**Treatment.** The most reliable therapeutic agents thus far employed in treating *T. gambiense* infections are tryparsamide and "Bayer 205." The former has proved very effective, but is re-
BLOOD AND TISSUE FLAGELLATES

reported to be of little value in *T. rhodesiense* infections. The most satisfactory results are obtained by intravenous administration, the initial dose being 1 gm. weekly. This may be gradually increased to 2 or 3 gm. weekly but the dose should never exceed 0.035 gm. per kilogram of body weight. The total amount required to effect a cure varies from 24 to 80 gm. Larger individual doses may be required in the lethargic phase. Eye pain, photophobia, and lacrimation, as premonitory signs of optic neuritis, are likely to occur with the use of this drug. Tryparsamide is reported to be most effective prior to the appearance of trypanosomes in the spinal fluid. After the development of cerebrospinal symptoms, favorable results may be expected in not more than 50 per cent of the cases. However, tryparsamide appears to be the drug of choice in treating the lethargic stage of this disease.

In the early stages of both the human African trypanosomiases, "Bayer 205" (germanin) has been found to be very effective; it appears to be useless after the development of the lethargic state. It is given intravenously in doses of 1 gm. for an adult, 0.7 to 0.75 gm. for children at intervals of one week, for ten weeks. This drug is cumulative in its action and albuminuria may develop, but apparently little permanent damage is done to the kidneys. Other drugs, such as atoxyl and tartar emetic, have been employed, but the results are less satisfactory than those following the proper use in selected cases of tryparsamide and Bayer 205.

Prophylaxis. The measures to be adopted for control and prevention of *T. gambiense* infection are: (a) isolation and proper treatment of all infected persons; (b) protection of man from bites of tsetse flies; (c) destruction of breeding places of tsetse flies, particularly *Glossina palpalis*; (d) control of persons traveling from infected areas to noninfected areas; (e) removal of inhabitants from endemic areas; (f) use of prophylactic drugs, particularly Bayer 205, in suitable doses.

RHODESIAN FORM

Causative Organism. The rhodesian type of trypanosome infection is caused by *Trypanosoma rhodesiense* (Stevens and Fantham, 1910).
This organism was first recognized by Stevens in 1910 in a patient from Rhodesia who was suffering from symptoms suggestive of trypanosomiasis. Morphologically, this parasite is very similar to *T. gambiense*, except that when it is inoculated into mice, rats, and guinea pigs, posterior nuclear forms sometimes develop which are similar to those of *T. brucei* inoculated in the same way (Fig. 16).

**Epidemiology.** As previously pointed out, this variety of African trypanosomiasis is quite limited geographically (Fig. 14) and appears to have an important reservoir in the antelope inhabiting the localities where it occurs.

The epidemiology of Rhodesian trypanosomiasis may be briefly summarized as follows:

(a) *G. morsitans* is the usual transmitting agent.

(b) Epidemics seldom occur.

(c) Incidence is small, the actual cases being sporadic.

(d) Certain game animals afford suitable reservoirs for keeping the infection alive.

(e) Laboratory animals are easily infected experimentally and the ultimate result is rapidly fatal.

(f) Cross immunity and diagnostic serologic tests thus far have been unsatisfactory.

**Pathology and Symptomatology.** The disease resulting from *T. rhodesiense* runs a much more rapidly fatal course than does that resulting from *T. gambiense* infection, both in man and in experimental animals. In fact, it often ends fatally before there is time for the development of the nervous symptoms so typical of *T. gambiense* infections.

**Diagnosis, Treatment, and Prophylaxis.** Conduct of these measures should follow principles and procedure adequately discussed in connection with *T. gambiense* infection.

**Brazilian Trypanosomiasis (Chagas' Disease)**

This disease is caused by *Trypanosoma cruzi* and occurs in two distinct clinical forms which may appear separately or consecutively as acute and chronic syndromes, or in some intermediate
phase. In contrast to the African trypanosomiases, the transmitting agent is a triatome bug, and not a biting fly. A brief description of the Triatomes is given in Part III.

![Life history of Trypanosoma cruzi.](image)

**Distribution.** Although this infection was at first believed to be limited to Brazil, it is now known to occur also in Venezuela, Chile, Peru, Guatemala, Panama, Uruguay, and the Argentine (Fig. 14), and probably also in Mexico. The causative organism, as already stated, has even been found to occur in related triatome bugs in California, Texas, and Arizona, although no cases of infection have been reported from those states.

**Causative Organism.** *Trypanosoma cruzi* (*Schizotrypanum cruzi*) Chagas, 1909, is the parasitic agent. This is a trypanosome in which both the trypanosome and leishmania forms occur in man and crithidia forms in the insect transmitter. (Fig. 18 shows the relationships of these forms.) In man, *T. cruzi* occurs in the blood as a trypanosome, and in the endothelial and other tissue
cells as typical leishmania bodies, the various stages (leptomonas, crithidia, and trypanosoma) probably appearing within the cells involved preparatory to re-entering the circulation as adult trypanosomes. In the insect vector, only crithidia and trypanosomes have been observed.

**Life Cycle in Man.** In the blood of man, two forms of the trypanosome (Fig. 18) occur—one long and slender, the other short and broad (C). It is questionable whether this difference in form has sexual significance, as was formerly believed. Both forms are considerably longer than the diameter of a red blood cell; the nuclei are situated at about the middle of the body, and the kinetoplasts well posterior. The kinetoplast is very large in the short forms and stains deeply. Other characteristic features of this trypanosome in stained preparations are the few convolutions of the undulating membrane, the C shape, and the absence of dividing forms in the blood.

Transformation into and binary division of leishmania forms occur in tissue cells, especially of the striated and heart muscle (Fig. 18, A). The leishmania bodies divide repeatedly and eventually fill and destroy the invaded cell. These leishmania forms are round or oval in shape and measure from 1.5 to 4 μ in diameter, each presenting a large nucleus and a rodlike or spherical dark-staining kinetoplast. While their progressive evolution into leptomonas, crithidia (D) and trypanosome forms (B) most likely occurs before rupture of the distended and weakened cyst wall, only trypanosome forms (C) are usually found in the circulating blood.

**Life Cycle in Insect.** In the transmitting insect’s stomach (Fig. 18, S) long, short, and intermediate crithidia forms of *T. cruzi* occur, with the kinetoplast situated anteriorly. The “metacyclic” or infective trypanosomes with posterior kinetoplasts and well-developed undulating membranes and flagella are also found in the transmitting triatome. Instead of progressing anteriorly to the salivary glands and the base of the proboscis (the so-called anterior station), as in the evolution of trypanosomes in the tsetse fly, these crithidia forms pass posteriorly into the triatome’s
hindgut or rectum (Fig. 18, R) and are passed out with the feces after developing into metacyclic forms.

Although authorities are not in agreement concerning certain phases of the life cycle (particularly in the insect vector) this brief summary comprises the views which are generally accepted. Eight to ten days are required for the small metacyclic or infective forms to develop from the crithidial forms first appearing in the insect's rectum. The former are passed out with the feces and are infective for man and other suitable animals when rubbed into the puncture wound produced by the insect or into any other trauma of the skin.

**Mechanism of Transmission.** Infection with *T. cruzi* is incompletely biological since the feces of the transmitting insect containing the infective metacyclic trypanosomes must be introduced mechanically into the wound or abrasion. *Triatoma megista*—the immediate reservoir and transmitter of *T. cruzi*—frequently defecates at the time of biting, the infective feces being supplied at the most opportune time for introduction of the trypanosomes into the fresh wound by rubbing and scratching. As has been stated, triatomes become infective in from eight to ten days after biting an infected person or animal, and may remain so for as long as two years. The adult triatomes, rather than their larvae or nymphs, are usually the transmitters.

**Reservoir Hosts.** Various animals, such as the armadillo, the bat, and the monkey, may serve as reservoirs of *T. cruzi*. In California, the opossum and the wood rat have been found to harbor a morphologically similar trypanosome. Thus far, however, no case of this trypanosome infection in man has been reported from that region. Dogs and cats may also serve as reservoirs of infection, and various laboratory animals have been experimentally infected.

**Epidemiology.** According to a few observations and experiments, it seems probable that *T. cruzi* may be transmitted through coitus, through the milk of an infected nursing mother, and through unbroken conjunctiva or mucosa.

Social status of the patient is of significance, since in endemic areas Chagas' disease occurs among the very poor. The triatomes
live in cracks, holes, thatch, adobe walls, and similar places, and bite at night, retiring to their hiding places before daylight. The bite is practically painless. The mucocutaneous junctions, particularly of the lips, are sites of preference; hence, the common name, “kissing-bug.” Children are frequently observed sleeping quietly while one or more triatome bugs feed upon them. Such circumstances probably account, in part at least, for the greater prevalence of this disease among young children.

Pathology. The essential pathology of Chagas' disease is dependent upon the predilection of T. cruzi for the reticulo-endothelial system, heart muscle, and neuroglia cells of the central nervous system. The destruction of cells of the various organs, and the blockage of the reticulo-endothelial system by T. cruzi lead to various degrees of functional impairment, and not infrequently death is the final outcome. In severe infections the leishmania forms of the parasite may be seen in great numbers within the invaded cells which are swollen, fragmented and eventually destroyed. These pathologic changes explain the varied nature of the symptomatology which depends upon the extent of damage done by the parasite in the invaded tissues and organs.

Symptomatology. Although the acute form of Chagas' disease in endemic areas usually occurs in children, it is also seen sometimes in adults. The chronic form, which follows the acute form in children surviving the acute attack, may constitute the primary clinical manifestations of adult infection with T. cruzi. The period of incubation in man appears to vary between seven and fourteen days.

Acute phase. The acute phase of Chagas' disease is characterized by high fever, which may be intermittent, remittent, or continuous; this is followed about two weeks later by a deposit of mucoid material in the invaded tissues, resulting in various degrees of tumefaction. The face is swollen and there is marked edema of the eyelids and conjunctivae (often unilateral). The general appearance of the patient may suggest myxedema; the swelling does not pit on pressure and the skin is dry. Keratitis is frequently observed. The thyroid, the glands about the angle of
the jaw, the lymph nodes, liver, and spleen are usually more or less enlarged. Trypanosomes may be found frequently in the peripheral circulation. Death may occur, it is said, within from two to four weeks, or the chronic phase of the disease may supervene.

**Chronic phase.** In the chronic phase the symptoms depend on the localization of trypanosomes. Cardiac, myxedematous, meningo-encephalic, ovarian, and suprarenal types have been described. Acute febrile paroxysms may occur, during which trypanosomes may be found in the blood; otherwise, they are not usually found in the peripheral circulation. However, trypanosomes may occur in the blood without any related symptoms, suggesting a possible carrier state.

**Diagnosis.** While the diagnosis may be made most readily when trypanosomes are present in the peripheral circulation, this is usually possible only in the acute phase and during acute febrile exacerbations of the chronic phase. Other valuable means of diagnosis are:

(a) Inoculation of guinea pigs with blood of persons suspected of having the disease.

(b) At times, splenic or sternal puncture may be indicated in special cases.

(c) Brumpt's (1914) method of "xenodiagnosis," which consists in permitting laboratory-bred triatoma to bite and feed on the exuding blood of individuals suspected of having the disease.

(d) Complement fixation. Of the methods proposed, that of Kelser (1936), in which the antigen is prepared from cultures of *T. cruzi*, is the most reliable. A survey of some 1600 persons in Panama by this method gave 3.7 per cent positive reactions, indicating that in endemic areas latent infections probably exist quite commonly, and that such individuals may serve as reservoir-carriers of the causative organism.

**Treatment and Prognosis.** Since no effective treatment for this disease is known the prognosis is discouraging in both children
and adults, although spontaneous recovery may occur in children. The various therapeutic agents frequently effective in treating the African trypanosomiases are of little use in the treatment of Chagas' disease. Thyroid extract has been tried in myxedematous cases but the results have not been encouraging.

Prophylaxis. Individual protection from bites of triatomines and other trypanosome-infected arthropods, and household and community destruction of the transmitting insects and reservoir animals are essential in the control and prevention. The extreme poverty of the persons most frequently exposed to the disease in endemic areas makes the application of both control and preventive measures difficult and discouraging in many instances.
Sporozoa are cell parasites that are essentially pathogenic since they destroy the parasitized cells. A clear understanding of the clinical conditions caused by this group of parasites requires an intimate knowledge of their rather complicated life cycles. Their development, however, is in general the same, whether the parasite is one of the malaria plasmodia or a coccidium. Although the latter parasite is comparatively rare in man and probably produces little tissue damage in most instances, it occurs very commonly among the lower animals, invertebrate as well as vertebrate. So-called "grouse disease" of game birds and coccidiosis of rabbits often cause serious economic losses. Among mice, rats, cats, and dogs, however, the condition is usually transient and tends to disappear spontaneously as the animals grow older.

COCCIDIA

Although coccidiosis in man is of comparatively little importance clinically, the causative organism will be briefly discussed in order that the student may comprehend more readily the intricate details of the far more important human malaria parasites.

LIFE CYCLE OF COCCIDIA

Coccidia, which are limited to the epithelial cells of the wall of the small intestine or the bile passages, ordinarily require but one host; whereas the malaria plasmodia require two hosts, and in man parasitize red blood corpuscles.

In Figure 19 is shown semidiagrammatically the complete life cycle of a one-host coccidium. The primary infective elements
(sporozoites, \(r_9, 5\)) are produced at two points in the life cycle, and give rise to two different phases of development. Both asexual or schizogenous multiplication and sexual or anisogamous develop-

Fig. 19. Life cycle of coccidia. Diagrammatic representation (X 400 app.) of the asexual and the sexual cycles within intestinal epithelium and of the development of the cysts (stages 13–18) outside the body. 1–6, Asexual schizogonous phase. 7–10, Male gametogonous phase. 11–14, Female gametogonous phase. 15–19, Sporogonous phase. (After Lynch, Protozoan Parasitism of the Alimentary Tract, 1930. Courtesy of the Macmillan Company.)

ment occur. These will be briefly outlined in the following paragraphs.

**Schizogony.** Oocysts, each containing four sporocysts (Fig. 19, 18), on reaching the small intestine of a suitable host through its
mouth, have liberated from them the infective sporozoites. These slender motile vermiculate bodies penetrate epithelial cells, grow and multiply asexually. This so-called schizogonous process recurs in regular cycles, but in some of the parasitized cells evidence of development of a sexual phase soon appears. The sexual phase (Fig. 19, 5-14) constitutes gametogony, the later stages of which take place outside the body of the host and are known as sporogony.

The motile sporozoite (Fig. 19, 6), having gained entrance to a mucosal epithelial cell, rounds up, and through nuclear divisions and attendant changes within its confining cell wall evolves into a “schizont.” The schizont when fully mature is known as the merocyte and contains slender bodies, the so-called “merozoites,”* which escape into the intestinal lumen. These merozoites attack and enter fresh epithelial cells to repeat the same process of asexual reproduction again and again (Fig. 19, 1-6).

Gametogony. After a time, as already indicated, immature sexual forms, or “gametocytes,” arise in some of the parasitized cells. These are the microgametocytes and the macrogametocytes, which give rise to microgametes (male elements) and macrogametes (female elements), respectively. Various intermediate steps in these evolutions of the gametogonous process are shown in 7, 8, 9, and 10 of Figure 19 for the microgametes arising from a single infected cell, and in 12, 13, 14 for a single macrogamete.

Sporogony. Upon escape of the fertilized macrogamete, ovum or “zygote” into the intestinal canal, by the bursting of the cell housing it, extracellular development begins. In warm-blooded animals† this development terminates with the maturing oocyst in the external world (Fig. 19, 15, 16, 17, and 18), ready to parasitize (by its sporozoites, 19) the intestinal epithelium of a new host of the same species.

The progressive development of the zygote into sporoblasts

* Merozoites are comparable to sporozoites but in this case result from asexual multiplication of schizogony in contradistinction to the true sporozoites developed in the oocyst in the feces, the latter process often being referred to as “sporogony.”
† In cold-blooded animals, sporogonous development may take place entirely in the body of the host or hosts as in the Aggregatidae.
(Fig. 19, 16, 17), sporocysts (18), and eventually sporozoites (19) which are freed through rupture of the oocyst is termed sporogony. It constitutes in reality a completion of the gamatogonous process.

and some authors prefer not to employ a separate name for this phase of the life cycle. However, the term, when properly understood, tends to make description of the life history of both coccidia and the malaria parasites easier to understand.

**Diagnosis, Pathology, and Symptomatology**

The coccidial parasites reported for man and those of domestic animals belong chiefly to two genera, *Eimeria* and *Isospora*. Mature oocysts found in feces are oval or round; those of *Eimeria* contain four sporocysts, each of which has two sporozoites; *Isospora* oocysts contain two sporocysts, each of which has four sporozoites. The diagnostic features of oocysts of these two genera, as reported for man, are shown in Figure 20.

Only a few cases of human infection by *Eimeria* have been reported in the literature, and not more than a few hundred cases by *Isospora*. It is maintained by some workers that the liver lesions in the alleged *Eimeria* infection in man are similar to those produced by *Eimeria stiedae* in the rabbit.
THE SPOROZOA

In *Isospora hominis* the pathology is unknown since the lesions of the alleged clinical condition have never been observed at autopsy. Infections by *Isospora* are certainly self-limited and are probably only accidental in man, the result of aberrant parasitism. Some parasitologists now question the validity of *Isospora* as a human parasite.

Little of a positive nature is known regarding the pathology and symptomatology of coccidiosis in man. One may only reason from analogy in the lower animals as to its probable effects on the tissues and the symptoms likely to arise therefrom. Therefore, it would be unprofitable to speculate further on the various other aspects of this condition which naturally suggest themselves.

MALARIA PLASMODIA

LIFE CYCLES OF MALARIA PLASMODIA AND OF COCCIDIA CONTRASTED

The malaria parasites, like coccidia, have an asexual and a sexual phase of existence. In coccidiosis one host species is usually sufficient for the completion of both these phases, while in malaria two host species (man or monkey etc.; and mosquito) are required. If, however, the several stages in the life cycles of malaria plasmodia and of coccidia are compared, a striking similarity becomes apparent. In the anopheles mosquito *(the definitive, primary, or transmitting host of the human malarias)*, sporogony takes place. This begins with sexual union of male gamete (microgamete) and female gamete (macrogamete) and terminates with the production of sporozoites.

In *coccidiosis*, a motile male microgamete enters the immotile macrogamete through one pole, either before or after the latter leaves the epithelial cell housing it. The subsequent phases of this cycle of development (sporogony) are completed independent of the host.

In *malaria*, both fertilization by union of sexual gametes and subsequent phases of evolution to sporozoites take place within

*For entomogical details see pages 249 to 261.*
the stomach and salivary glands of the mosquito the primary host. Schizogony (asexual reproduction) takes place in the blood of man, who is the intermediate or secondary host.

**Mosquito Phases of Life Cycle: Gametogony and Sporogony.** When females of certain species of the genus *Anopheles* ingest blood from a malaria-infected person harboring mature gametocytes, or when such blood is drawn and kept under proper conditions of moisture and temperature, the following transformations involving partial gametogony and all of sporogony have been observed to occur.

1. Within about fifteen minutes the male gametocyte develops 6 to 8 cytoplasmic, flagella-like appendages which lash about violently and soon separate from the parent cell to become the microgametes or spermatozoa.

2. The female gametocyte, during the same period, undergoes a process of maturation, being now known as the macrogamete, in preparation for fertilization by a microgamete to become a zygote.

3. The zygote rapidly assumes a motile vermicular form known as the ookinete, which penetrates the wall of the female mosquito's stomach, rounds up beneath the serosa of that organ, and becomes immotile. This resting form is called the oocyst, the chromatin content of which divides up rapidly into thousands of granules. These granules (nuclear in nature) become grouped together, usually in from ten to twenty clumps, which may be compared to the sporoblasts and sporocysts of coccidia. Finally, each chromatin granule develops into a slender fusiform body, the sporozoite. Ultimately the mature oocyst ruptures into the body cavity of the mosquito.

4. Liberated motile sporozoites soon make their way to the female mosquito's salivary glands, there to await an opportunity to pass into the blood of man or other suitable vertebrate at time of feeding.

This process of sporogony, as revealed by appropriate sectioning and staining, is well depicted in the lower half of Figure 21.* An

*Reference to Figure 64 (page 251) is suggested as an aid in more readily comprehending sporogony as well as gametogony and schizogony.
Fig. 21. Life history of the malaria parasite *Plasmodium falciparum* in man and the anopheles mosquito. (Modified from Herms, Medical Entomology. Edition 3. Courtesy of the Macmillan Company.)
anopheline female mosquito is shown performing two acts consecutively: (1) injecting her infective sporozoites (left), and (2) drawing into her stomach blood containing mature gametocytes (right), initiating the sexual sporogonous cycle in her own body. While a single already infected anopheline female biting an already infected person might reinfect both herself and her victim, infection is usually independent in both hosts. A careful study of this figure should make these relationships clear.

To summarize, both coccidia and malaria plasmodia live essentially a two-phase sexual existence; namely, intrinsic and extrinsic with reference to the vertebrate host. In both parasites the intrinsic phase begins in the vertebrate host; but in vertebrate coccidia it is completed in the feces and external environment, whereas in malaria parasites it is completed in the mosquito, the definitive or primary host. The schizogonous cycle (asexual) is confined to the vertebrate; the gametogenous process (sexual), while beginning in the vertebrate host, requires a change of biological environment for its completion and ultimate consummation in the so-called sporogonous process. Simple examples of this are the one-host coccidioses which have been briefly described. In the aggregatid family of coccidia, however, a second host is required for continuation of the sexual process. These may be looked upon as marking a transition from a one-phase to a two-phase sexual existence.

**Schizogony and Gametocyte Formation in Vertebrate.** The malaria plasmodia differ from coccidia in that the intrinsic or endogenous cycle of development takes place not in epithelial cells but in erythrocytes of the vertebrate host in which the phases of schizogony and formation of gametocytes take place. The merocyte constitutes the terminal infective body in schizogony and the gametocytes, appearing a little later, become gametes in the stomach of an appropriate anopheline mosquito, or in drawn blood under proper conditions of moisture and temperature.

In the schizogonous stage of development of the malarias, the associated symptomatology varies somewhat, according to the species of plasmodium involved.
THE SPOROZOA

*Plasmodium vivax* gives rise to benign or simple tertian *malaria.

*Plasmodium malariae* gives rise to quartan *malaria.

*Plasmodium falciparum* gives rise to malignant, subtertian,*
estivo-autumnal, or the so-called tropical, malaria.

In all three instances it is the asexual or schizogonous generation
that causes the specific fevers associated with the presence of each
of these three malaria parasites in man, the intermediate host.

*Plasmodium vivax* (Grassi and Filetti, 1890)
AND BENIGN TERTIAN MALARIA IN MAN

Schizogony. The sporozoites of *Plasmodium vivax* are injected
into the blood stream by the infected female anopheline mosquito,
where they shortly parasitize red blood corpuscles, round up
and form the typical “signet rings,” which are one-sixth to one-
third of the diameter of the containing cell. While, to some extent,
these organisms may be studied in the fresh wet coverslip prepara-
tion, stained, thin, dry smears or spreads are preferable. After the
necessary experience in ordinary thin film diagnosis has been ac-
quired, diagnosis by means of the thick, laked film is also desirable,
particularly when the parasites are few in number.

After several hours the intracorpuscular parasite shows con-
siderable enlargement and an irregular ameboid form. In fresh
coverslip preparations, ameboid movement of the parasite may be
detected under subdued transmitted light. This particular species
tends to enlarge the containing cell in the cytoplasm of which
rather fine dots or stippling (Shüffner’s dots) frequently occur.
Small granules of brown pigment also appear in the cytoplasm of
the parasite. Growth is rapid, and by the end of thirty-six hours
the plasmodium fills about two-thirds or more of the enlarged,
parasitized cell. From four to six hours later the parasite prac-
tically fills the entire cell and the chromatin divides ordinarily
into from fifteen to twenty-four fragments. At this stage, yellowish
Fig. 22. Malaria Parasites, x 2000 app. (Assembled and modified from various sources.)

**Plasmodium vivax**

1. Trophozoite, young ring form.
2. Trophozoite, early ameboid form.
4. Schizont, young form undergoing nuclear divisions. Schüffner’s dots abundant.

**Plasmodium malariae**

1. Trophozoite, young ring form.
2. Trophozoite, ameboid form. Fine black pigment granules present.
3. Trophozoite, young band or equatorial form.
4. Trophozoite, mature band form. Chromatin in single band along one side of parasite.
5. Schizont, binucleate form. Chromatin in two large clumps at opposite poles.

**Plasmodium falciparum**

1. Trophozoite, young ring form.
2. Trophozoites, young ring forms showing peripherally situated parasites.
3. Trophozoites, ring forms showing double “signets” and appliqué form.
4. Trophozoites, young ameboid forms in severe multiple infection, showing several Maurer’s dots (malignant stippling).
5. Schizont, mature form. This as well as the earlier schizont are rarely seen in the peripheral circulation but occur internally in such places as the brain capillaries.
Plasmodium vivax (Benign Tertian)

Plasmodium malariae (Quartan)

Plasmodium falciparum (Malignant Tertian)

Figure 22
THE SPOROZOA

brown pigment is scattered throughout the cytoplasm of the parasite; later it is found clumped toward the center of the organism, at which time a definite segmentation of the cytoplasm around chromatin fragments also occurs. These dozen or more small bodies each consisting of a small mass of chromatin surrounded by cytoplasm are known as “merozoites.”

At the end of forty-eight hours the corpuscular membrane bursts, liberating the merozoites, and “rigor” or chill begins if the magnitude of this phenomenon has been great enough. Various stages of this schizogonous development are shown in Figure 22, A (7–8). The relationships of these several stages to the temperature curve are co-ordinated in time with a typical simple tertian fever curve in Figure 23. Owing to the fact that infecting anopheline bites are likely to occur not only at various times but in variable numbers at different times, the actual blood census may be quite different from that shown in Figure 23, presenting a wider variety of forms at any one time. Also, all sporozoites injected at one time may not succeed in penetrating erythrocytes simultaneously, resulting in a lag of development for some of them. However, in the majority of instances one developmental stage predominates, and release of its merozoites sets the time of rigor and rising temperature.

Gametocytes—Formation and Morphology. Within about seven days from the time of onset of the initial fever, gametocytes, the forerunners of sexual forms, begin to appear in the peripheral circulation. These gametocytes arise from aschizogonous ring forms, about four days being required for completion of their development. They become infective for the female anopheline mosquito after they are from seven to ten days old, and their entire span of viability is said to be only about twenty days. They are probably incapable of reproduction in man or of initiating the sexual cycle without transfer to a suitable female anopheline mosquito.

The gametocytes when fully grown are rounded in form and the cytoplasm is fairly homogeneous. The male gametocyte has a diameter of from 7 to 8 μ. It occupies a somewhat enlarged red
Fig. 23. Life cycle of *P. vivax* in man showing relation to temperature curve. Parasites ×1000 app. (Courtesy of the Army Medical School and Museum.)
blood cell without completely filling it. Shüffner’s dots may be present close to the margin of the cell. The chromatin is spread over a rather wide area near the center or edge of the cell, sometimes in the shape of a band. The cytoplasm of the parasite stains light bluish green or gray with Giemsa and contains numerous scattered granules of brown pigment. The female gametocyte is somewhat larger (8 to 10 μ in diameter) than the male; it fills the enlarged parasitized cell more completely, and its cytoplasm stains a darker blue. The chromatin is collected into one compact clump, usually near the edge of the parasite, and the pigment occurs in coarse, compact, brown granules. In the peripheral circulation, it has been estimated that female gametocytes outnumber male gametocytes in the proportion of from 3 to 1 to 6 to 1. The morphology of these sexual forms is shown in Figure 22, A (7–8).

**Plasmodium malariae** (Laveran, 1881) Grassi and Filetti, 1890

*Plasmodium malariae* causes quartan malaria fever with paroxysms every fourth day. It is the rarest of the three generally recognized malaria parasites although it was formerly more commonly seen, particularly in temperate regions. At present it appears to be limited for the most part to tropical and subtropical regions. Its distribution, however, is very irregular. The symptoms produced by *P. malariae* are often much more severe than the number of parasites observed seems to warrant. The majority of new cases usually occur during the autumn months. Practically all stages of the parasite may be present in the peripheral circulation at the same time, although one of them is usually predominant.

Reproduction of *P. malariae* is similar in principle to that of *P. vivax* and *P. falciparum*. There are, however, certain specific differences.

**Schizogony:**

(a) The early ring forms tend to become larger than those in the other two species during the first six hours.

(b) When the organism is twenty-four to forty-eight hours old, the characteristic “band” forms are often seen.
The segmenting forms develop only from 2 to 12 merozoites.

The pigment is coarse, and dark brown to black in color.

The infected cell usually is not enlarged and Shüffner's dots of tertian malaria, or Maurer's dots of subtertian malaria are rarely present (Ziemann’s stippling).

Seventy-two hours are required for the completion of schizogony.

Gametogony:

The red cells are not enlarged and rarely show Shüffner's dots.

The time required for the development of gametocytes is about seven days.

Sporogony:

More time is required to complete the cycle of development than in the case of *P. vivax*.

Various stages in these three phases of the life cycle of *P. malariae* are shown in Figures 22, B (1–8) and 24.

*Plasmodium falciparum* (Welch, 1897)

*Plasmodium falciparum* causes estivo-autumnal, malignant, subtertian, or tropical malaria. Although next to *P. vivax* in prevalence, it does not occur so far north and south of the tropical and subtropical zones. Estivo-autumnal malaria is the most severe and often the most fatal of the malaria fevers. Early diagnosis and prompt energetic treatment are essential for a favorable prognosis.

In principle, reproduction is similar to that of *P. vivax* and *P. malariae*, already briefly described. There are, however, certain important differences which distinguish this organism from the other malaria parasites.

Schizogony:

The early ring forms are often smaller and more slender in appearance.

Multiple infection commonly occurs and the ring forms may show more than one chromatin dot (double signet).
Fig. 24. Life cycle of *P. malariae* in man showing relation to temperature curve.
Parasites X 1000 app. (Courtesy of the Army Medical School and Museum.)
The ring forms may be present in the peripheral circulation for twenty-four hours, during which time the size may be doubled or even trebled.

The parasitized blood cells are not enlarged. Schüffner's dots are not present, but large granules staining purplish red, called Maurer's dots (malignant stippling), may be present occasionally.

Ring forms and mature gametocytes (crescents) are the only stages usually seen in the peripheral blood.

The later schizont stages and immature gametocytes are usually seen only in the capillaries of the internal organs and the brain. These have a strong tendency to clump and lodge in the capillaries.

The merozoites produced number from 12 to 16.

In very severe infections, bizarre, filamentous and other forms may be seen in the peripheral blood.

Duration of Schizogony is frequently less than forty-eight hours.

Gametogony:

(a) The gametocytes are crescent-shaped with pointed to rounded ends, the male having usually the more rounded ends. Both have a greater length than the diameter of the containing red cell.

(b) The male gametocyte's cytoplasm stains bluish gray and has a central nucleus with diffuse chromatin. The pigment is in coarse grains of brownish black color, and is scattered.

(c) The female gametocyte stains sky-blue; its nucleus is composed of a compact mass of chromatin and pigment.

Sporogony:

(a) This phase of the life cycle is essentially the same as that of P. vivax except that about one-third to one-half more time is required to complete it.

Various stages in these three phases of the life cycle of P. falciparum are shown in Figures 21 and 22, and in correlation with a simple fever curve in Figure 25.
Plasmodium ovale (Stephens, 1922)

Among several malaria plasmodia of less certain or doubtful status reported for man, P. ovale (Fig. 26) merits mention very briefly. In morphology it resembles somewhat P. malariae in fresh, unstained coverslip preparations, the schizonts being non-ameboid and round or oval in shape. Segmenting forms contain only from six to ten (usually 8) merozoites. The red corpuscle is often oval in shape, somewhat larger than normal, and the pigment is finer and lighter brown in color than that of P. malariae. Ring forms stain a deep blue and the chromatin dot is larger. Nearly every parasitized corpuscle shows numerous Shüffner's dots, whereas in P. vivax they are few in number at this early stage of the trophozoite, and stain poorly.

Differential Diagnosis of Malaria Plasmodia

As an aid in distinguishing among the several species of malaria-producing organisms, Table VII is presented. Positive recognition of malaria plasmodia is of the greatest importance
both clinically and epidemiologically. In the matter of prophylaxis also, differentiation of gametocytes from schizonts is most essential. While schizonts are usually not difficult to recognize.

![Fig. 27. A cluster of blood platelets lying upon a red blood cell and simulating malaria parasites, X 850 app. (After Todd and Sanford. Clinical Diagnosis by Laboratory Methods, 1935. Courtesy of W. B. Saunders Company.)](image)

some hesitancy may be experienced in determining what constitutes the gametocytes. In this connection Craig and Faust (1940) point out:

1. The gametocytes of *P. vivax*, *P. malariae*, and *P. ovale* almost entirely fill the infected red blood corpuscle, and in well stained specimens do not show any division of the chromatin into distinct masses distributed throughout the cytoplasm, while those of *P. falciparum* are identified by their crescentic or kidney-bean shape.

2. The microgametocytes of all four species show a pale blue or dark violet-red staining cytoplasm, while the nuclear chromatin of those of *P. vivax*, *P. malariae*, and *P. ovale* is arranged in a loose mass of fibrils or spindle-shaped granules (lying in an unstained oval area stretching across the body of the gametocyte). The microgametocyte of *P. falciparum* is not typically crescentic in that it has broadly rounded ends.

3. The macrogametocytes of all four species show a deep blue staining cytoplasm; the nuclear chromatin of *P. vivax*, *P. malariae*, and *P. ovale* is collected in a dense mass lying in an unstained area either at the center or to one side of the gametocyte. The macrogametocyte of *P. falciparum* is more slender than the micro-
<table>
<thead>
<tr>
<th>Duration of schizogony</th>
<th><em>Plasmodium vivax</em></th>
<th><em>Plasmodium malariae</em></th>
<th><em>Plasmodium falciparum</em></th>
<th><em>Plasmodium ovale</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>48 hours</td>
<td>72 hours</td>
<td>36 to 48 hours</td>
<td>48 hours</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Motility</th>
<th>Actively ameboid until about half-grown</th>
<th>Slightly ameboid during trophozoite stage</th>
<th>Actively ameboid during trophozoite stage</th>
<th>Slightly ameboid during trophozoite stage</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Pigment (hemozoin)</th>
<th>Yellowish brown, in fine grains and minute rodlets</th>
<th>Dark brown or almost black in coarse grains, rods, or irregular small clumps</th>
<th>Very dark brown or black in coarse granules or small masses</th>
<th>Dark brown in coarse granules or irregular masses</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Infected red blood corpuscle</th>
<th>Much enlarged, pale with eosinophilic stippling (Schüffner's dots) at times</th>
<th>Not enlarged. Normal color. No granular stippling</th>
<th>Not enlarged or may be smaller than normal. Darker green (Brassy). Basophilic dots and brick-red clefts sometimes seen in cytoplasm (Maurer's clefts or dots)</th>
<th>Somewhat enlarged, oval or irregular in shape with eosinophilic stippling (Schüffner's dots)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Stages of development seen in peripheral blood</th>
<th>Trophozoites, schizonts, merocytes and gametocytes</th>
<th>Trophozoites, schizonts, merocytes and gametocytes</th>
<th>Usually only trophozoites and gametocytes. In pernicious infections rarely schizonts may be seen</th>
<th>Trophozoites, schizonts, merocytes and gametocytes</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Multiple infection of red blood corpuscle</th>
<th>Occasionally</th>
<th>Very rare</th>
<th>Very common</th>
<th>Rare</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Area of red blood corpuscle occupied by fully developed schizont</th>
<th>Entire red blood corpuscle which is enlarged</th>
<th>Almost entire red blood corpuscle which is not enlarged, may be smaller</th>
<th>From two-thirds to three-quarters of red blood corpuscle which is not enlarged</th>
<th>About three-quarters of red blood corpuscle which is enlarged</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trophozoites</td>
<td>Small and large rings. Usually one chromatin dot. Ameboid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------</td>
<td>----------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Early Stage (ring-forms)</td>
<td>Small and large rings. Usually one chromatin dot. Sluggishly ameboid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(b) Late Stage</td>
<td>Oval or round. Chromatin in coarse granules or irregular clumps. Band forms often seen</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizonts</td>
<td>Red cell enlarged. Contour of parasite irregular. Chromatin (nuclei) in 2 or more masses</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nearly fills red cell which is normal sized or smaller. Cytoplasm of parasite rounded and deeply pigmented. Nucleus in state of division</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Not usually seen in peripheral blood</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Merocytes</td>
<td>Fill greatly enlarged red blood corpuscle. 12 to 24. merozoites (usually 18 to 20) irregularly arranged about a mass of pigment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Almost fill a normal sized red blood corpuscle. 6 to 12 merozoites (usually 8 to 10) arranged like the petals of a flower surrounding a central pigment mass</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Not usually seen in peripheral blood. Fill two-thirds to three-quarters of red blood corpuscle. 8 to 36 merozoites (usually 18 to 24) arranged about a central pigment mass</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gametocytes</td>
<td>Round to oval and fill the enlarged red blood corpuscle. Chromatin undistributed in cytoplasm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Round and fill the normal sized red blood corpuscle. Chromatin undistributed in cytoplasm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Crescentic or kidney-bean in shape. Usually apparently free in blood. Chromatin undistributed in cytoplasm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Round and fill about three-quarters of the enlarged red blood corpuscle. Chromatin undistributed in cytoplasm</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
gametocyte and is a typical crescent with sharply rounded or pointed ends.

A frequent source of confusion in the microscopic search for malaria plasmodia in both fresh blood and stained films is blood platelets. This is particularly so with regard to the younger stages of the parasites. Figure 27 shows a cluster of blood platelets and two individual platelets superimposed upon a red blood cell. They frequently show bizarre forms and the edges are apt to be more or less fuzzy.
CHAPTER VI

THE MALARIA FEVERS

SPECIFIC FORMS AND THEIR DISTRIBUTION

Of the three generally recognized malaria fevers of man, the benign tertian type is the most widely distributed, occurring between 45 degrees north and 45 degrees south latitude. Quartan malaria is restricted in its range, locally as well as regionally. In some areas P. malariae is very rare, while in others it is the prevailing parasite. Malignant tertian, or estivo-autumnal malaria is essentially a tropical or subtropical disease although P. falciparum, the causative organism, may be transmitted for a time in cooler areas of the temperate zone if suitable conditions obtain and appropriate species of anopheline mosquitoes are present. The distribution and intensity of malarias in general in the southeastern United States is shown in Figure 28.

Malignant tertian malaria is seldom endemic in regions in which the average summer temperature is lower than 70° F. or the average winter temperature colder than 48° F. It occurs commonly in the Mediterranean region and in the tropical and subtropical belts of both hemispheres. P. ovale has been found by reliable research workers in widely separated countries. More information, however, is necessary before its clinical status as a new malaria disease will be generally accepted.

EPIDEMIOLOGY AND PATHOGENESIS

An understanding of the epidemiology of malaria requires a detailed knowledge of the life cycles of the various species of
plasmodia involved in its causation (Chapter V) and the factors concerned in transmission and infection. The latter will be discussed briefly with reference to the progressive growth of malaria plasmodia in the primary and secondary hosts (mosquito and man) and to the reactions of the secondary host (man) to the presence of the parasite.

DEVELOPMENT OF PLASMODIA IN THE TRANSMITTING MOSQUITO

In nature only certain anopheline species transmit malaria plasmodia, and according to all authorities one or more of these species must be present in any locality in order for transmission to take place regularly. Unless favorable temperatures prevail, plasmodia do not develop in the transmitting mosquito. In mosquitoes kept continuously at low temperatures, the parasites remain dormant, but they may retain their viability for weeks and even months. The infection is not passed on to offspring. Sporozoites, under normal conditions, have been found to be infective
THE MALARIA FEVERS

for as long as ninety days. The optimum temperatures for development are for:

\( P. \text{ vivax} \): 25° C.; complete development requires about eleven days at this temperature.

\( P. \text{ malariae} \): 22° C.; development is completed in from eighteen to twenty-one days.

\( P. \text{ falciparum} \): 30° C.; development is completed in ten or eleven days.

Humidity, also, is a most important factor in the development of plasmodia within the mosquito, as well as in the breeding of these insects and their biologic activities. High relative humidities approaching saturation provide a favorable atmosphere for the plasmodia.

The percentage of infected anophelines present in a locality is of great epidemiologic importance; it need not necessarily be high where malaria fevers are common, provided conditions are such as to facilitate contacts of susceptible anophelines with malaria patients and carriers of gametocytes. In some persons, however, successful transfer of the infection to anophelines does not appear to take place readily.

The particular habits of anophelines with regard to breeding places and blood-sucking influence the transmission. Thus, \( A. \text{ maculipennis} \) and \( A. \text{ quadrimaculatus} \), the most common transmitters of malaria in Europe and in the United States, are often found close to and frequenting the habitations of man. On the other hand, some species of Anopheles are more apt to be found where domestic or other animals roam, seeming to prefer their blood to that of man. Other factors which may be of importance in the transmission are the age of the gametocyte, and possibly inherent qualities of the ingested blood, or the condition of the mosquitoes at the time of ingesting gametocyte-infected blood.

The success of mosquitoes as efficient transmitters of malaria depends on a wide variety of factors, such as: (a) the location of breeding places of the insects and their distance from human settlements; (b) the usual length of their flight-range; (c) their abundance; (d) the seasonal prevalence; (e) their susceptibility
to infection; (f) tendency to enter houses; (g) time at which they bite; (h) the percentage of human blood in the stomach, the sporozoite index, and sporozoite viability.

*A. crucians* and *A. punctipennis* are usually abundant within their range but seem to prefer animal blood (zoophilous). A series of transmission experiments have been reported successful with *A. crucians* but unsuccessful with *A. punctipennis*, whereas in another series a high percentage of infections was obtained with *A. punctipennis*. *A. atropos*, a brackish water species which is found along the south Atlantic and Gulf coast of the United States, has been found to be a good experimental host but has not thus far been shown capable of transmitting malaria to man in nature. For reasons little understood, *A. maculatus* in the Federated Malay States is known to be a most important vector of malaria, while *A. ludlowi* is comparatively unimportant. In Sumatra, however, the situations in this respect are reversed. As contrasted with Java, where *A. ludlowi* appears to prefer to breed in brackish water and is largely limited to the coast, this species breeds principally in fresh water in the interior of Sumatra. In the Philippines *A. minimus* is considered more important as a vector of malaria than *A. maculatus*.

Even in the same locality a change of conditions, which favors the breeding of mosquitoes, may result in converting a species previously relatively harmless into a dangerous vector. Such was the case with *A. hyrcanus* when rice cultivation was introduced into Sumatra after the first World War. This anopheline is known to breed abundantly in the rice fields of Java and other adjacent islands where it is not regarded as an important transmitter.

In fact, quoting Hackett (1937), "everything about malaria is so moulded and altered by local conditions that it [malaria] becomes a thousand different diseases and epidemiological puzzles. Like chess, it is played with a few pieces but is capable of an infinite variety of situations."

In Chapter XII (pages 254–260) a practical classification of anophelines is presented and the geographic distribution of the principal malaria-carrying species of the world is briefly discussed.
Man becomes infective for the transmitting mosquito only when mature gametocytes are present in the peripheral circulation. Craig (1940) found that about 50 per cent of persons infected with *P. vivax* and 33 per cent of those infected with *P. falciparum* show gametocytes in the peripheral circulation. Not only must mature gametocytes be present in the blood ingested by the mosquito but they must be present in a viable state in sufficient numbers to insure the successful transmission of each of the generally recognized species. Green gives the following relative figures:

- *P. vivax* ............. 1 parasite to 1000 leucocytes
- *P. malariae* ........... 1 parasite to 330 leucocytes
- *P. falciparum* ........ 1 parasite to 200 leucocytes

### REACTION OF MAN TO PLASMODIA

Repeated exposure to malaria-bearing anopheline mosquitoes in endemic areas often leads to development of immunity to particular species and strains of *Plasmodia*. Under such circumstances resistance to infection seems to increase with age, children and newcomers furnishing the greatest number of fresh infections. This immunity in man may be diminished by factors which tend to reduce individual resistance, such as fatigue, exposure, starvation, mental anxiety, and strain. Immunity against one species may be largely ineffective against another species or even against another strain of the same species. In fact, it has been shown that different strains of the same species tend to vary in virulence.

Intertransfer of plasmodia between man and the lower animals is apparently an uncommon occurrence. It has proved difficult to inoculate even the higher apes with man's malaria plasmodia. The only successful transfer in the reverse direction has been achieved with *P. knowlesi* from the monkey to human volunteers. This recent work of Knowles and Das Gupta (1932) has been confirmed by a number of others, most of whom have employed *P. knowlesi* in the treatment of paresis. According to Milam and
Coggeshall (1938), the initial infection in man with the latter parasite confers immunity to reinfection.

Destruction of red blood corpuscles by the plasmodia results in corresponding degrees of secondary anemia which may take on a pernicious character in severe and long-continued infections. Eaton (1934) called attention to the relatively greater susceptibility of immature erythrocytes (reticulocytes) to the malaria parasite. Kitchin (1939) found that *P. vivax* has a definitely greater tendency to invade reticulocytes than mature erythrocytes. Hegner (1938) believed that this parasite has a special affinity for reticulocytes and that *P. falciparum* and *P. knowlesi* seem to prefer mature erythrocytes. Leucopenia is evident except at the time of the paroxysm, and the mononuclears are usually relatively increased in number. Other significant blood features are central paleness of uninfected erythrocytes, poikilocytosis, phagocytes containing pigment and occasionally plasmodia, and free pigment. These features, while not so constant or prominent in the peripheral circulation, are always present in blood obtained by splenic puncture. The urine shows little of significance except the presence of hyaline and granular casts in pernicious *P. falciparum* infections and occasionally also in severe tertian and quartan infections.

During the uninterrupted infection with repeated paroxysms, the principal gross pathologic changes are: (a) enlargement of the spleen, which at times may be considerable (in fresh infections this organ is soft, tending to become harder with repeated paroxysms); (b) enlargement and congestion of the liver; (c) congestion of the kidneys, meninges, brain, and bone marrow.

The capillary vessels of all these organs are frequently filled with plasmodia-bearing corpuscles, melanin, and a yellow pigment consisting of altered hemoglobin which, however, is not confined to the vessels. In subtertian malaria particularly, capillaries may become completely occluded with such materials, as well as with degenerated erythrocytes and plasmodia associated with increased mobilization and infiltration of histiocytes. In long-continued infections the spleen becomes markedly enlarged and firm; the liver
is enlarged, and pigmentation is present in various organs, including the brain. These comprise only the principal pathologic features to be found in malaria fevers. For further information, the reader is referred to more detailed works on malaria.

**Symptomatology**

**Incubation Period.** The period of incubation of the malarias varies greatly with the resistance of the individual and the number of sporozoites introduced by the infecting anophelines. The period for *benign tertian* is about two weeks, for *quartan*, three weeks, and for *malignant tertian*, two weeks or less. These incubation periods, however, may be much longer owing to a tendency toward latency and to the effect of factors increasing the host's resistance. In patients with paresis, who are inoculated with *P. vivax*, the incubation periods vary from five to thirty-one days.

During the past decade, various investigators have accumulated evidence suggesting an initial extra erythrocyte development in man of naturally inoculated sporozoites. Such a phase intervening between sporozoite and trophozoite was hypothecated by James (1931). The incubation period in man following natural inoculation with sporozoites is called *intrinsic* to distinguish this phase from *extrinsic incubation* which applies to sporogony in the mosquito until its bite becomes infective for man. If the existence of an extra erythrocytic developmental phase in man proves to be true, we might perhaps be able to explain in part at least, inability hitherto of malariologists to confirm Schaudinn's claim (1902–03) to have observed penetration of erythrocytes by sporozoites *in vivo* and likewise the failure of quinine and similar drugs as total specific causal prophylactic agents. In Figure 64 is pictured graphically the present status of our knowledge regarding an intrinsic extra erythrocytic phase in naturally acquired malaria in man.

**Onset.** Prodromal symptoms, such as malaise, vague muscular pains, headache, and anorexia, may arise; sometimes there is a tendency to periodicity in their occurrence. Paroxysms occur only when the number of parasites that sporulate simultaneously is sufficient to release enough toxic material to cause symptoms;
when smaller amounts of poison are liberated, a mere suggestion of an attack of "ague" may be manifested. In many cases the onset may be sudden, with scarcely any prodromal symptoms. The malaria paroxysm is more likely to occur between midnight and noon or early afternoon than later in the day.

Multiple and Mixed Infections. In highly malarious regions, multiple and mixed infections occur not infrequently. Thus, a double tertian or perhaps a triple quartan may give rise to so-called "quotidian fever." In Figure 29 is shown diagrammatically the relationships of two generations of *P. vivax* giving rise to a daily fever. Often benign and malignant tertian, and less frequently benign tertian and quartan, occur in the same person. It is possible that on rare occasions all three species of malarial parasites may be present in an individual's blood at the same time.

Except for a difference in periodicity, the paroxysms of benign tertian and quartan malaria are very much alike clinically. After a day or two of general malaise, muscular pains, headache and
possibly anorexia, the typical attack commences, usually consisting of cold, hot, and perspiration (chills, fever, and sweating) stages.

**Cold stage.** The patient shivers violently, the teeth chatter, the skin is pale, blue, and has a "goose flesh" appearance. There is a feeling of anxiety, with hurried breathing, and perhaps nausea and vomiting. Surface temperature is low and the superficial capillaries are extremely contracted. The urine is copious, pale in color, and of low specific gravity. Duration of this stage is from a few minutes to an hour or more, and toward the termination of the period the temperature may have risen from 103° to 106° F.

**Hot stage.** The onset of this stage may be gradual or sudden. The skin becomes hot, red, and burns. There is noticeable throbbing of the carotid and temporal arteries with intense headache, and even delirium may ensue. The pulse is rapid and full, the temperature sometimes reaches 106° F. or higher. Labial herpes is common, and epistaxis or diarrhea may occur. The urine is scanty, of a high specific gravity, contains a large quantity of urea, urates, and frequently albumin. Towards the end of this stage, nausea and vomiting disappear and the temperature begins to fall. Enlargement of the spleen can often be demonstrated.

**Sweating stage.** This stage lasts from one to several hours. Sweating frequently begins at the roots of the hair but soon becomes general and profuse. The urine continues to be scanty and of high specific gravity, the urates becoming more abundant than the urea. Gradually the temperature returns to normal, the pulse becomes softer, and the spleen diminishes in size. Although the patient then feels more or less normal, an anemic appearance persists. The average duration of defervescence is generally from two to four hours.

Without treatment the whole paroxysm, which in tertian and quartan malaria may last from ten to twelve hours, may recur at regular intervals or in cycles as indicated for the various types in Figures 23–25, and 29. Paroxysms are said to "anticipate" when they occur earlier each succeeding time, and to "postpone" when they come on a little later. As a result of these tendencies marked
variations from the typical clinical picture may arise, particularly in subtertian or tropical malaria.

REMITTENT AND IRREGULARLY INTERMITTENT TYPES OF PAROXYSM

Paroxysms remittent or intermittent in character are generally subtertian. They are often ushered in by a slight chilliness instead of a frank chill; this is followed by a prolonged and intensified hot stage, absence of marked terminal sweating, and a tendency to continuous, remittent or irregularly intermittent fever. A characteristic feature is the patient's failure to feel relief during periods of abatement of fever.

At times the temperature may fall to normal during the initial attack, but in succeeding attacks it tends to remain above normal, often showing a tertian periodicity in such exacerbations of the fever. Rise in temperature is usually gradual and subsidence is by lysis rather than by crisis as in benign tertian and quartan fevers. The paroxysm may last from twenty to thirty-six hours. A new paroxysm is likely to come on before the tertian period of forty-eight hours has expired, and having appeared, tends to delay its termination. Owing to the extreme irregularity of the paroxysms thus developing, the term "dumb fever" is often applied to this syndrome.

PERNICIOUS MANIFESTATIONS

These manifestations of malaria are due almost entirely to *P. falciparum* and fortunately are comparatively rare in properly treated cases. They occur chiefly in the tropics among persons who are exposed to extreme hardship or whose resistance has been reduced by intemperance or previous attacks of malaria or other debilitating disease. The onset is usually sudden, and the condition is always one of great gravity. The principal forms recognized are:

*Hyperpyrexial.* The temperature may rise to 107° F. and often considerably higher.
**THE MALARIA FEVERS**

**Cerebral.** This type is due to occlusion of cerebral capillaries by plasmodial and histiocytic emboli. Comatose, convulsive, and paralytic forms due to the accompanying anemia of parts affected have been described.

**Algid.** There is no febrile reaction after the cold stage; the chill terminates in collapse. This is a most serious condition.

**Choleraic and dysenteric.** Bile is conspicuously present in the feces.

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**Malaria Cachexia**

Patients who have suffered repeated or long-continued attacks of malaria fever, and those who have resided in the tropics for a prolonged period, may develop cachexia. This condition is characterized by: (a) anemia, which is often intense, the skin being of a sallow ashen hue; (b) a tendency to hemorrhage, such as epistaxis, retinal hemorrhage, purpura; (c) irregular attacks of fever; (d) great enlargement of the spleen and, to a lesser degree, of the liver.

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**Blackwater or Hemoglobinuric Fever**

Blackwater fever occurs almost exclusively among Caucasians who have lived in tropical malarious regions for prolonged periods, and who have suffered repeated attacks of malaria. Its etiology is uncertain, but many investigators associate the condition with the previous history of malaria, although plasmodia may be absent or scanty during an attack or in the internal organs after death. Blood hemolysis occurs extensively, numerous casts being found in the renal tubules and hemoglobin in the urine. Some authors attribute the hemolysis to long-continued use of quinine in the treatment or prophylaxis of malaria, but blackwater fever also occurs in untreated cases.

One attack of blackwater fever seems to predispose to another. Certain it is that an unusual instability of erythrocytes exists. Frequently the condition appears to commence as an ordinary attack of malaria fever, but this is followed in a few days by
a severe chill associated with very dark or even black urine. The urine, at first copious, tends to diminish in quantity and may even be completely suppressed. In the latter case, the kidney tubules are often found at autopsy to be completely plugged with erythrocytic debris. Other signs and symptoms are yellow discoloration of the sclera and the skin, frequently associated with bilious vomiting and diarrhea and sharp pain in the epigastrium and loins.

The signs and symptoms of blackwater fever may subside in a few hours, but more often recurrences take place with each attack of fever or there is no remission at all. While the individual is in the malaria zone, the slightest chilling or overstrain may incite an attack, and therefore both treatment and prophylaxis require that the patient be removed to a temperate climate when circumstances and conditions permit.

**Clinical Diagnosis**

In the bedside diagnosis of malaria fever, the previous history of the patient, the character of the fever (particularly its periodicity), and the amount of splenic enlargement should be given careful consideration. Tertian and quartan periodicity are not observed in any other disease. In malignant tertian infections, however, the fever may be most irregular in character; at times there may be little or no fever.

Enlargement of the spleen is a clinical sign common to all malaria fevers. In long-standing infections the spleen may be very large, occupying the greater part of the abdominal cavity ("ague-cake"). In early, mild, and sometimes even in severe infections the spleen may not be measurably enlarged. A palpable spleen may be due at times to some other and unrelated cause.

Suggestive diagnostic features of the malarias are: (a) response to drug therapy, particularly to quinine; (b) the presence of melaniferous leucocytes; and (c) a relatively high large mononuclear count in the presence of leucopenia. Any one of these features is suggestive and the presence of all three or even two at the same time should lead one to suspect the presence of one or more of the human malaria plasmodia.
Differential Diagnosis

The most reliable method for making an accurate diagnosis of malaria fever is by the demonstration of specific plasmodia in the blood or in material obtained by splenic puncture. For detailed features of the human malaria plasmodia, the reader is referred to the preceding chapter. The salient points in this connection are summarized in Table VIII. Even in tertian and quartan infections, in which the symptoms are often clinically diagnostic, confirmation should be sought by finding the specific parasite in the blood.

Among serological tests, complement fixation and precipitin tests have been advocated for diagnosis of malaria and also a pigment flocculation and protein tyrosin test. Thus far, however, none of these tests have been shown to be of much practical value and ordinarily would seem unnecessary refinements.

Clinically differential diagnosis of the malarias must be made from various other disease conditions which at times may resemble malaria in their symptomatology. This entails a fair knowledge of all fevers, nontropical as well as those considered specifically tropical. Manson-Bahr (1936) points out that previous recurrent malaria attacks may predispose to periodicity in other fevers occurring subsequently. Further details may be found in the clinical treatises on tropical diseases.

Treatment

About three hundred years ago, cinchona bark containing quinine and other related alkaloids was found to be effective in treating malarias. Ever since then, this bark or its extracted quinine has been widely employed in controlling the clinical manifestations of malaria. Recently, however, synthetic substances have been produced which in some respects appear to surpass quinine. These therapeutic agents are plasmochin and atabrin. The salts of quinine generally employed are the sulphate and the hydrochloride, the former being administered by mouth and the latter by mouth or by intramuscular or intravenous injection in the form of the bihydrochloride.
**Table: Differential Table of The**

(Courtesy of Army Medi-

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<thead>
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<th>Parasitized Red Cells</th>
<th>Schizogony</th>
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<tr>
<td><strong>P. vivax</strong> (tertian or benign tertian)</td>
<td>Unstained</td>
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<td><strong>P. malariae</strong> (quartan)</td>
<td>Unstained</td>
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<td>Stained</td>
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<td><strong>P. falciparum</strong> (malarial, malignant tertian, subtertian or pernicious tertian)</td>
<td>Unstained</td>
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**Routine Treatment**

As a routine method of treatment for the average malaria infection, the National Malaria Committee (1940) has recommended the following procedures:

1. **For the acute attack:** 10 grains (0.65 gm.) of quinine sulphate by mouth, three times a day, for a period of three or four days, or until the acute symptoms have disappeared.

2. **Thereafter for a period of eight weeks:** 10 grains of quinine sulphate every night.
For individuals who do not have symptoms at the time, but show evidence of the presence of parasites, only the nightly administration of 10 grains of this drug for eight weeks is considered necessary.

In pernicious malarial infections, quinine dihydrochloride should be administered intravenously in doses not to exceed 20 grains (1.3 gm.). Ordinarily, however, from 10 to 15 grains of this salt of quinine are sufficient at a single dose.

(3) When gametocytes are present, the use of quinine as out-
lineal above should be supplemented by plasmochin, a derivative of quinoline, which destroys these forms, rendering the individual noninfective to anophelines. After the symptoms have been controlled by quinine, it is considered good practice in every case to administer plasmochin in doses of \( \frac{1}{6} \) grain (0.01 gm.), three times a day, for a period of five or six days, the usual treatment with quinine being continued at the same time.

**ATABRIN**

Atabrin is a derivative of acridine, which has proved to be rapidly curative. Relapse occurs less often after its use than after the administration of quinine. Atabrin is marketed in 1.5 grain (0.09 gm.) tablets. The adult dose is 1 tablet, three times a day, for five successive days; the dose for children from one to four years of age, is \( \frac{1}{2} \) tablet (0.75 grain) twice a day for five days; for children from four to eight years of age, 1 tablet, twice a day, for five days. In relapsing cases this treatment may be continued for two days longer, but it is recommended that a second course of atabrin treatment should not be given before thirty days have elapsed in order to prevent too high a concentration of this drug in the body.

Although atabrin destroys both schizonts and merozoites of the various malaria plasmodia, it cannot be relied upon to kill gametocytes, particularly those of *P. falciparum*. Therefore, in atabrin therapy, the use of plasmochin simultaneously or subsequently * is indicated, in the same dosage that is employed when this drug is given in conjunction with quinine. Intravenous use of atabrin is considered hazardous because of the danger of too rapid concentration in the blood. However, the intramuscular injection of atabrin solution is reasonably safe and does not cause necrosis at the site of injection, as quinine frequently does. Atabrin followed by plasmochin is considered by many as the most efficient remedy in preventing relapses of quartan and subtertian malaria.

* Some clinicians feel that the simultaneous administration of atabrin and plasmochin seems to aggravate the toxicity of each. It has been stated that such toxic manifestations are prevented if the patient has had, just previously, a full meal. Therefore, it may be best to administer only quinine until food can be taken. Atabrin followed by plasmochin is considered by many as the most efficient remedy in preventing relapses of quartan and subtertian malaria.
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is preferred to quinine in the treatment of blackwater fever because of the greater tendency of quinine to incite hemoglobinuria. A side-effect of atabrin in some persons is lemon yellow coloration of the skin, but this is not a toxic symptom nor is it necessarily indicative of injury to the liver, as has been feared by some persons.

Prognosis

Generally speaking, malaria infection is much more debilitating in children, women, and undernourished and weak individuals. The severity of infection as a whole depends largely on the specificity of the plasmodia, malignant tertian (P. falciparum) being the one to be most feared per se. Although the Caucasian may suffer from repeated relapses, the general expectation of life is good, provided that adequate early treatment is given and the stability of the erythrocytes has not been seriously impaired. However, it must be remembered that the mortality in the pernicious type, even when properly treated, may be as high as 25 per cent.

Prophylaxis

The control and prevention of malaria may be summarized as follows: (a) destruction of anopheline larvae and adults; (b) protection of the individual from bites of anopheline mosquitoes; (c) chemotherapy.

Destruction of Mosquito Larvae and Adults

Interrupting the life cycle of the mosquito while it is in the larval stage constitutes the keystone of malaria prophylaxis. Accordingly an intimate knowledge of the breeding habits of anophelines is quite as important as specific identification of the adults. Without such knowledge and a "sleuth-like" epidemiological sense, success in the control and elimination of specific malaria-bearing anophelines, although adequately financed and vigorously pursued, may be more or less futile. Hackett (1937) in his admirable discussion of "Malaria in Europe" cites various most enlightening examples bearing on these brief statements. To any
one proposing to engage in mosquito control, a most careful study of this monograph would be of the greatest assistance.

Elimination of breeding places may be accomplished by filling-in pools and making ditches with smooth sloping sides to carry away the water. When practicable, permanent measures are to be preferred. Associated agricultural developments aid greatly in lessening the cost of drainage of swamps and similar breeding places. Subsoil drainage is preferable as it tends to minimize the luxuriant growth of grass and weeds which favor the sluggish circulation of water.

In lieu of filling-in or drainage, oiling the surface of the water is a very useful procedure. The application of one-half pint of crude petroleum to each 100 square feet of surface, repeated according to specific conditions, is recommended. Cresol has also been found effective as a larvicide and is preferable to crude oil under certain circumstances.

Where oil is ineffective, Paris green mixed with dust is being extensively employed. This larvicide is scattered in such a way as to form a minute surface deposit. To accomplish this rapidly and on a large scale, an airplane flying low has been used for its dissemination when circumstances and conditions permitted.

Certain kinds of top-feeding minnows, such as species of the genus *Gambusia*, are natural enemies of mosquito larvae. These have been employed successfully in keeping down the growth and development of the larvae into adults.

The clearing away of brush, grass, and undergrowth from the vicinity of houses and other buildings exposes approaching mosquitoes to the heat of the sun to which they soon succumb. Inside the house, where anophelines may hibernate during winter, these insects may be killed by sulphur fumigation (1 or 2 pounds of sulphur for each 1000 cubic feet, the effective time of exposure being about two hours). The burning of pyrethrum powder (2 pounds per 1000 cubic feet) is also a useful procedure in enclosed spaces. An excellent alternative method of applying pyrethrum powder is spraying with "Giemsa spray" diluted with twenty parts of water. The formula is as follows:
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Pyrethrum tincture .................. 480 gm.
Potash soap, odorless ................ 180 gm.
Glycerine .......................... 240 gm.

PROTECTION OF THE INDIVIDUAL.

Screening to prevent entry of any mosquitoes into closed spaces occupied by man constitutes the most effective and practical method of protecting the individual directly from mosquito bites. Avoidance of exposure to mosquitoes, particularly after sunset or just before sunrise (anophelines bite mostly at these times), is an excellent practice in endemic areas.

Houses should be as far removed from native quarters as is practicable. The upper floors should be reserved for sleeping quarters, since anophelines usually do not fly high above the ground level. At night the use of mosquito nets and adequate protection of the face, hands, and ankles from mosquito bites are important accessory protective measures.

The fabric employed in making window and door screens and mosquito nets should have eighteen meshes to the inch in order to be effective against mosquitoes and at the same time permit the free exchange of air between the enclosed space and the outside. Screened doors should always open outward and close automatically by means of spring hinges. All external openings must be included to make the screening secure.

Mosquito nets for the beds should always have the borders tucked well under the mattress. Persons thus protected should endeavor to avoid contact with the net while sheltered beneath it. As mosquito repellents, the following substances have been found useful: (a) oil of citronella smeared over exposed areas; (b) epsom salts (1 ounce dissolved in 10 ounces of water) daubed on exposed parts and allowed to dry (sometimes referred to as Neal's method); (c) proprietary repellents, those containing oil of pine being fairly satisfactory.

From a practical standpoint, direct protection of the individual against malaria includes the use of some form of chemotherapy as a prophylactic agency. Such a means of protection must not be
accepted, however, as a substitute for the more effective methods of destruction of the transmitting agent and shielding against its bite.

QUININE PROPHYLAXIS

The most that can be said for use of quinine prophylactically is that when administered according to the most approved methods, it appears to hold in check, temporarily at least, the vigorous multiplication of malaria parasites. Consequently, quinine prophylaxis may be very valuable on exploring trips and expeditions where illness of personnel may be most detrimental. The principal objection to its employment in this way is that protracted administration of the drug may induce quinine-fastness of the strain of plasmodia harbored. Thus, the individual will benefit less from its use as a curative agent.

In east and west Africa, and other tropical localities where systematic quinine prophylaxis has been practiced for many years, the following methods of administration have been widely used: (a) the administration of 5 grains of quinine hydrochloride or sulphate in solution daily, every evening, or morning if preferred; (b) the administration of 15 grains of quinine hydrochloride (preferably in divided doses) twice or thrice weekly at regular intervals, or on successive days at the middle or the end of the week.

Yorke and Macfie (1924) concluded that experimentally quinine does not prevent malarial infection but its continued use for ten days to two weeks after exposure to infecting mosquito bites always prevents any subsequent malarial paroxysms. The experience of the United States Army in Panama and the British Army in Macedonia during the first World War are in accord with these conclusions. Wenyon, according to Strong (1942*), at a British symposium on malaria in war (1939), stated as his opinion that prophylactic use of quinine prevents actual attacks of malaria although it does not prevent infection. The varying results published regarding the prophylactic use of quinine may be due to such factors as: (1) varying susceptibility of individuals in
different communities and countries; (2) diverse infectivity rates of mosquito hosts in different localities; (3) extent of exposure to mosquito bites.

**PLASMOCHIN FOLLOWING QUININE AND ATABRIN IN PROPHYLAXIS**

Clark and Komp (1939), after eight years observation on the prophylactic use of plasmochin, following quinine or atabrin, found the results were equally good provided the same degree of attention was given to their administration. The doses employed for the drugs and the time intervals between administrations were:

1. For quinine in the form of sulphate, 1 gm. doses daily for five days followed during the succeeding week by 0.01 gm. (1/6 grain) doses of plasmochin twice daily for five successive days.
2. For atabrin 0.1 gm. (1 1/2 grains) doses three times daily for a period of five days followed by 0.01 gm. doses of plasmochin three times daily for a succeeding period of five days.

Although relapses following the use of both these methods were not uncommon, it is claimed that clinical illness from malaria was greatly lessened. Complete eradication of the disease was not realized, and it was thought doubtful if such a result would be possible under conditions existing in Panama, namely: the ever-present anopheline, the newly arrived carrier, and the all year transmission period.

In general, there is no unanimity of opinion as to whether quinine or atabrin is the more valuable prophylactic agent. However, most authorities agree that plasmochin has an almost specific effect upon mature gametocytes. Often in experimental infection James et al (1932) believed that atabrin will postpone a malarial attack for as long as twenty-eight weeks. Among advantages claimed for atabrin alone in contrast to quinine are that it can be more easily dispensed in rural districts and that the time element required is much shorter.

Strong (1942) tersely sums up the question of chemoprophylaxis in malaria in the following words: "While it is true where only benign malaria exists, or where there is satisfactory means of carrying out diagnosis and treatment, quinine and especially
atabrin prophylaxis is not to be recommended, yet in intensely malarious districts, with heavy infections of natives, it is advisable for the European (or American) when actually exposed to infection to take from 5 to 15 grains of quinine every day in tropical regions where malignant tertian is a menace." One must not, however, accept this in lieu of other obvious means of prophylaxis which may be applicable such as the various means of screening, particularly at night.
Ciliates comprise those protozoa which are covered by hairlike processes known as "cilia." These serve both for locomotion and capture of food. They usually have two nuclei, a large one (the macronucleus) and one or more small ones (the micronuclei); one or more contractile vacuoles also may be seen. *Balantidium coli* (Malmsten, 1857) Stein, 1862, is the only species of importance which is generally accepted as being pathogenic for man, although others have been reported in human fecal material from time to time. This ciliate is a comparatively rare parasite of the human colon, but a very common one in the pig, which is probably the usual source of man's infection.

**Pathogenicity**

The habitat of *B. coli* is primarily the lumen of the colon, but invasion of the mucosa and submucosa, particularly of the cecum, occurs in man. The resultant ulcers are often indistinguishable clinically from those of *E. histolytica* infection. In acute cases, dysenteric symptoms arise, but in chronic ones the abdominal discomfort seems to be unrelated to any very definite symptomatology.

**Morphology**

*B. coli* (Fig. 30) is the largest protozoan parasite of man, the trophozoite usually measuring from 60 to 100 μ in length and from 50 to 70 μ in breadth. It is roughly ovoid in shape, the anterior end being less blunt than the posterior. A comparatively large funnel-shaped mouth or "peristome" indents the anterior end.
and a small slitlike anus or "cytopyge" subterminally indents the posterior. The body surface is covered with oblique, parallel rows of cilia, which are greatest in number and length around the peristome. The peristomal cilia move rhythmically toward the "cytostome," creating currents which aid in the capture and ingestion of food. Besides the two contractile vacuoles, the endoplasm contains a varying number of food vacuoles.

The two contractile vacuoles are situated near the periphery of the body, one usually just anterior to the middle and the other close to the posterior end. These vacuoles pulsate at regular intervals, the posterior one, it is thought, emptying its contents into the cytopyge or anus through a connecting tube. Within the middle third of the endoplasm is situated the large kidney-shaped macronucleus. The micronucleus, with a prominent centrally placed karyosome, lies close to the concavity of the macronucleus. The micronucleus usually stains more or less deeply and at times appears to contain from five to ten chromosome-like masses and chromatin granules, the exact character and function of which is not known. A neuromotor mechanism, believed to serve as a co-
A PATHOGENIC CILIATE: BALANTIDIUM COLI

ordinator of feeding and swimming movements, has also been described.

LIFE HISTORY

The vegetating trophozoites of B. coli live and feed in the lumen and tissues of the colon. They are derived from cysts and possibly from trophozoites ingested with food and drink, or are transferred to the mouth through contaminated hands, utensils, and the like. Details regarding encystment are not well understood. It seems probable, however, that encystment occurs from time to time to facilitate the transfer of the organism to fresh hosts and to insure the perpetuation of the species.

Ordinarily, reproduction is by transverse fission, although conjugation has also been reported to occur in this species. In maturation divisions and the production and exchange of the pronuclei, conjugation is essentially similar to that of ciliates in general. The cysts (Fig. 30) of this organism are roughly spherical in shape, measuring from 50 to 60 μ in diameter. They resemble, in general, rounded trophozoites surrounded by a double-walled translucent covering. Each cyst contains a single parasite and upon excystation only one daughter B. coli results. Conjugation of trophozoites in feces has been reported. Cysts containing two organisms have also been observed; it is thought that these may be the result of an encystment of conjugating parasites.

CULTIVATION

B. coli has been successfully cultivated at 37° C. on liquid media containing inactivated blood serum or Loeffler's dehydrated blood serum. Maximum growth occurs in from two to three days. It is said that conjugating trophozoites commonly occur in culture, but cysts have been reported only in culture of guinea pig balantidia.

EPIDEMIOLOGY

Infection in man is very frequently associated with a history of direct contact with pigs, particularly during their slaughter. It seems probable, therefore, that transmission of infection to man
may result from direct transfer of trophozoites, since these organisms may live at room temperature for as long as ten days. It has been reported that trophozoites are capable of passing unharmed through the stomach and small intestine and into the cecum of guinea pigs. Encystment is not frequent in the pig and is comparatively rare in man. The cysts are very resistant under moist conditions, but are quickly killed by drying or by direct sunlight.

According to reports in the literature, the cat and the monkey have been infected with *B. coli* from man, but thus far attempts to transmit to man *Balantidia* from the pig or the monkey have been unsuccessful. In the latter instance, failure seems to indicate a high resistance normally of man to infection with *Balantidia* from these animals. On several occasions large numbers of cysts were given to human volunteers, with entirely negative results. It may be, as shown by Nauss and Rappaport (1940) in the case of *E. histolytica*, that accessory factors which cause injury to the mucosa and thus lower its resistance to invasion may also play an important determinant role here as well.

**Pathology and Symptomatology**

Although *B. coli* may live in the human colon without producing any suggestive clinical signs or symptoms, a dysentery which is indistinguishable from amebic dysentery occurs in most cases of chronic infection. In pigs and monkeys, as well as in man, the resultant condition may at times terminate fatally. Ordinarily, persons harboring this parasite, have some diarrheic symptoms. In the more persistent and severe cases, there is evidence of the presence of *Balantidia* in the mucosa or even the submucosa, and also at times in the muscular layers of the colon. Even where there is no evidence of tissue invasion, the superficial mucosa may be hyperemic and show signs of necrosis and capillary hemorrhage.

Within the tissues, *Balantidia* multiply as they do in the lumen of the colon and produce small abscesses which lead to ulceration. The abscesses are filled with mucoid material containing numer-
A PATHOGENIC CILIATE: BALANTIDIUM COLI

ous trophic *Balantidia*. The ulcers are round, oval, or irregular in shape, with undermined edges. Their floors are covered with pus and necrotic material, which contain *Balantidia*. These ulcers resemble very closely the ulcers produced by *E. histolytica*.

The mucosa between ulcers may appear normal, swollen, or hyperemic, even hemorrhagic in places. Ulcers may communicate with one another in various ways, as they do in amebiasis. Histologically, sections show round cell infiltration and coagulation necrosis in the walls of the abscesses and ulcers. Other microscopic features of tissue sections are hemorrhagic areas with *Balantidia* (frequently in nests) in the tissues, or in the capillaries, lymph channels, and neighboring lymph glands.

While some individuals harboring *B. coli* in the colon present no symptoms, the majority complain of typical diarrhea or dysentery characterized by abdominal colic, tenesmus, and, at times, nausea and vomiting. Loss of appetite and weight, insomnia, and muscular weakness are some of the other symptoms to be expected. Usually, physical examination reveals anemia of the mucosa and skin and more or less tenderness over the colon. In severe cases, the stools may contain an abundance of bloody mucus, while in others constipation may be present.

**Diagnosis**

Specific identification of the disease is dependent upon positive microscopical demonstration of *B. coli* in freshly passed feces. The usual diarrheic or dysenteric stool contains motile trophozoites while semiformed and formed stools may show cysts. The methods of examination which are recommended are those already described for intestinal amebae and flagellates.

**Treatment, Prognosis, and Prophylaxis**

There is no specific therapeutic agent for the treatment of *B. coli* infections. Protargol and other organic silver compounds, and methylene blue (1 to 3000) given as enemata have been recommended. Methylene blue given by mouth has been advocated by some clinicians. Carbarsone has been successfully em-
ployed, administered as for amebiasis. Emetine appears to be of little value in treating balantidial infection.

In healthy persons, the infection tends to disappear spontaneously, while in debilitated individuals it may be serious and even fatal. Proper treatment will eliminate most infections and clear up the carrier state.

Methods for the control and prevention of balantidial infections are essentially the same as those recommended for amebic infections. In addition, pigs should be considered as an important source of human infection. Consequently, contact with these animals and contamination of man's drink or food with their feces should be given primary consideration by the sanitarian.
PART TWO

WORMS PARASITIC IN MAN
Worms parasitizing man belong almost exclusively to either the roundworms (NEMATHELMINTHES) or the flatworms (PLATYHELMINTHES). The former are unsegmented, lack special articulated organs of locomotion, but possess a protective cuticle composed, so far as is known, of an albuminoid material allied to collagen and gelatin or to the keratins. The flatworms may be segmented or unsegmented; they lack special organs of locomotion and a general body cavity, and possess a comparatively soft integument. The great majority of the roundworm infections of man (with the exception of trichinosis * and filariasis) are associated more or less directly with pollution of the soil by fecal excrement. Flatworm infections, on the other hand, result most frequently from eating food infested by the larval forms of the parasite. *Hymenolepis nana* is one of the important exceptions.

*Enterobius (Oxyuris) vermicularis* (Linnaeus, 1758)
Leach, 1853

**Distribution.** This worm, popularly known as the pinworm or seatworm, is one of the most common and widely distributed of the human parasitic worms. Surveys made under the auspices of the National Institute of Health showed an incidence of 85 per cent in 600 persons examined. Among various similar surveys the results have ranged from 10 or 15 per cent to 50 or more per cent infection. An improved method of collecting the eggs by

*Trichinella spiralis* by reason of its epidemiologic similarity to meat-infesting worms, and trichinosis will be described in Chapter IX. The important group of filarial worms and the filariases will be discussed in Chapter X, with the *Schistosoma* (sexually differentiated parasitic flatworms) and the schistosomiases.
means of cellophane swabs was employed. A follow-up of the families of persons found harboring these worms revealed that three-fourths of the family groups were infected. Among negroes, oxyuriasis appears to be less common than among whites.

**Life Cycle.** Figure 31 shows the essential details of the life cycle of this intestinal parasite. The adult gravid females work their way out of the anus to deposit their eggs on the surrounding skin. Marked itching, more pronounced at night, is an outstanding symptom.

The newly laid eggs of *E. vermicularis* develop rapidly and the mature egg contains a coiled-up embryo, within a doubly contoured shell which is somewhat flattened on one side. They measure approximately 50 μ in length by 20 μ in width. After being ingested, the embryos escape from the egg shell and develop into adult males and females, usually in two weeks or less. Copulation occurs in the small intestine, and the males, it is believed, soon disappear in the feces. The fertilized females pass into the cecum and colon where they remain until mature. They then make their way to the perianal region to deposit their eggs.

**Morphology.** The adult male pinworm is much smaller than the female, the length being about 4 mm., whereas the size of the female varies from 8 to 13 mm. in length and from 0.3 to 0.5 mm. in diameter. The anterior end, in both male and female, is slightly bulbous; the mouth opening has three retractable lips which serve as organs for attachment to the intestinal mucosa. Likewise, both male and female have a characteristic bulbous expansion at the base of the esophagus. The digestive tract is practically a straight tube.

The female pinworm is fusiform in shape and the caudal end tapers to a rather slender extremity. The caudal end of the male is somewhat bulbous, curved ventrally, and provided with a retractile copulatory spicule with an acutely curved tip. The vagina of the female opens at about the junction of the anterior and middle thirds, the anus at the junction of the middle and posterior thirds of the body. The vagina extends posteriad for some distance to meet the paired genital organs (one directed ante-
Fig. 31. Life cycle of *Enterobius vermicularis*; gravid females, × 1 app.; eggs, × 120 app. (After Kouri. In Kouri and Basnuevo: *Lecciones de Parasitología y Medicina Tropical*, 1940.)
riorly and the other posteriorly) which consist successively of
the uteri, oviducts and ovarian tubules which are coiled back
and forth several times in the middle half of the body. In the
gravid female, the uteri become so distended with eggs that they
more or less completely fill the entire body space. Pressure ex­
erted on the esophagus causes her to relax and lose her hold on
the bowel wall. Detached in this manner, the worm is transported
to the rectum and escapes through the anus, near which it is
usually crushed resulting in the liberation of many embryonated
eggs.

Laboratory Diagnosis. Laboratory diagnosis of pinworm infec­
tion depends on finding the adult gravid females in the feces or
the demonstration of the typical eggs (Figs. 31 and 41, H) in
scrapings or swabbings from the perianal region. Dirt beneath
the tips of the fingernails of infected children often contains
eggs. More rarely, eggs may be found in the feces as well. In
freshly passed feces female worms make their way to the surface
and may be recognized readily as short, whitish, threadlike ob­
jects. Directions for making and using the perianal swab devised
by Hall (1939) are given in Appendix VI.

Treatment of Infection. Since the mature adults live in the
small intestine and the gravid females inhabit the large intestine,
treatment to be effective must be directed toward dislodging the
worms from both these portions of the intestinal tract. Many
anthelmintics have been tried with varying degrees of success.

Hexylresorcinol crystoids has been found a reliable therapeutic
agent. The dose is 1 gm. for adults, 0.6 gm. for children under
school age, and 0.8 gm. for children from six to ten years old,
given on an empty stomach in the morning. The capsules or
"seal-ins" should be swallowed without chewing and should be
followed by fasting for about five hours. In the evening the
bowel is thoroughly cleaned with a warm-water enema, after
which from 250 to 400 cc. of "S.T. 37"—an alkaline solution of
hexylresorcinal (0.1 per cent)—is instilled as a retention enema,
the patient being encouraged to hold it for from fifteen to thirty
minutes. This dual procedure is designed to destroy the parasites
ROUNDWORMS AND FECAL SOIL POLLUTION

in both the small and large intestine. Two or three courses of this treatment are considered adequate to eradicate the worms, provided reinfection does not occur in the meantime.

Since the publication of the reports of Wright, Brady and Bozicevich (1938), and of Wright and Brady (1938), the oral administration of gentian violet in tablet form has been used extensively. The adult dose recommended is 1 grain, three times a day, before meals for a period of eight days (the dose for children is 1/6 grain daily for each year of apparent, not chronological, age). For adults, the treatment may be repeated after a week's rest. Ninety per cent or more of cures have been claimed and it is said that minor discomforts follow in only a small percentage of treated cases.

Prophylaxis of Infection. Control and prevention of pinworm infection are primarily matters of personal and group hygiene. Fingernails should be kept short and thoroughly cleaned, particularly before meals. Infected children should be provided with closed sleeping garments made of nonporous material to prevent contamination of fingers and bed linen. All clothing should be periodically sterilized and toilet seats must be regularly scrubbed and sterilized. Finally, periodic diagnostic check-ups should be made, both of known infected individuals and of persons immediately in contact with those infected. Successful eradication of pinworm infections demands the most persistent and meticulous care in carrying out these measures.

*Trichuris (Trichocephalus) trichiura* (Linnæus, 1771)

BLANCHARD, 1895

Distribution and Gross Appearance. This parasitic worm commonly known as the whipworm or threadworm is found in the southern United States and throughout the warmer, moist parts of the world. The infection, which occurs largely in the cecum, is persistent, but usually it does little serious damage unless a considerable number of worms are present, which is not often the case in man. The term “whipworm” is a key to identification of both the adult male and female. The anterior three-fifths of the
worm is threadlike, corresponding to the lash, while the posterior two-fifths is thick and fleshy like the handle or stock end of a whip. Males and females are about the same size—from 130 to

150 mm. long. The broad portion of the male, however, tends to be slightly smaller than that of the females and is generally coiled as shown in Figure 32.

**Morphology.** The anterior portion consists primarily of a greatly elongated neck which enables the worm to burrow beneath the mucosa parallel to the long axis of the host’s cecum or

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**Fig. 32. Life cycle of Trichuris trichiura.** The male (a) and female (b) worms (X 2.5 app.) parasitize principally the cecum (m) of man (i) their host. The fertile female (b) deposits her eggs (c, X 350 app.) in the locality (n) of her attachment to the cecal mucosa. These are carried to the external environment with the feces (h) and after a time (often considerable) embryonate (d), soon becoming infective for man.

When the fully developed eggs are ingested from the ground (i, j), directly on the hands or by means of food or drink (k, l), the embryos escape in the intestine and develop into adult male (a) and female (b) worms. These anchor themselves firmly to the mucosa of the cecum (m). The fertilized female (b) produces eggs which reach the external environment as before to begin the cycle again. (After Kouri. Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical, 1949.)
colon, in this manner securing a very firm anchorage. The larger, sausage-shaped free part of the worm contains the digestive and sexual organs, the anal opening being at the caudal extremity and the vulva at the junction of the threadlike neck and the thick posterior part of the body. In addition to coiling of the body, males may be recognized by the ventrally curved caudal end, which is equipped with a retractile copulatory spicule. The eggs are brown in color, barrel or lemon-shaped, with knob or pluglike structures at either end. As passed in the feces, they are unsegmented and measure 50 by 25 μ.

**Life Cycle.** The life history of *T. trichiuria* (Fig. 32) is comparatively simple. After the egg is passed in feces, from twelve to sixty days (depending on temperature, humidity, and available oxygen) are required for development of the embryo within the very resistant egg shell. In contaminated soil, these eggs have been known to retain their viability for as long as five years.

Man is infected by mature embryonated eggs, conveyed to the mouth mechanically in various ways, or in fecal contaminated food or water. Larvae are liberated from the protective shells by the action of the digestive juices. After a short period of development in the small intestine, they migrate to the cecum principally, and attach themselves by burrowing into the mucosa, reaching adult size in from six to eight weeks.

**Diagnosis.** The diagnosis is readily made by finding the characteristic eggs in the feces. These are generally light to dark brown in color when passed. They are scarcely larger than the eggs of *Enterobius vermicularis*. Their general appearance is shown in Figures 32 and 41, D.

**Treatment of Trichuriasis.** Anthelmintic drugs found to be effective against other parasitic roundworms have been employed with some degree of success in eradicating this worm, but treatment of light infestations is frequently ineffective. The latex, *leche de higuerón* (sap of fig tree), of certain species of the genus *Ficus* is said to be the only known therapeutic agent which will eradicate all worms, but this substance at present is not readily available commercially.
Prophylaxis of Trichuriasis. As with the other roundworms, discussion of which follow, sanitary disposal of human feces is most important in both control and prevention of trichuriasis. Measures to minimize hand to mouth transfer of eggs and to prevent direct fecal contamination of food and drink are likewise of importance in individual prophylaxis.

Ascaris lumbricoides (Linnaeus, 1758)

Distribution and Mode of Infection. This large intestinal roundworm of man is probably the most common and cosmopolitan of all human parasitic worms. It flourishes everywhere in temperate and in warm, moist climates, especially where personal hygiene and environmental conditions favor embryonation of the eggs in or on polluted soil. Infection is acquired generally through the ingestion of fully developed eggs from fecally polluted hands, food or drink. Children are more commonly infected, and pollute the soil more frequently than do adults.

Morphology. Living adult worms are pinkish, round, and long; they taper bluntly anteriorly and are more attenuated posteriorly. The head is provided with three prominent lips, one broad and dorsal, the other two somewhat smaller and ventrolateral. The "lateral lines" may usually be seen as a pair of whitish streaks running the entire length of the body. The females measure from 20 to 35 cm. or more in length. The males are smaller, measuring from 15 to 30 cm. in length and from 2 to 4 mm. in diameter. Posteriorly, the male is curved ventrally and is provided with a pair of simple copulatory spicules. The vulva of the female is situated near the junction of the anterior and middle thirds of the body. The vagina is cone-shaped and branched; to each division is attached a long coiled genital tubule consisting of uterus, seminal receptacle, oviduct and ovary. These genital tubules are coiled through the middle and posterior thirds of the worm. They may have a total length several times that of the worm and may contain a great many eggs.

The fertilized eggs (Fig. 41, C) are broadly ovoid, with a thick,
transparent shell surrounded by a coarsely mammillated albuminous outer layer which is usually bile-stained. The latter may be absent (Fig. 41, A) since it is not firmly attached. Recently fertilized eggs measure from 45 to 75 μ in length and from 35 to 50 μ in breadth. They are unsegmented and the cytoplasm contains lecithin granules when freshly passed. A single female, according to Brown and Cort (1927), may lay an average of 200,000 eggs a day. Embryonic development occurs after the eggs are passed in feces, and is dependent on various environmental factors. Unfertilized eggs (Fig. 41, B), which are usually considerably longer and thinner walled than fertile ones, are not infrequently seen in feces. Artefacts closely resembling ascaris eggs are often found in feces. The pollen grains of broccoli are sometimes confused with ascaris eggs; the former are, however, considerably smaller in size.

**Life Cycle.** For many years the life history of *Ascaris lumbricoides* (Fig. 33) was thought to be a very simple one, but this is not the case. Development of infective "rhabditiform" larvae within the eggs, after passage with the feces, requires about a month under favorable conditions. These larvae are extremely long lived; the embryoated eggs have been found to be infective in moist soil after a period of five to six years. In 2 per cent formalin solution they have been reported to be motile within the egg shell after two years. Even emulsification of fresh feces in cold 10 per cent formalin does not prevent embryonation.

Eggs containing viable larvae hatch soon after ingestion by man, and the larvae shed their sheaths. These freed rhabditiform larvae are threadlike, tapering at both ends, and are from 0.2 to 0.3 mm. in length and from 12 to 15 μ in width. They penetrate the intestinal mucosa, enter the blood or lymph streams, and are carried to the lungs. Here the majority of them lodge and undergo further development, usually molting twice in about ten days, until they are about 1.5 mm. long. The inflammatory reaction in the lungs is in proportion to the number of larvae present.

After the period of growth and development in the lungs, the larvae enter the air passages, are carried up to the trachea and are
swallowed with accompanying mucus. Development continues in the small intestine for from six to eight weeks before maturity is attained. The adult worms are able to anchor themselves to the mucosa by means of their three lips and the small buccal cavity behind them.

Fig. 33. Life cycle of *Ascaris lumbricoides*. The male (a) and female (b) worms (×1; app.) parasitize various parts of the digestive tract of man (r). The fertilized female (b) deposits her eggs (c, ×200 app.) principally in the intestines. These are carried to the external environment with the feces and after two to three weeks in summer under favorable conditions embryonate (d), soon becoming infective for man when the fully developed eggs are ingested from the ground (i, j), directly from the hands or by means of food or drink (k, l).

The mature embryos become free in the intestine of the host and after an extended circulatory migration in the larval state in the liver, heart, and lungs, the developing larvae return to the intestine. They then grow rapidly to adult male (a) and female (b). The fertilized female (b) produces eggs which reach the external environment as before to begin again the cycle. (After Kouri. Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical, 1940.)
Pathology and Symptomatology of Ascariasis. The pathology and the symptomatology of ascariasis may be logically discussed in relation to (1) the migrating larvae, and (2) the adult worms.

The lungs bear the brunt of injury caused by the migrating larvae. The diameter of the incoming larvae is often greater than that of the capillaries, and consequently considerable trauma occurs during the growth and subsequent passage of the larvae into the alveoli. A few larvae may produce small petechial hemor-rhages; in heavy infections the total tissue damage is extensive, and is followed by varying degrees of consolidation of the lobules. Extensive lung involvement may occur, particularly in children. In the event that larvae reach the general circulation and are filtered out in various arterial terminals, they may incite mild or severe symptoms depending on their numbers and locations.

Normally, the adult worms live in the lumen of the small intestine, sometimes attached to the mucosa, which they are probably able to pinch off in small masses. They feed principally on partially digested food. Ordinarily their presence causes comparatively little disturbance, although infected children are likely to be less mentally alert and in poorer physical condition than uninfected children. Indiscretions in diet, or indigestion, may result in the spontaneous passage of the worms through the external orifices or into organs directly connected with the alimentary canal.

The symptoms commonly attributed to the presence of adult ascarids are abdominal discomfort and colicky pains in the epigastrium. Symptoms suggestive of abdominal tumor and gastric or duodenal ulcer may occur. Owing to the wandering of the worms into abnormal loci, various acute symptoms may arise at times, simulating acute or subacute appendicitis, blockage of the common bile duct, liver abscess, and hemorrhagic pancreatitis. Actual perforation of the intestinal wall with resulting peritonitis may also occur. In resection of the bowel the surgeon must be particularly careful to guard against ascarids escaping into the peritoneal cavity during operation. In selecting anthelmintics for the eradication of other intestinal worms in the presence of
ascarids, one should be chosen that is not likely to induce their migration.

**Diagnosis of Ascariasis.** Eosinophilia is usually present in moderate degree, but it is not an index of the intensity of infection. Otherwise, the blood picture is not appreciably altered in uncomplicated ascariasis.

The diagnosis is definite when the adult worms are seen, but often it is made solely on the finding of the characteristic eggs in the feces. If only male worms are present, a provisional diagnosis may be made from the clinical symptoms in lieu of the passage of one or more worms. According to Bachman, reported by Craig and Faust (1940), skin sensitivity to the specific ascaris antigen may be retained for a considerable time and a positive reaction does not necessarily mean the presence of infection.

**Treatment of Ascariasis.** Drugs commonly employed in treatment of this condition are briefly as follows: (a) Santonin alone, or in conjunction with calomel or cascara, was formerly used extensively in treating upper intestinal worm infections, especially in children. This drug, in doses reasonably tolerated by the patient, is said to be inefficient, and in effective doses, rather toxic. (b) Oil of chenopodium is a specific, but unfortunately it is rather toxic. Ascariasis associated with hookworm infection may, however, be treated fairly safely with a combination of oil of chenopodium and tetrachlorethylene. The method of administration is discussed under hookworm infection (pages 159 and 160). (c) Hexylresorcinol crysoids (Caprokol) may be given in the same dosage as for pinworm infection. It should be followed after two hours by a saline purge to remove the dead worms. This method is said to be quite efficient and is convenient for both patient and physician.

**Prophylaxis of Ascariasis.** The control and prevention of ascariasis are based primarily upon the recognition of the following epidemiologic facts: (a) Ascariasis is essentially a dooryard and household infection, as demonstrated by Cort (1931) and other investigators. (b) Under favorable conditions “seed beds” from the promiscuous droppings of children may remain infec-
tive for many months. (c) Sanitary control measures such as those employed for hookworm are inadequate unless suitable disposal of feces of small children is included.

THE HOOKWORMS

Geographical Distribution. The two generically different species of hookworm in man have interesting geographical distributions.

Necator americanus (Stiles, 1902) Stiles, 1906, is often referred to as the "New World hookworm." While this worm occurs principally in the southern United States, Mexico, Central America, and South America east of the Andes, it is also found widely distributed in tropical Africa, southeastern Asia, including the Malay Archipelago, the Philippine Islands, and northern Australia. It is generally believed that N. americanus was not originally endemic in the Americas, but was probably first introduced from West Africa in the early days of the slave traffic. Such an origin seems to be likely, and fits in best with what is known of the distribution of ancylostomiasis in the Americas.

Ancylostoma duodenale (Dubini, 1843) Creplin, 1845, is popularly known as the "Old World hookworm." This worm occurs endemically in the Mediterranean region, southern Asia, the Malay Archipelago, northern Australia and adjacent islands, east and central China, and Japan. Migration of infected persons from endemic areas undoubtedly constitutes an important factor in present-day distribution of A. duodenale. This has been notably the case of infestations of miners in both European and Californian gold mines. I found that conditions in latter mines were similar to those in the Cornish tin mines where formerly ancylostomiasis was widely prevalent among the miners but not in other parts of Great Britain.

Morphology. The essential differences between adults of the two species of hookworm in man are given in Table IX.

Adult hookworms (Fig. 35) live principally in the jejunum and to some extent in the duodenum of man. They are grayish
Fig. 34. Hookworm mouth-capsules.

A. *Necator americanus*, × 375 app. *v. pl.*, ventral plate; *d. pl.*, dorsal plate; *v. pap.*, ventral papilla; *b. cap.*, buccal capsule; *l. pap.*, lateral papilla; *d. l. pap.*, dorsal lateral papilla; *d. pap.*, dorsal papilla; *d. t.*, dorsal tooth (orifice of esophageal gland at tip); *v. lan.*, ventral lancel; *d. lan.*, dorsal lancel; *oes.*, esophagus. (Courtesy of National Institute of Health.)

B. *Ancylostoma duodenale*, × 250 app. *v. t.*, ventral teeth; *d. t.*, dorsal tooth; *v. pap.*, ventral papilla; *l. pap.*, lateral papilla; *d. pap.*, dorsal papilla; *v. lan.*, ventral lancel; *or. oes. gl.*, orifice of esophageal gland. (Adapted from Loos, The Anatomy and Life History of Ancylostoma duodenale. Duh. Rec. Egyptian Gov't School Med., Vols. III and IV.)
yellow or yellowish white, plump, rather rigid roundworms with the morphologic characteristics tabulated in Table IX. The esophagus is club-shaped and merges into the straight tubular intestine. It is one-sixth or less of the whole length of the worm, and is separated from the intestine by a muscular sphincter valve. The rectum is near the caudal end.

The reproductive organs of the female consist of a pair of long, curved tubules, one located mostly anterior to the vulva and the other posterior to it. The distal portion of each tubule is the ovary, the middle portion the oviduct, and the terminal por-
tion the uterus. The two uteri unite to form a common vagina which opens into the vulva.

The male genital organs (Fig. 35) consist of a long winding tubular testis occupying the middle anterior part of the body, a vas deferens, a spindle-shaped seminal vesicle, a long muscular ejaculatory duct opening into the rectum which is continuous with the cloaca and copulatory bursa. Two long, extrusible.
bristle-like spicules, contained in a sac on the terminal portion of the intestine, can be extended into the copulatory bursa guided by a chitinous thickening called the gubernaculum.

The eggs of the hookworms (Fig. 36) in man have a colorless, transparent shell, are oval in shape and measure about 60 by 40 μ, as observed in fresh feces. They usually contain a 4- to 8-celled embryo, and a clear space between the embryo and the shell is distinctly visible. Figure 36 shows various stages in the development of the egg. Slight differences in size and shape exist but they are not considered reliably constant features. A special property of the shell is its adhesiveness, of which advantage is taken in collecting a surface film containing the eggs. The techniques by which this can be accomplished are briefly described in Appendix VII.

**Life Cycle.** Under suitable conditions the eggs (Fig. 36, a, b, or
Fig. 38, c) deposited on the ground continue to develop, and in from twenty-four to forty-eight hours they hatch, becoming the first stage, or “rhabditiform” larvae (Fig. 36, h; Fig. 37, a; Fig. 38, c). These larvae (about 0.3 mm. long) are actively motile, and feed on organic debris. They have a narrow buccal chamber, a flask-shaped, muscular esophagus (about one-fourth to one-fifth the length of the digestive tract), a midgut, and a short rectum. After several days of feeding and growth, the larvae moult, or shed their skins (first moult), continuing to feed and grow to a length of about 0.5 mm.

Between the fifth and the eighth day, the mouth closes, the esophagus elongates, and the transformation into nonfeeding, infective, “filariform” larvae is complete (second larval stage). These larvae are considerably larger than the mature “rhabditiform” larvae, and also differ from them morphologically, as shown in Figure 37 for _N. americanus_ (A. duodenale similar). Ordinarily the skins remain as protective coverings around the still active larvae until chance contact with the skin of man occurs. Then they free themselves (second moult) and endeavor to penetrate the epidermis by way of the hair follicles or through abrasions, and burrow deeply into the corium and surrounding tissues. Many of them then enter superficial veins or else die and are phagocytosed.

Young larvae are carried by the returning venous current through the right side of the heart to the lungs where they soon break through from the capillaries into the alveoli. They are then transported further by the rising stream of respiratory mucus and after being swallowed finally reach the small intestine where they again moult (third moult).

By this time a provisional buccal capsule has formed, by means of which these “fourth stage” larvae are able to attach themselves to the intestinal villi. Here they feed, grow, and become sexually differentiated, developing a permanent capsule within the provisional one. The fourth larval skin, together with the provisional capsule, is then shed, and these worms (4 to 5 mm. long) quickly develop into adults. Fifteen to twenty days are required for com-
pletion of development in the small intestine. In about five weeks from the time of exposure of the patient to soil-infesting filari-

**Fig. 37. Larvae of Necator americanus contrasted with those of Strongyloides stercoralis.**

- Rhabditiform larva ($\times 100$ app.): *N. americanus* (a); *S. stercoralis* (a').
- Anterior end of rhabditiform larva (magnified): *N. americanus* (b); *S. stercoralis* (b').
- Filariform larva before moulting ($\times 100$ app.): *N. americanus* (c); posterior end magnified (d); *S. stercoralis* (c').
- Infective filariform larva ($\times 100$ app.): *N. americanus* (e); *S. stercoralis* (e'), posterior end magnified (e').


form larvae, the fertilized adult females begin to expel their eggs into the intestinal lumen (graphically shown in Fig. 38).

**Epidemiology.** The infective filariform larvae come to maturity in moist, shaded, sandy soils in warm climates such as the southern United States, where summers are long and winters are comparatively short and mild. These actively motile, slender larvae penetrate the skin with which they come in contact, or perhaps the
Fig. 38. Life cycle of Necator americanus. Adult male (a, X 1 app.) and female (b, X 4 app.) are found in duodenum and jejunum, the eggs (c, X 275 app.) being passed in feces (d). Under favorable conditions a rhabditiform larva develops within the egg (d) and after hatching (e) it feeds, molts and develops a protective sheath, becoming the infective form (g).

Penetrating the skin, infective forms (i, X 3 app.) migrate within the body of man (i) passing through the heart to the alveoli of the lungs, ultimately reaching the small intestine in swallowed mucus to become adults (a, b) undergoing two molts meanwhile.

The intrahuman or parasitic phase comprises two larval stages and the adult stage (a, b).

The extrahuman or free phase comprises the egg (c) and three larval stages (c, f, g). (After Kouri. In Kouri and Basnuevo: Lecciones de Parasitologia y Medicina Tropical.)
mucous membrane when taken in through the mouth as contaminants of food or drink. \textit{N. americanus} is better adapted to warmer climates, but the females lay two or three times fewer eggs than do the females of \textit{A. duodenale}.

In the absence of factors predisposing to reinfection, 70 per cent of the worms are usually eliminated without treatment within one year, but a few may persist considerably longer. Under natural conditions, however, reinfections commonly occur in endemic areas. Man alone constitutes the source of infection for other human beings.

\textbf{Pathology. Cutaneous lesions.} During the time infective larvae are penetrating the skin, there is usually local itching and burning, followed by erythematous edema and perhaps papule formation terminating in vesicles. This “ground-itch” is most commonly noted on the more tender areas of the skin, such as those between the toes and on the dorsum of the foot. Certain forms of “creeping eruption” in man that occur in hookworm-infested areas, are caused by the infective larvae of canine or feline hookworms (particularly \textit{A. braziliense}), which apparently are unable to develop further in man.

\textbf{Pulmonary lesions.} As the larvae break through the pulmonary capillaries into the alveoli, minute hemorrhages occur, causing various degrees of pneumonitis; the sputum often has a rust-streaked appearance if examined during this stage of larval migration. However, pulmonary inflammation attributable to migrating larvae has not been reported so frequently in hookworm infections as in ascariasis and strongyloidiasis.

\textbf{Intestinal lesions.} The maturing and adult worms are attached mostly to the mucosa of the middle third of the small intestine, where they produce varying degrees of injury and hemorrhage as they shift from one site to another. In a general way, the degree of pathologic change and symptomatology produced depends on such factors as the number of worms, the resistance of the host, the duration of infection, and the number and character of reinfections. Some investigators have made successful attempts to correlate the hemoglobin index with the number of hookworms present.
Symptomatology. Mild and moderately severe cases. In mild cases, anemia is slight and definite symptoms are often absent. However, when blood loss is moderate, various symptoms suggestive of gradually increasing secondary anemia arise; those of direct intestinal origin often can be relieved temporarily by eating bulky foods or by the use of laxatives. Malnutrition contributes greatly to the character and the rapidity of development of symptoms in all cases of hookworm infection.

Severe cases. Persons presenting grave symptoms almost invariably harbor a large number of adult hookworms; they not only show a marked secondary anemia but suffer much from indigestion, constipation and diarrhea, or both alternately. The hair and skin are usually dry and harsh to the touch, and there is a relatively yellow pallor of the skin and sclera. Most important signs in children are edema of the face—particularly around the eyes—and "potbelly" which is extremely common in endemic hookworm areas. In heavily infected adolescents a disturbance in endocrine balance is evidenced by delayed puberty.

Diagnosis. In endemic areas a history of ground-itch may be of value in diagnosis, but in regions in which schistosomiasis occurs the possibility of schistosome dermatitis must also be kept in mind. The appearance within a week or two of symptoms pointing to pulmonary involvement is highly suggestive of infection in progress. The later appearance of signs and symptoms indicating loss of blood from intestinal lesions offers further corroboration. However, the demonstration of the characteristic hookworm eggs in the feces must be relied upon to confirm the clinical diagnosis in all such cases, as well as to establish the presence of hookworms in many other cases in which there are few suggestive signs or symptoms, or possibly no evidence at all, of their presence.

In most hookworm infections of clinical significance, microscopic examination of three fluid slide films of the feces should afford sufficient positive evidence. Methods designed to concentrate the eggs—such as rapid sedimentation followed by centrifugation of the suspended eggs and brine or zinc sulphate flotation—are of value in diagnosing light infections. The Stoll (1923) tech-
In this connection it should be remembered that stools containing hookworm eggs, which have stood for hours at summer temperature, may contain the hatched rhabditiform hookworm larvae. These might easily be mistaken for larvae of *Strongyloides stercoralis*, which regularly occur in fresh stools from cases of strongyloidiasis.

**Treatment.** The primary treatment in hookworm disease is prompt elimination of the adult worms by means of appropriate chemotherapy. Since some anthelmintics are dangerously toxic under certain conditions, great care must be exercised in the choice of drugs and in the estimation of the ability of the patient to withstand their action. Saline purgation before the drug is administered, and again as soon as the drug has had time to exert its maximum effect, is an important part of therapy with all the drugs. Following is a list of the drugs which have been, and still are, employed to a greater or less extent.

*Thymol:* Adult dose, 60 grains (4 gm.). This drug was formerly extensively used but it is employed much less today. Combined with chloroform it is very effective but rather too dangerous.

*Oil of chenopodium:* Maximum adult dose, 3 cc. (3 minims for each year of age for children). This drug is considered to be more effective than thymol, but it is quite toxic. For mixed infection of ascaris and hookworm, it is still sanctioned when given in conjunction with carbon tetrachloride or tetrachloethylen. Contraindications to the use of oil of chenopodium are hepatic cirrhosis, kidney and respiratory diseases, pyrexia, and pregnancy.

*Carbon tetrachloride:* Dose for adults, 3 cc. (3 minims for each year of age for children). It is considered very effective and is usually well tolerated. Particular contraindications are alcoholism, hepatic cirrhosis, kidney and respiratory diseases, and serum-calcium deficiency. In mixed infections of hookworm with ascaris this drug should not be administered except in combination with oil of chenopodium.

*Tetrachloethylen:* The dose is the same as that for carbon tetrachloride. This drug is considered to have no important contraindica-
tions, and is said to produce ordinarily no more than a transient burning sensation in the pit of the stomach, slight nausea, and dizziness. It is believed by many clinicians that this is the drug of preference in treating the average hookworm infection.

Hexylresorcinol (crystoids) in hard gelatin capsules: Administration and dosage are similar to those for treatment of enterobiasis (oxyuriasis). A follow-up saline purgative may be used if desired. The effectiveness of this drug for hookworm eradication is said to be only 75 per cent, but it is considered to be relatively nontoxic when given according to directions.

Important details in the administration of all these drugs are:

1. The saline purgative of choice is sodium sulphate—1 ounce (30 gm.) in a half glass of water—the preliminary purge, if employed, being given the night before, and the follow-up purge two hours after the administration of the anthelmintic.

2. In the morning, the patient should remain in bed, abstaining from food; water, tea, or black coffee are permitted.

3. A light meal may be given only after a good bowel movement has resulted from the follow-up purge.

4. In heavy infections, the feces should be examined three days after completion of treatment, in order to gauge its effects. A second course of treatment may be given one week following the termination of the first.

Oil of chenopodium, carbon tetrachloride, and tetrachlorethylene may be conveniently administered in gelatin capsules, or mixed with bread crumbs sweetened with sugar. In no instance should alcohol be permitted or oil purgation substituted for saline purgation.

Prophylaxis. Control and prevention of hookworm infection entails not only individual prophylaxis but also community control of soil infestation. On a limited scale, success in control of hookworm infection achieved during the construction of the St. Gotthard tunnel and in the Cornish tin mines demonstrated the feasibility of such measures in controlled groups. However, it remained for the Rockefeller Hookworm Commission and its successor, the International Health Board of the Rockefeller Foundation, to demonstrate the practicability of mass population con-
tro1. The policy of the International Health Board has been to attack the problems involved on broad economic and sociologic lines in co-operation with local governmental units. At first, as an accurate means of determining the incidence and intensity of hookworm infection, actual "worm counts" were made following treatment; later these counts were replaced by the Lane (1932) and the Stoll (1923) egg-counting methods (Appendix VII). Special methods—notably the use of the Baerman apparatus (Appendix VIII)—provided a means of determining the degree of soil infestation.

In Puerto Rico, Ashford and Gutierrez (1911) demonstrated that a community receives hookworm infection from the soil in proportion to the amount of infestation the population puts into it. This fact suggested the idea of mass treatment of heavily infected populations. It was later maintained that the administration of an efficient anthelmintic to all members of a heavily parasitized group should remove 95 per cent of all the worms and result in 85 per cent individual cures. Subsequently it was ascertained that a second treatment after about four weeks caused such a drop in soil infestation that human infection later became negligible, except for the introduction of infected individuals from without.

Finally, individual and group education regarding the advantages of properly constructed "pit privies" in rural areas and of sanitary community sewage disposal in towns and cities, has contributed much to the prophylaxis of hookworm infection in our southern states. At the present time, the typical "hookworm family" is rapidly disappearing. Among primitive peoples, the bored-hole latrine with concrete top is considered less hazardous than the pit privy. Where human feces and urine (night soil) are used as fertilizer for growing crops, the problem of the control and prevention of hookworm and other intestinal parasitic infections is practically untouched.

Types of privies. The problem of safely disposing of human excrement, when suitable means of flushing with water are not available, is often a difficult one. However, certain types of
privies have been evolved, the intelligent installation, care and use of which will justify the effort. The distinctive types which have become more or less standard are shown in Figure 39.

![Figure 39. Types of privies, scale 1/8 in. to foot app. (After Rosenau, Preventive Medicine and Hygiene, Edition 6. Courtesy of Appleton-Century Company.)](image)

The outdoor surface privy is the simplest and least expensive. Excreta are deposited on the ground surface, and the liquids evaporate or leach away in porous soils. Devoid of simple refinements, such as the daily use of absorbents preferably with deodorant properties (e.g., quick lime) and the frequent manual removal of its contents, this privy is likely to become a nuisance. Therefore, one of the other five types shown in the sketch is to be preferred, the choice depending on the particular circumstances and conditions in each instance.

Use of a pit tends to minimize fly breeding, provided it is deep enough, and is kept dark. Covering the excreta with an absorbent material such as soil or sawdust constitutes a helpful refinement. Deep pits are a grave menace to nearby wells, because the organic
matter is likely not to be readily broken down by bacterial activity. They are particularly objectionable in places where soil water rises into them. The vault, septic, and chemical privies are obviously much safer; they are expensive in order of their enumeration. If low cost with safety and reasonable freedom from offensive odors is the first consideration, substitution of the surface pail privy for the ordinary surface privy constitutes a logical choice. Obviously it is essential to apply regularly earth, ashes, sawdust, or similar material, as an absorbent and at least a partial deodorant, and to empty the pails regularly, disposing of the contents in a sanitary manner.

With running water readily available, the problem of sewage disposal may be solved more satisfactorily, although not always more safely. In order of cost and simplicity, cesspools and septic tanks come first, the latter being the safer but often more troublesome unless properly constructed and operated. There are also available more refined methods of septic-tank treatment followed by appropriate means of effluent oxidation. Finally modern community water-carriage sewage-disposal systems relieve the individual of much of the direct annoyance and burden involved in disposal of excreta, which is usual under the primitive conditions of life still existing in many rural and sparsely populated areas.

HOOKWORM LARVAE AS ABERRANT PARASITES OF MAN

Brief mention should be made of two species of the genus *Ancylostoma* which occur naturally in lower vertebrates, but whose larvae have been reported to infect man at times.

*Ancylostoma braziliense* (de Faria, 1910) has been reported from various parts of the tropical world as an aberrant parasite of man. Larval forms of this worm are believed to be the etiologic agent in a so-called “creeping eruption” of man. The adults of this species, normally found in dogs and cats, are larger than *N. americanus* and smaller than *A. duodenale* of man. They have a characteristic buccal capsule, one pair of small median teeth, and one pair of large outer teeth. The bursa of the male is also quite distinctive.
Ancylostoma caninum (Ercolani, 1859) Hall, 1913, is a common parasite of dogs and cats, particularly in the Northern Hemisphere. One case in man has been reported from the Philippine Islands. It is a large hookworm with three pairs of ventral teeth. The male has a large floating bursa supported by long slender rays. A great deal of fundamental research into the bionomics and host-parasite relationships of hookworms has been possible through study of this species.

Strongyloides stercoralis (Bavay, 1876)

STILES AND HASSALL, 1902

This parasitic roundworm is found in warm, moist climates throughout the world, and the specific infection, like that of hookworm, is confined almost entirely to man. Infections in animals are caused by other species. Although dogs may be inoculated experimentally with the strongyloid worm of man, the infection soon dies out.

A careful study of Figure 40 should make clear the generally accepted facts regarding the intricate life cycle of this worm, which as yet is not perfectly understood. Some authors believe that the free-living sexual generation in the soil is the natural one, and that parasitization represents a subsequent adaptation of this worm to a new, and in some respects a more secure, environment.

There are several points of contrast between Strongyloides stercoralis and the hookworms which should be remembered: (a) The eggs of S. stercoralis are already embryonated when seen in stools after a purge. (b) Free rhabditiform larvae are the forms usually seen in stools. These have a strikingly prominent esophageal bulb, more like that of A. duodenale than N. americanus. (c) The parasitic males are rhabditiform in type, being almost identical with the free-living males, but they are said to have a slightly larger buccal cavity. (d) The parasitic females are more delicate filiform worms than the free-living females are. They measure, according to Faust (1939), up to 2.2 mm. in length and from 30 to 75 \( \mu \) in diameter.

* Ancylostoma malayanum, a parasite of bears in India, also has been reported in man.
Fig. 40. Schematic life history of Strongyloides stercoralis. Parasitic female. × 50 app.; rhabditiform larvae. × 100 app.; infective stage (filariform larvae) × 40 app. (After Kouri. In Kouri and Basuñev: Lecciones de Parasitología y Medicina Tropical.)
Epidemiology. Although *S. stercoralis* is generally considered to be an exclusively human parasitic worm, dogs have been found to harbor a strikingly similar strongyloid worm. The infective stage larvae are developed in fecal-polluted soil and their return to man is believed to be dependent mainly on direct skin penetration, as in the hookworms. In view of the greater complexity of the life cycle of *S. stercoralis*, there may be other methods of infection. Faust's views regarding a secondary sexual phase in the parasitic generation with hyperinfective larvae arising in the intestine are very interesting. Convincing proof of their correctness will depend largely on more carefully correlated experimental and epidemiologic data.

Pathology and Symptomatology of Strongyloidiasis. In so far as penetration of the skin by the infective larvae and their subsequent development in the body are concerned, one would expect the essential pathology to be similar to that of hookworm infection. This seems to be largely true at first, but owing to the disparity in size and specific habits of the adult worms, these earlier parallels tend to become considerably divergent as the life cycles of the parasites unfold themselves. Because of the intensity of local reaction to trauma resulting from their passage through the alveolar walls, it is suspected that some of the strongyloid larvae are temporarily detained in the alveoli and smaller bronchioles of the lungs. During this arrest in their forward migration, some authors believe that strongyloid larvae may develop to adolescence, and that females may invade the bronchial epithelium as they do ordinarily the intestinal mucosa. However this may be, the burrowing habits of the pregnant females in the intestinal mucosa produce a catarrhal inflammation, resulting in cell necrosis and associated mucous diarrhea.

Several recent investigators, including Faust and De Groat (1940), state that they have found evidence of more or less general migration of filariform larvae of *S. stercoralis*. The beliefs of these workers on the various points raised may be derived from Faust's (1936) summation of his views regarding phases of development in the life cycle of *S. stercoralis*, which is as follows:
FIG. 41. Eggs of the most important human helminths. (All eggs are of the same magnification, X 320 app.)

(a) *Indirect development*, based essentially on the free-living phase of growth and transformation into the parasitic phase when unfavorable environmental conditions supervene; present in warm moist climates. (b) *Direct development*, rarely separated completely from the indirect mode, but primarily responsible for human infection, since it requires the parasitic phase for its continuation; the usual or predominant type in temperate climates. (c) *Hyperinfection*, which provides filariform (or rarely rhabditoid) larvae for internal reinfection; usually seen in warm climates.

**Diagnosis and Prognosis of Strongyloidiasis.** A definite eosinophilia, often quite high, usually occurs in *S. stercoralis* infections. Therefore, the presence of this symptom should at once suggest a search by microscopic examination of the freshly passed feces for parasitic worm eggs, and rhabditiform larvae which are specifically diagnostic of *S. stercoralis*. In case of light infections, the material to be examined should first be concentrated. At the time of passage through the lungs, the rhabditiform larvae may be found in the sputum.

Formerly an unfavorable prognosis in strongyloidiasis was rarely if ever considered. However, according to reports in recent literature, the prognosis may be unfavorable or even grave in heavily infected chronic cases.

**Treatment of Strongyloidiasis.** Among the many therapeutic agents which have been tried, gentian violet (medicinal) is reported to be the most satisfactory. This drug should be administered three times daily before meals, in 1 grain (0.06 gm.) entericoated tablets until 50 grains (3.3 gm.) have been given. In obstinate cases, 25 cc. of a 1 per cent aqueous solution of the dye may be intubated into the duodenum. This method is said to give excellent results. Even intravenous injection of a 0.5 per cent aqueous solution, in volume not exceeding 25 cc., has been suggested in cases of severe chronic intestinal and pulmonary infection.

**Prophylaxis of Strongyloidiasis.** Measures for control and prevention are similar to those outlined for hookworm infections.
Based on the newer conceptions regarding the life history of *S. stercoralis*, and of strongyloidiasis, effective therapeutic measures are most important, not only to lessen soil infestation but also to guard against a so-called hyperinfection or reinfection of afflicted persons.
CHAPTER IX
FOOD-INFESTING WORMS: TRICHINELLA SPIRALIS AND FLATWORMS

*Trichinella spiralis* (Owen, 1835) Railliet, 1895
("Trichina" Worm)

The larvae of the viviparous parasitic roundworm, *Trichinella spiralis*, encyst in striped muscle of man, hog, rodents, and other carnivorous animals, thus giving rise to the clinical condition known as trichinosis (trichiniiasis or trichinelliasis). This disease is widespread geographically, the incidence in man varying from about 3 to 25 per cent in the United States.

**Epidemiology.** Under natural conditions, it is believed that trichinella infection is kept alive by rats which are cannibalistic, both larvae and adults developing in the same species. Man acquires the disease almost exclusively by eating infected pork. Hogs may be infected through eating uncooked scraps of trichinous pork or infected rats. In some localities as many as 15 per cent of the hogs have been found to be parasitized.

Raw or undercooked sausages or hamburgers containing pork are very common means of transmission of the larval forms of *T. spiralis*. Although ordinary meat inspection is a most inadequate safeguard against this infection, thorough cooking throughout of pork and pork products at a temperature of at least 137°F. (58° C.) affords security. Storage for twenty days or more (depending on size of pieces and method of storage) at a temperature no higher than 5°F. (—15°C.) will kill the larvae. Rapid freezing to lower temperature and shorter storage of meat also kills the larvae.

**Mechanism of Infection.** When man or another susceptible animal ingests meat containing viable, encysted *T. spiralis* larvae,
the latter are freed in the stomach and intestine by action of digestive juices on the muscle and cyst capsule. They soon lodge, principally in the glandular crypts of the duodenum and the jejunum, feed, and develop ordinarily in from two to three days into adult males and females (Fig. 42). The females measure from 3 to 4 mm. in length and from 60 to 75 μ transversely. The males * are only about one half this length and have a cloaca surmounted by two terminal conical papillae.

After copulation the viviparous females burrow through and beneath the mucosa and begin to deposit larvae, usually in the small swollen lymph spaces in which the adult females are often found coiled (Fig. 43, B).

The larvae that reach the lymphatics and venules (measuring about 100 μ in length and 6 μ in diameter) are carried to the liver and the lungs, thence to the general arterial circulation and capillary beds, particularly in striated muscle where blood flow is good. Encystment occurs within the muscle fibers. They are present in greatest numbers in the muscles of mastication, phonation, and respiration. Larvae may be found in muscle in increasing numbers from the ninth to the fortieth day after infection.

Pathology and Symptomatology of Trichinosis. Encystment (Fig. 43) often occurs near the tendinous insertions of muscle fibers when the larvae have increased approximately threefold in size. A single female may produce from 1000 to 1500 larvae during her lifetime of about six weeks.

The encystment capsule is the result of round cell and eosinophile infiltration around the larvae which become tightly coiled within, the long axis being parallel with the muscle fibers. Accompanying and subsequent pathologic changes are degeneration, cloudy swelling of muscle fibers, thickening and other changes in the sarcolemma, proliferation of interstitial connective tissue, and calcification within from six to eight months.

* Most textbooks state that in the intestine the males are lost soon after fertilization of the females. However, it has recently been found experimentally in mice by Rappaport (1942), that the females outnumber the males in a ratio of about 3:1 during the first two weeks of the infection but that soon afterward this sex ratio is reversed, the females being lost more rapidly than the males.
Fig. 42. Life cycle of *Trichinella spiralis* (assembled). Various mammals may serve as hosts of *Trichinella spiralis*; rats and pigs are probably the most important. In man, except where cannibalism is practiced, the cycle terminates with encysted larvae in striated muscle. Adult male and female, × 20 app.; encysted larvae, × 50 app.
Fig. 43. Successive phases in evolution of pathology of T. spiralis in experimental mouse. (Sections and photomicrographs by Irving Rappaport.)


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Both the pathology and the symptomatology vary with the stages of the disease, which may be referred to as invasion, migration of larvae, encystment, and tissue repair. During the first few days following the ingestion of trichinous meat, the rapidly maturing female worms are impregnated by the males and become embedded in the mucosa. Associated symptoms, such as nausea, vomiting and diarrhea are due to the irritation and inflammation of the duodenal and jejunal mucosa. Embryos discharged by the female worms are disseminated by means of circulating blood and find lodgment in various tissues and organs. The inflammatory reactions caused by the presence of the larvae bear a causal relation to lesions of the skin, muscular soreness and pain. Edema particularly of the face and hands often occurs early and continues during this phase of the disease.

With encystment symptoms of widely divergent character may be expected, the severity of which depends largely on the extent and intensity of the migration. In the more severe cases the patient may die from such conditions as toxemia, myocardial involvement, pneumonia or nephritis. Further details may be found in regular clinical descriptions of this disease.

**Diagnosis of Trichinosis.** The important diagnostic features of trichinosis are (a) eosinophilia (often 50 per cent or more); (b) the presence of adult worms in the feces during the diarrheal stage, but difficult to find; (c) larvae in the blood during the period of migration; (d) presence of encysted larvae in biopsy of striated muscle; (e) Bachman’s intradermal reaction and precipitin tests.

Bachman’s intradermal test as modified by Sawitz, and other serological tests have been described in detail by Faust (1939). They are considered very useful in the detection of light infections, for purpose of confirmation, or in doubtful cases.

Differentially, trichinosis may be confused particularly with acute food poisoning, cholera, typhoid fever, diarrhea or dysentery from other causes.

**Treatment and Prophylaxis of Trichinosis.** Treatment is largely symptomatic since no effective parasiticide has thus far been dis-
FOOD-INFESTING WORMS

covered. Prevention, therefore, must be relied upon in combating trichinosis, both from the individual and the community standpoint. Important points in prevention are (a) thorough cooking of pork and pork products; (b) refrigeration of pork at 5° F. (−15° C.) for not less than twenty days; (c) destruction of carcasses of infected and suspected animals; (d) rat control; (e) thorough cooking of garbage particularly when fed to hogs.

Pork must be cooked for a length of time proportionate to its weight in order to insure thorough penetration of the heat to the center. Experimentally it has been shown that at least one half hour's boiling is required for each two pounds of meat. The United States Department of Agriculture goes further than this in recommending, as a general rule, boiling one half hour for each pound of meat. Rapid roasting is likely not to destroy all the parasites, owing to the relatively lesser effect of the heat toward the center. Among the various methods of cooking either by boiling, braising or roasting, boiling is the most effective in heat penetrability. Extreme care must always be exercised to insure thorough penetration of the heat to all portions of the meat.

THE DIGENETIC TREMATODES OR FLUKES

An intelligent understanding of the diseases caused by members of this group demands a knowledge of the interesting cycles of development which they have evolved in order to insure their perpetuation. Except for the schistosomes, which will be discussed separately in Chapter X, these flukes are monoecious, or hermaphrodite, as are also the individual proglottids or segments of the tapeworms which will be discussed later in this chapter.

Among the hermaphrodite flukes, four are of sufficient general interest and medical importance to be briefly considered here. The life cycle of Fasciola hepatica—a common fluke disease in sheep and cattle—is typical of these parasitic flatworms. A considerable number of cases of fascioliasis of man have been reported recently from Cuba by Kouri et al. (1938) and the Argentine by Bacigalupo (1938).
Fasciola hepatica (Linnaeus, 1758)
(Sheep Liver Fluke)

General Morphology. The adult stage of Fasciola hepatica is leaflike, somewhat spinose, measuring up to about 30 mm. in length and 13 mm. in width. It has two anteriorly placed suckers known as the oral, and the ventral or acetabulum. Respiration is believed to be anaerobic. The excretory system is characterized by “flame cells,” the vibrations of the hairlike processes of which are necessary in maintaining the excretory function. The nervous system is simple, while the reproductive organs in both male and female are rather complicated. Detailed descriptions of these organs may be found in standard works on parasitology.

Life Cycle. The eggs of F. hepatica are immature while in the biliary passages and when evacuated in the feces. After maturing in water (9 to 15 days), they hatch into free-swimming ciliated “miracidia.” The large, ovoid, operculate, yellowish brown eggs measure about 140 μ in length and from 65 to 85 μ in width. Within a few hours of emerging, the ciliated “eye-spotted” miracidia invade the bodies of certain snails if opportunity offers. Here, during a period of thirty days, they undergo metamorphosis successively into sporocysts, rediae, and ultimately cercariae (Fig. 41, a, b, c, d, e, f). An adult F. hepatica (× 1 1/2 app.) is shown at j in this figure and an encysted metacercaria at g (× 55 app.).

If the snail is in water, the mature cercariae literally swarm out of their host at night, and after swimming about lose their tails within a few hours and encyst as small, round, whitish bodies (metacercariae) preferably on aquatic vegetation or in the water. Mammals grazing or drinking from pool bottoms in damp, infested pastures contract the infection. The ingested metacercariae excyst in the small intestine of the vertebrate host and are said to migrate through the intestinal wall into the body cavity (or possibly up the common bile duct), thence into the parenchyma of the liver or the portal vein. Eventually they make their way into the bile passages, where they grow to maturity and frequently become encapsulated.
The adult flukes (a. × 11/2 app.) live in the bile passages of the definitive host (cattle, i, man i').

The egg (a. × 115 app.) is passed to the intestine with the bile and thence to the external environment in feces (b). The egg (b. × 115 app.) in which embryonation has occurred and from which a miracidium (infective for specific snail intermediate hosts) is about to escape through the operculum (o), the cap of which is slightly elevated at one side.

Ciliated miracidia (c) which swims about freely in water seeking a suitable snail host (e) in which they may develop successively into sporocysts (d), rediae (e), and cercariae (f. × 30 app.). The latter swim about freely upon escape from the body of the snail host and soon encyst as metacercariae (g. × 55) in the water, free or attached to stems or leaves of water plants.

Metacercariae (g) are ingested by the ordinary definitive host (i) while grazing, or are ingested by an accidental definitive host such as man (i') through his food (i) or drink (i'). In the intestine, the metacercariae excyst, make their way through the intestinal wall to the capsule of the liver where a contact necrosis occurs giving them access to the parenchyma and the biliary vessels. In the latter the invading metacercariae become hermaphrodite adults, eggs of which reach the external environment with the feces to begin again the cycle.

(After Kouki. In Kouki and Basnuevo: Lecciones de Parasitología y Medicina Tropical.)
FIG. 45. Life cycle of Clonorchis sinensis. The adult fluke (center, ×1 app.) lays its operculated eggs (b, × 400 app.) in the bile vessels of the definitive host (man, dog, cat) and aided by the flow of bile these eggs reach the intestine, later appearing in the feces (i). A ciliated miracidium develops within the egg (b) and in water it escapes (c) through the opercular end of the egg (b) to seek a suitable snail-host (z)—first intermediate host—and passes through the sporocyst (d) and redial (e) stages, successively, to form cercariae (f) similar to Fasciola hepatica.

Encystment of cercariae (f, × 50 app.) to form metacercariae requires a second intermediate host (certain fish) in which the metacercariae (g) develop. Infested raw, improperly cooked, or salted fish (i), ingested by man (i), dog or cat, upon digestion supply free metacercariae which reach the liver in the same manner as do metacercariae of F. hepatica, establishing their habitat in the bile ducts, and produce eggs and miracidia similarly to begin again the cycle in the snail, continuing through the fish and back to man again.

(After Faust and Khaw, Germain and Neveu-Lamaire, Neveu-Lamaire and Pellegrin. In Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical.)
liferation of biliary epithelium and later desquamation resulting from local trauma and toxic irritation. Cysts form in the bile ducts, followed by new formation of bile capillaries, connective tissue hyperplasia and fibrosis, particularly around arrested eggs. All these tissue changes are progressive and the impairment of liver function depends upon the total number of parasitizing worms resulting from single or multiple infections over a period of years. Clinically, Inouye (1903) recognized three stages of the disease; namely, mild and essentially symptomless; progressive, with irregular appetite, fullness in the abdomen, diarrhea, edema, and enlargement of the liver; severe, with a syndrome associated with portal cirrhosis.

**Diagnosis, Treatment, and Prophylaxis of Clonorchiasis.** Eosinophilia is a frequent finding, and experimental studies indicate hyperplasia of bone marrow with respect both to eosinophiles and histiocites. Positive diagnosis is based on recognition of the characteristic eggs in the feces or in duodenal drainage fluid.

Therapeutically, intravenous administration of sodium-antimony tartrate appears to be the best method of treatment. Judging from Kouri's (1935) recent successes at Havana in treatment of cases of *F. hepatica* infection with emetine hydrochloride, this chemical may be recommended tentatively in the treatment of clonorchiasis.

Prevention depends upon thorough cooking of all fresh-water fish likely to be infected. In endemic areas the addition of ammonium sulphate to fresh night soil is recommended as a control measure.

**Paragonimus westermani** (Kerbert, 1878) Braun, 1899
*(Oriental Lung Fluke)*

As in clonorchiasis, the heavily infective endemic foci of disease caused by *Paragonimus westermani* are in the Far East. Establishment of new endemic foci depends on the local availability of suitable molluscan species to serve as the first intermediate host. Specificity of the second intermediate host (the crab or the crayfish) is less restricted. Generally the adult is found heavily en-
gawa (1919), migrate successively through its wall, peritoneal cavity, diaphragm, and pleural cavity into the lungs, coming to rest in the vicinity of the bronchioles.

The growing cercariae soon develop into mature worms within fibrous tissue capsules, which represent a reaction on the part of the host against the presence of the parasites. Several weeks are required to complete this phase of the life cycle. The eggs are usually coughed up with the sputum, or if swallowed, pass out with the feces. Thus two sources of infection are provided for the first intermediate host, a snail.

**Pathology and Symptomatology of Paragonimiasis.** The metacercarial larvae of *P. westermani* usually produce no significant pathology or symptomatology until they develop into mature worms in the lungs, and other less usual loci. In the lungs the adult flukes initiate tissue reactions leading to the growth of a thick fibrous tissue envelopment. Such cysts are commonly found in the deeper portions of the lungs and are frequently the size of a hazel nut. Remnants of lung structure, such as small blood vessels and bronchioles, are frequently seen in section, the worm itself being surrounded by a thick, blood-tinged purulent fluid containing rusty-looking masses of golden brown eggs.

The pathologic lung picture is essentially that of local, more or less generalized cirrhosis with irregularly situated cystic dilations of the bronchial tree and tubercle-like abscesses. This condition is accompanied by cough and hemoptysis, frequently associated with pulmonary pain likely to be intensified on physical exertion. The development of adult worms in other localities gives rise to an analogous symptomatology.

**Diagnosis of Paragonimiasis.** Positive diagnosis is made by finding the characteristic eggs in sputum, feces, or purulent material from other foci of development of adult worms. The sputum is frequently blood tinged and is spotted with rusty brown flecks consisting of masses of eggs.

Clinically, the differential diagnosis must be made from other lung conditions, and when localization occurs elsewhere the other more common diseases of similar symptomatology affecting such
parts of the body must be ruled out. Complement fixation tests are reported to be positive in the majority of cases.

Treatment and Prophylaxis of Paragonimiasis. Thus far, the treatment of paragonimiasis has not been very satisfactory. Some clinicians, however, believe that good results are obtained by the administration of emetine hydrochloride. Individual infections may be controlled to a certain degree by removing the patient from the endemic area. Avoidance of infection depends on abstinence from raw, freshly salted, or inadequately cooked crabs or crayfish.

_Fasciolopsis buski_ (the Large Intestinal Fluke) and Other Intestinal Flukes

Among flukes which may parasitize the intestine of man, three deserve special mention. They are often classified as the large and the small intestinal flukes. It might be stated in passing that other than _F. buski_, all the intestinal trematodes reported for man are comparatively small, frequently measuring considerably less than one centimeter in length.

_Fasciolopsis buski_ (Lankester, 1857) Odner, 1902

Morphology and Life Cycle. _Fasciolopsis buski_ is primarily a parasite of the pig, but it occurs frequently in man in parts of China, and various other endemic foci in southeastern Asia. It lives attached principally to the wall of the duodenum or the jejunum. Although the adult resembles _F. hepatica_ superficially, the internal structure is significantly different. In _F. buski_ there is a fairly clear delimitation of the various organ systems, the absence of a distinct cephalic zone, closer proximity of acetabulum to oral sucker, unbranched intestines, and highly branched testes.

The life cycle of _F. buski_ resembles somewhat that of _F. hepatica_. The cercariae, resembling those of _F. hepatica_, encyst as metacercariae on water plants cultivated in endemic areas. Infection in man is associated with the customary removal between the teeth of the hull or skin of the raw pods, bulbs, roots or stems of these plants. Although many of the infective metacercariae are
removed in this manner, not a few are ingested with the raw edible parts of the plants and excyst in the small intestine. They attach themselves to adjacent parts of the intestinal wall and become adults in the course of about three months.

**Symptomatology and Pathology.** Points of attachment of adult worms to the mucosa become centers of hyperemia, inflammation, and ulceration. Erosion of capillaries gives rise to hemorrhage. Toward the end of the prepatent period, hunger pains and toxic diarrhea appear, and if infection is mild, these continue to be the outstanding features of the disease. In heavier infections the resulting ulcerative condition of the mucosa may simulate other ulcerative lesions. Large numbers of worms may produce acute ileus. Later symptomatic developments include asthenia, edema (particularly of the face, abdominal wall, and legs), and ascites with generalized abdominal pain.

**Diagnosis and Treatment.** Diagnosis of *F. bushi* infections should be based on a history of residence in endemic areas; and identification of the characteristic eggs in the feces (Fig. 41, R). A definite eosinophilia frequently occurs and lymphocytosis may be manifested. Drugs, such as betanaphthol, carbon tetrachloride, or tetrachlorethylene, have been recommended as therapeutic agents, but caution is urged regarding both administration and contraindications.

**Prophylaxis.** Immediate protection against infections by *F. bushi* consists in immersion of infested vegetables in boiling water for from ten to fifteen seconds. Important control measures are: (1) disinfection or sterilization of night soil in endemic areas; and (2) killing of the snail host by the use of 1 to 50,000 copper sulphate solution in the water of flooded fields where the vegetables are grown and where infected feces are employed as a fertilizer.

**TWO HETEROPHID INTESTINAL TREMATODES**

The adults of these small intestinal flukes, known as *Heterophyes heterophyes* (V. Siebold, 1852) Stiles and Hassall, 1900, and *Metagonimus yokogawa* (Katsurada, 1912), do not exceed
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2.5 mm in length, the latter usually being the larger. Morphologically they resemble each other somewhat closely, and their life cycles are similar, the encysting or second intermediate host being either a fresh- or salt-water fish for *H. heterophyes* and a fresh-water fish for *M. yokogawa*, so far as is known. *H. heterophyes* has been reported from the Nile Delta and the Far East, and *M. yokogawa* is a common heterophid of the Far East, northern Siberia, and the Balkan States.

In most instances these flukes appear to be comparatively harmless to the host. However, such symptoms as abdominal discomfort, nausea, and diarrhea disappear from infected persons upon expulsion of the worms by appropriate treatment. The eggs (Fig. 41, Q) are of about the same size as those of *C. sinensis* (Fig. 41, P) and frequently have a somewhat superficial resemblance to them.

THE CESTODES OR TAPEWORMS

1. Man as Primary Host

*Diphyllolothrium latum* (Linnaeus, 1758) Lühe, 1910

(Broad or Fish Tapeworm)

*Diphyllolothrium latum*, a tapeworm of man and other vertebrates, is similar to the trematodes or flukes in the following respects: (a) The eggs are operculate. (b) The embryo is ciliated and upon hatching is able to swim about in the water. "Coracidium," the name applied to it, is the homologue of miracidium as employed in describing the equivalent life cycle phase in the trematodes. (c) The genital atrium in individual segments or "proglottides" is similarly situated along the midline of the ventral surface, and not laterally, as in most other tapeworms. (d) Egg production and egg laying continue simultaneously over a period of time. (e) Two intermediate hosts are required, as in *C. sinensis* and *P. westermani*.

Distribution, Biology, and Morphology. *D. latum* is a common parasite of man in the Baltic states, Russia, and the Great Lakes region of America. The infection in the intestine of man is ac-
quired through eating fresh-water fish infested with the encysted final larval stage (plerocercoid).

The body of the adult worm (the strobila) may have 3000 or more individual segments (proglottids) and may attain a length of from 8 to 10 meters. The head (scolex) usually measures approximately 1 mm. across and 2.5 mm. lengthwise. Immediately behind the scolex is a slender neck region which is several times the length of the head. Beyond this the growth of individual proglottids begins, which gradually mature as they become more distal. Fertilization, egg production, and egg laying continue more or less simultaneously over a considerable period of time. There is less tendency for the terminal proglottids to separate from the strobila than in the Taenias, and as they cease to function disintegration takes place. The gross appearance of a series of unstained mature and gravid segments, together with diagrams of the female and the male sex organs, are shown in Figure 47.
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Since eggs are discharged regularly through the uterine pore of each of many functional gravid proglottids, it has been estimated that a single large worm may give off as many as 1,000,000 eggs a day. These eggs (Fig. 41, E) are operculated, usually yellow to golden brown in color, and measure about 70 by 45 μ. Those of *F. hepatica* and *F. buski*, which have a similar appearance, are considerably larger.

**Life Cycle.** In water the eggs (Fig. 48, a) develop rapidly, and under favorable condition the ciliated "hexacanth" embryos (b) mature in from two to three weeks. These, the "coracidia," escape through the opercular end of the shell and swim about by means of cilia. If ingested within twelve hours by the proper cyclops (2), they transform in from two to three weeks into "procercoid" larvae (c), which measure approximately 500 μ in length. They still retain their six hooklets located at the caudal end.

Further development depends upon ingestion of the procercoid-containing cyclops by a fresh-water fish (Fig. 48, 3). These larvae, liberated from the crustacean by the digestive juices of the fish host, work their way through the tissues and come to lie between the muscle fibers where they grow to lengths as great as 6 mm. They are then known as "plerocercoid" larvae (d); these are the infective forms.

Man, dog, and wolf, eating raw, insufficiently cooked, or poorly smoked fish infested with this second larval form, become the definitive host in which adult worms may be found. Such infective larvae already show the anlage of the slitlike suckers called "bothria," so characteristic of the fish tapeworm. Upon release from the encompassing muscle fiber, they attach themselves to the mucosa of the duodenum or the upper jejunum, feed on digestive juices by absorption, and attain maturity in from three to eight weeks.

**Epidemiology.** Although mammals other than man may also become infected, probably man himself is primarily responsible for spread and maintenance of the infection. He seeds the water with eggs; here they mature and hatch into ciliated coracidia,
FIG. 48. Life cycle of *Diphyllobothrium latum*. The adult worm (*e*, *e*, $\times \frac{1}{2}$ app.) lives in the small intestine of man (*1*), who is one of its definitive hosts. There it produces eggs which are carried to the external environment in the feces (*h*). In water these operculated eggs (*a*, $\times 185$ app.) soon develop a hexacanth embryo within, usually referred to as the “coracidium,” which pushes off the opercular covering of the encasing eggshell and swims about freely (*b*) by means of its numerous cilia.

Upon ingestion by a fresh-water cyclops (*2*) the coracid embryo undergoes transformation and encysts as the “procercoid” larva (*c*, $\times 62.5$ app.) in the general body cavity of the crustacean. If the crustacean is ingested by a suitable fish intermediate host (*3*), the procercoid larvae develop into “plerocercoid” larvae (*d*, $\times 3$ app.) which encyst in the viscera and muscles of this second intermediate host.

When man (*1*), dog, cat, or fox (possible definitive hosts of *D. latum*) ingests raw, poorly cooked, or salted infested fish, the plerocercoid larvae (*d*) are released by digestion and develop into adult *D. latum* which attach themselves by means of their lateral suckers to the intestinal wall. They produce eggs in great abundance which reach the external environment with the feces (*h*) to renew again the cycle.

(After Kouri. In Kouri and Basnuevo: Lecciones de Parasitologia y Medicina Tropical.)
which develop partially in the first intermediate host (cyclops). This first larval stage upon ingestion by the second intermediate host (fish) continues to grow and encyst, becoming the dormant infective larva. Finally, if man (or other suitable mammal) ingests such infested fish, raw or improperly prepared, he becomes the definitive or final host of the adult worm.

*D. latum* and pernicious anemia. Formerly it was thought that *D. latum* was responsible for a pernicious type of anemia frequently observed in Finland, associated with the presence of these worms in the small intestine. It is now generally believed, however, that a few of these parasites may be harbored for years without serious symptoms developing. In the United States, according to Birkeland (1932), the actual number of alleged cases of diphyllobothrid anemia is very small compared with the total number of infected individuals. Massive infection probably accounts for the associated anemia reported.

**Diagnosis, Prognosis, and Treatment of Diphyllobothrid Infection.** Diagnosis is based primarily on recognition of the characteristic eggs or proglottids in the feces. In the presence of pernicious anemia the prognosis depends largely upon the amenability of the anemia to treatment, together with the successful removal of the worms.

The most satisfactory drug for treating the infection is the oleoresin of aspidium (*Dryopteris filix-mas*). This drug should be administered as follows: On the night preceding the administration of the drug, the patient is given a sodium-sulphate purge (1 tablespoonful dissolved in a glass of water). On the morning of treatment, breakfast is omitted (plain tea or black coffee excepted), and the patient remains in bed. At 7:00, 7:30, and 8:00 A.M., three equal doses of 10 to 20 minims (0.6 to 1.2 gm.) of oleoresin of aspidium are administered in gelatin capsules. The dose for children is 1 minim for each year of age. At 10:00 A.M. another sodium-sulphate purge should be given, no food being allowed before a copious bowel movement occurs.

For forty-eight hours after treatment, all stools should be searched for the presence of scolecis, as these are often discharged
separately after the main body of the worm has been expelled. Repetition of the treatment, if necessary, should not be undertaken before the lapse of at least one month. Contraindications to the use of this anthelmintic are nephritis and pregnancy.

**Prophylaxis of Infection.** Immediate protection consists in thorough cooking of all suspected fresh-water fish. In endemic areas sewage should be thoroughly disinfected before it is discharged into lakes or rivers. Fishing should be restricted, and the shipping of infected fish banned. *Gefüllte* fish and raw minced fish should not be eaten unless thoroughly heated or frozen.

**Larval Forms in Man and Other Vertebrates.** Brief mention should be made of the fact that plerocercoid larvae—most of which have not been identified and are referred to in the literature as “sparganum”—have been reported in man. Morphologically similar forms are also known to be common in snakes, birds, and some mammals in certain districts. Infected material from animals harboring these forms, when injected into susceptible hosts, develop into diphyllobothria resembling one another. In the case of *Sparganum mansoni* the adult *D. mansoni* is found in cats, dogs, and related carnivores in China, Japan, Australia, and East Africa.

**Taenia, Saginata and solium (Beef and Pork Tapeworms)**

**Distribution.** Both forms of *Taenia* are world-wide in distribution. The pork tapeworm, however, has been reported to be unusually prevalent in certain parts of Europe and Asia where insufficiently cooked or raw pork is commonly consumed, but is relatively rare in the United States. It is sometimes referred to as the “armed tapeworm” because the scolex is provided with a crown of hooklets (arranged in circles on two slightly different planes).

**Development of Infection.** In man, infection from both parasites is acquired through eating raw, insufficiently cooked or cured meat, infested with the larval stage (Fig. 49, c; Figs. 50, 53, c), or “bladderworm,” the so-called *Cysticercus bovis* of beef, and the *Cysticercus cellulosae* of pork, respectively. The adult worm, or “strobila,” develops directly from a single cysticercus.
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After being released from the surrounding muscle fiber and its protective wall, the cysticercus evaginates (Fig. 50), exposing a miniature scolex or head by which it attaches itself to the intestinal mucosa of man through use of suckers, and hooklets when present (Fig. 50, 1). Evolution of the adult tapeworm continues directly from this point, the time required to attain maturity being several months under average conditions. The complete life cycle is shown graphically in Figure 49 for *T. saginata*; Figure 53 for *T. solium*. 

![Diagram](image)

**Fig. 49.** Life cycle of *Taenia saginata*. The definitive host is man (1) who harbors in his small intestine the adult worm (d, d, × 1/6 app.) and passes in his feces (h) the detached mature gravid segments (a, × 34 app.) containing the eggs (b, × 200 app.). The latter, already "hexacanth" embryos, are ingested by the intermediate host (2) into the muscles of which they migrate and encyst as larvae (c) known as cysticerci.

The latter (c, × 1 app.) when ingested by man (1) in raw, insufficiently cooked, or cured beef (i), excyst, attach themselves to the intestinal wall and grow rapidly to become adults composed of a head or scolex and many segments or proglottids as depicted in (d, d) and in Figure 50.

(After Kouri, in Kouri and Basanezo: Lecciones de Parasitología y Medicina Tropical.)
Fig. 50. *Taenia saginata* and *solium* contrasted.

*Center (top)* adult worm (×3/4 app.) reduced from wall chart (×7).

*Bottom row* (both species): *center*, egg with primitive vitelline membrane intact, ×400 app.; *left*, cysticerci in muscle, ×1 app.; *right*, cysticercus with scolex evaginated, ×4 app.

*Left column: T. saginata* (1) scolex, ×10 app.; (2) egg, ×400 app.; (3) mature gravid segment showing uterine branches, ×1.5 app.

*Right column: T. solium* (1) scolex, ×10 app.; (2) egg, ×400 app.; (3) mature gravid, segment showing uterine branches, ×1.5 app.

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Comparative Morphology. The essential characteristics of these two species of *Taenia* may be more readily comprehended by comparison of the summaries in Table X with Figure 50.

**Table X**

<table>
<thead>
<tr>
<th>THE TAENIAS, SAGINATA AND SOLIUM, CONTRASTED</th>
</tr>
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<tbody>
<tr>
<td><strong>Egg</strong></td>
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<tr>
<td></td>
</tr>
<tr>
<td><strong>Larval form</strong></td>
</tr>
<tr>
<td><strong>Scolex (head)</strong></td>
</tr>
<tr>
<td><strong>Rostellum (crown)</strong></td>
</tr>
<tr>
<td><strong>Proglottids</strong></td>
</tr>
<tr>
<td><strong>Branches of uterus (gravid segment)</strong></td>
</tr>
</tbody>
</table>

*Taenia saginata* (Goeze, 1782)

The gravid proglottids, or segments, average from 5 to 7 mm. in breadth and 20 mm. in length. In Figure 51 is shown such a mature proglottid made from a special preparation revealing some of the principal organs which are designated by name. The genital pores are lateral in position and roughly on alternate sides from segment to segment. When detached, the gravid segments of *T. saginata* are motile (Fig. 52), and as seen in fresh feces they manifest progressive undulating movements, apparently endeavoring to leave the fecal mass. Such segments are sometimes mistaken for flukes. It is thought that this motility may increase the possibility of the eggs being taken up ultimately by cattle, since spread of the eggs over a broader area is likely to occur. Contrary to the generally accepted view that eggs escape only upon disintegration
of isolated proglottids, recent investigations have shown that the uterus is ruptured upon separation from the inactive strobila or body.

The eggs of *T. saginata* are ordinarily indistinguishable from those of *T. solium*, except when the true egg shell or mother envelope is still intact (Fig. 50). In *T. saginata* this membrane, which is rather fragile, is characterized by the presence of delicate polar processes suspended in the extra embryonic yolk substance. These are not found in the otherwise similarly appearing eggs of *T. solium*, *Echinococcus granulosus*, or *Multiceps multiceps*.

In both species of *Taenia*, the eggs are usually of a pale buff
FOOD-INFESTING WORMS

to brown color and contain fully developed embryos with three pairs of hooklets which disappear before completion of the cysticercus stage. The embryonic envelope or the "embryophore" of

![Diagram of gravid proglottid motility]

FIG. 52. Motility of gravid proglottid of *Taenia saginata*, × 1 app. (Drawn from life.) The sketches from left to right illustrate the successive forms of a single mature living gravid segment as it undergoes more or less rhythmic contractions in the phenomenon of expelling the eggs from a ruptured end. (After Kouri. In Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical.)

the egg is a thick-walled structure perforated by minute pores or tubes, which under moderately high magnification have the appearance of fine radial striations.

*Taenia solium* (Linnaeus, 1758)

The adult stage of *T. solium* is exclusively a parasite of man, and frequently there is only one worm present. Several attached gravid proglottids (about the same size and general appearance as those of *T. saginata*) are usually passed together in the feces. Although these may manifest sluggish motility, as a rule they do not migrate actively. It is believed, therefore, that the gravid proglottids and eggs remain for the most part at or near the place of deposit, and are ingested principally by pigs (occasionally by sheep, dogs, man, or other primates).

It is thus possible for man himself to become the intermediate
host through contamination of his own food or mouth with *T. solium* egg-infested feces (perhaps his own). Furthermore, it is believed by some investigators that auto-infection sometimes takes place through gastro-intestinal upsets accompanied by reverse peristalsis. During this process, eggs may be carried from the small intestine backward into the stomach where action of the gastric juice frees the larvae. These are then returned to the intestine and ultimately become encysted as cysticerci in muscle, other tissue, or perhaps in a vital organ. In this way cysticercosis of the brain, a most serious complication, may arise.

**Life Cycle.** Essentially, the life cycle of *T. solium* is very similar to that of *T. saginata* as is graphically shown in Figure 53.

Within the gastro-intestinal tract of the intermediate host (Fig. 53, 2), the embryo larva, or “oncosphere” (b), emerges from its shell and penetrates the intestinal wall, thus gaining entrance to the lymphatics or blood stream, and eventually reaching striated muscle. Here the cysticercus or bladderworm develops. This is an ovoid body (c) which is frequently translucent, and measures from 7 to 10 mm. in length by 4 to 8 mm. in breadth. Inverted into the bladder is a miniature scolex which is characteristic of the species. Cysticerci are readily visible to the unaided eye, imparting to the meat a “measly” appearance. The average time required for development of infective cysticerci from the time of ingestion of eggs is about two months.

**Pathology and Symptomatology.** Ordinarily the adult worms of the two species of *Taenia* do comparatively little pathologic damage, and the associated symptomatology is correspondingly mild. At times there may be vague abdominal discomfort, hunger pains, chronic indigestion, persistent diarrhea, or diarrhea alternating with constipation. In debilitated persons, such symptoms as anorexia, loss of appetite, and hyperesthesia may arise. Eosinophilia of 10 per cent or more is likely to be manifested toward the end of the prepatent period. In infections with *T. solium*, perforation of the intestinal wall may occur in rare instances.

**Diagnosis.** A specific differential diagnosis can seldom be made from eggs of *T. solium* and *T. saginata*. Recovery of the scolex
Fig. 53. Life cycle of *Taenia solium*. The developmental cycle of this parasite is essentially the same as that of *T. saginata* except that the intermediate host of the cysticerci is the pig, rarely man himself.

The definitive host man (1) harbors in his small intestine the adult worm (d, d, X 1/5 app.) and passes in his feces (b1) the detached mature gravid segments (a, X 1/5 app.) containing the eggs (h, X 150 app.). The latter already containing “hexacanth” embryos (b) are ingested by the intermediate host (2) into the muscles of which they migrate and encyst as larvae (c, X 2 app.) known as cysticerci. The latter when ingested by man (1) in raw, insufficiently cooked, or cured pork (l), excyst, attach themselves to the intestinal wall and grow rapidly to be adults composed of a head or scolex and many segments or proglottids.

(After Kouri. In Kouri and Basnuevo: Lecciones de Parasitologia y Medicina Tropical.)
or a gravid proglottid and counting of the lateral uterine branchings of the latter must usually be relied upon for definite diagnosis. In order to render these branches visible, the gravid proglottids should be fixed in hot \( (70^\circ \text{C.}) \) solution of 3 per cent formalin for five minutes, then carefully pressed between two glass slides, cleared in 5 per cent KOH and held up to the light.

Clinical diagnosis of cysticercosis of subcutaneous tissue and somatic musculature in man is rarely made except in endemic areas. In the case of cysticercosis of the brain, differentiation from other similar symptom-producing lesions, including epilepsy, must be made. Group-specific intradermal and precipitin tests, employing extracts of the proglottids or of either one of the related cysticerci or even a non-human cysticercus such as \( C. \text{pisiformis} \), may be of some value here in differentiating further between these lesions.

**Treatment.** The preferred therapeutic agent is oleoresin of aspidium, administered as described for \( D. \text{latum} \) infection. An alternative drug which has been recommended is carbon tetrachloride employed as in the treatment of hookworm infections. When this drug is given for infections of \( T. \text{solium} \), every precaution should be taken to minimize nausea, vomiting, and reversed peristalsis.

**Prophylaxis.** Control and prevention of \( Taenia \) infections depend chiefly on two factors: (1) Personal hygiene in the matter of fecal cleanliness and food habits, particularly with regard to the consumption of raw meat likely to be infected. The possibility of self-infection with the eggs of \( T. \text{solium} \) should be kept in mind. (2) General hygienic measures such as strict sanitary rules regarding the disposal of human excreta in endemic areas, and the rigid inspection of pork and pork products according to federal standard requirements.

The following pertinent facts should also be remembered: (a) Pickled and salted pork is not necessarily safe food. (b) Thorough cooking of infected meat at \( 65.5^\circ \text{C} \) will kill cysticerci. (c) Chilling alone is not effective, but freezing renders the cysticercal larvae innocuous.
II. Man as Secondary Host

_Echinococcus granulosus_ (Batch, 1786) Rudolphi, 1805, and Hydatid Cyst

Mammals such as sheep, cattle, rabbits, pigs, and man serve as intermediate hosts of this flatworm. The clinical condition resulting from the infection is known as “hydatid disease.” Unlike the other intermediate hosts, man is not likely to be a source of infection for the primary hosts (dogs, wolves, coyotes, etc.). To become infected from man the primary host would have to eat his raw flesh or one of his organs containing one or more echinococcus cysts; this, of course, practically excludes man as a likely source of infection for the primary host.

**Epidemiology.** _E. granulosus_ infection of man occurs in cattle- and sheep-raising districts throughout the world. In North America, however, indigenous cases are comparatively rare. This observation is in accord with the fact that the adult worm is equally rare in dogs, whereas in the highly endemic areas dogs commonly harbor adults of _E. granulosus_. In a review of approximately 500 cases of human hydatid disease reported in the United States, it was estimated that probably not more than 5 per cent were actually contracted in this country. In some regions, however, hydatid disease appears to be rather common among certain wild animals.

**Morphology.** In so far as man is concerned, _E. granulosus_ is the smallest tapeworm of medical importance. Adult worms, as found in the small intestine of the dog (Fig. 54, z) and other primary or definitive hosts such as the wolf, the jackal, and occasionally the cat, are somewhat less than 1 cm. in length. The body, or strobila (Fig. 54, e; Fig. 55, left), comprises the head or scolex, neck, and three or possibly four segments or proglottids, of which the terminal one only is gravid.

The scolex (Fig. 54, d; Fig. 55, left) is somewhat pear-shaped,

*The possibility of man serving as intermediate or secondary host for _Taenia solium_ might also be discussed here. However, such occurrences are very rare compared to the more usual role of primary host played by man in the life cycle of this tapeworm.*
about 0.3 mm. in diameter, and is crowned with a “rostellum” armed with a double circle of hooklets varying in number from 30 to 50. Peripherally, the head is provided with four large, sym-

![Diagram of the life cycle of Echinococcus granulosus.]

**Fig. 54.** Life cycle of *Echinococcus granulosus*. The dog (1) is the definitive host, harboring in the small intestine the adult parasite (e, × 12.5 app.), infective eggs of which are expelled with the feces (h). These eggs (a, × 270 app.) already containing “hexacanth” embryos when ingested by man (2), pig (3), or sheep (3') or other intermediate hosts, are freed of their embryos, in the intestine, and migrate to the liver (i) and other viscera.

The cysts that form are known as “hydatids” (b) commonly varying from 1 to 10 cm. in diameter and contain many embryo scolices (c, d, × 80 app.), which may be ingested by the definitive host (1) in offal containing the infested liver, etc. These embryos evert as shown in (d), attach themselves to the intestinal mucosa, and grow rapidly to adult size giving rise to eggs appearing in the feces (h).

(After Kouri. In Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical.)

metrically placed suckers. The head narrows close behind the suckers to form the neck, and terminates at the first segment, which is often nearly square, frequently faintly delineated, and shows very little structure. The following segment or two shows evidence of rapid development to maturity, while the terminal
FOOD-INFESTING WORMS

segment is relatively much larger than the others and contains only the uterus filled with eggs.

Either before or shortly after detachment of the terminal gravid segment, the egg-filled uterus bursts open, liberating the eggs which are relatively few in number. They are still surrounded by the vitelline membrane, as shown in Figure 54, a. This soon disintegrates, leaving the morula-like hexacanth embryo, or “oncosphere,” enclosed only in the relatively strong radially striated covering, or “embryophore.” The eggs as found in the feces of the primary host (usually the dog) are indistinguishable from those of *T. saginata* and *T. solium* of man, and *Multiceps multiceps* of the dog, the bladder-worm (coenurus) of which causes “gid,” giddiness or vertigo in sheep.

**Life Cycle.** Infected dogs (Fig. 54, 1), which generally harbor considerable numbers of adult worms, scatter promiscuously the egg-containing excrement (4), contaminating pasture land, forage, and water. The eggs thus scattered may be ingested by swine (3), sheep (3'), or other less favorable herbivorous intermediate hosts (occasionally man, 2), in which they develop progressively into hydatid cysts in the manner described in the following paragraphs.

In the small intestine of the intermediate host (Fig. 54, 2, 3, 3') the oncospheres (hexacanth embryos) escape from the striated embryophore, penetrate the mucosa, enter the circulatory fluids, and are carried to various parts of the body. The greatest number accumulate in the liver (i), a lesser number in the lungs, and a few are scattered in other organs and tissues. At the end of three weeks, a larval cyst approximately 0.3 mm. in diameter has developed from each original surviving oncosphere, the tissues of the host reacting meanwhile to form a connective-tissue protective covering.

After five months, these cysts have attained a diameter of approximately 1 cm. and have developed an inner “germinal layer.” This is differentiated into an outer cuticular elastic, laminated part that encloses an inner granular part studded with small “bud-like” structures projecting inward, the cavity being more or less
filled with germinal fluid. These structures become the "brood capsules," some of which are frequently found free in the germinal fluid in fully matured cysts.

Numbers of invaginated hook-armed scolices are attached to the inner surface of each brood capsule. A capsule frequently bursts and everts, exposing the scolex-studded surface. In old cysts, there is found considerable sediment (hydatid sand) composed largely of scolices (single or clustered) and free hooklets.

Not infrequently in man, and occasionally in animals, daughter cysts are formed from the original or mother cyst. These may be endogenous (arising within the mother cyst) or exogenous (developing outside of the mother cyst). Various theories have been offered to explain their formation. It seems likely, however, that their immediate precursors are germinal buds or scolices formed, in some manner, in association with the original hydatid.

In Figure 55 the various possibilities in the evolution of hydatid cyst are depicted schematically. When offal or uncooked parts of animals suffering from hydatid disease are ingested by dogs, the scolices are liberated during the course of digestion and attach themselves to the intestinal mucosa, developing into mature worms in about three weeks.

If cysts are ruptured the infective scolices are freed to disseminate through the surrounding tissues where they may give rise to new cysts. The toxic fluid contents of the cysts may also cause symptoms of shock if absorbed into the general circulation. Thus an operation to remove an hydatid cyst is fraught with real danger.

The original primary cyst may remain unilocular or it may become multilocular, possibly in connection with the formation of daughter cysts. Multilocular cysts are thought by some authors to be due to the activities of a different but related race of *E. granulosus*, but this seems rather doubtful.

**Pathology and Symptomatology of Hydatid Disease.** The pathogenesis and associated symptoms of hydatid cyst in man depend largely on the location and the type of tumor produced. An embryo which lodges in a favorable site and develops as a simple unilocular cyst (accompanied by the usual inflammatory reaction
and fibrous tissue formation around it) may grow to considerable size over a period of years, causing no serious symptoms until adjacent structures are encroached upon. Incomplete insulation of

![Diagram of hydatid cyst](image)

**Fig. 55. Schema of development of the hydatid cyst. E. granulosus, x 25 app., last segment and cysts purely diagrammatic. (After Leuchart, Blanchard and Kouri. In Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical.)**

the fluid contents, or rupture of the cyst wall, may, however, be followed by a variety of allergic, lytic, and toxic symptoms of varying degrees of intensity. Lodgment of the embryo in the brain, the ocular orbit, the heart, the lungs, or bone produces most severe symptoms.

In contrast to the simple unilocular type of hydatid cyst, the so-called alveolar type is a malignant growth with a tendency to metastasize. The tissue involved is more or less spongy, with a fibrous matrix and gelatinous content. The tumor contains brood capsules which may or may not show scolices. As growth proceeds, the center of the tumor usually becomes necrotic.

**Diagnosis.** In addition to the clinical deductions that may be made by a skilled internist, a number of useful laboratory procedures are available in the diagnosis of hydatid disease:
1. In the differential blood count, an eosinophilia is present in a considerable percentage of cases.

2. An intradermal test is based on the injection of 0.2 cc. of sterile fresh hydatid fluid. If the reaction is positive, a wheal with an erythematous peripheral zone should appear within fifteen minutes, and should gradually disappear in about an hour, followed by a repetition of the original phenomenon several hours later.

3. A precipitin test, employing equal parts of phenolized hydatid fluid (unheated) and the patient's serum.

4. A complement fixation test, employing clear, sterile, unpreserved hydatid fluid from sheep or human cases as antigen.

The intradermal test is the one of choice except in old complicated cases; in these the complement fixation test is recommended. The precipitin test is good, but is said sometimes to be difficult to interpret correctly.

Treatment. All nonsurgical methods of treatment have thus far been unsuccessful. Surgical procedures are indicated in cases of unilocular cysts located in operable sites, but great care must be taken to prevent the escape of the cyst contents into the surrounding tissues.

Prophylaxis. Control and prevention depend upon hygienic and sanitary practices directed against the embryo-bearing eggs (oncospheres) passed by worm-infested dogs in their feces. The fondling of such dogs is particularly dangerous. In endemic areas, all carnivora should be prevented from eating offal from cyst-infected animals, such as sheep, cattle, and hogs.

III. Man as Primary and Secondary Host

Hymenolepis nana (v. Siebold, 1852) Blanchard, 1891

This small flatworm was formerly known under the names of Taenia murina and Taenia nana, and is commonly referred to as the "dwarf tapeworm." It is exceptional in that a second host other than man himself is not required, although recent experi-
ments show that the mealworm may serve as secondary or trans­mitting host of murine (mice and rat) strains.

Distribution. *H. nana* is widely distributed, being very com­mon in the south Atlantic states and in southern Europe. In some parts of Italy, 10 per cent of the children are said to be infected. The specific identity of this tapeworm with *H. nana* (var. *fraterna*) of the common rat seems probable.

Morphology. The adult worm, which usually lives in the upper ileum, measures from 10 to 40 mm. in length and from 0.6 to 0.8 mm. in breadth. It is composed of from 100 to 200 small segments, broader than long, all of which have unilateral genital pores. The globular scolex has four unarmed suckers and a retractile rostellum surrounded by a single row of from twenty-four to thirty hooklets. The cervical portion of the strobila is long and slender; the remaining segments become larger and broader as they mature, the distal ones being rounded with narrowing of the last few terminal segments.

The gravid segments are readily digested or ruptured within the intestine, the eggs being thus set free in the feces. These are round or oval in outline, and have two membranes, an outer and an inner, relatively far apart, and measure from 30 to 50 μ in di­ameter (Fig. 41, *M*; Fig. 56, *a*). The inner membrane envelops the hexacanth embryo and has two polar thickenings, from each of which arise four to eight filaments which are suspended in the intermembranous substance. The eggs are immediately infective as they are passed in the feces.

Life Cycle. Upon ingestion by man the embryo soon escapes from the egg into the small intestine and penetrates a villus where, in a short time, it becomes a cysticercoid. After four or five days of development a rostellum (encircled with hooklets) appears, and the larva leaves the villus, re-enters the small intestine, and attaches itself to the superficial mucosa. Rapid development con­tinues from this point, eggs appearing in the feces after about one month. In Figure 56 is shown graphically this simple one-host life cycle.

Epidemiology. In man, infection with *H. nana* takes place di-
FIG. 56. Life cycle of *Hymenolepis nana*. This parasitic worm (*c, g, × 3.5, app.*) is auto-infective, an intermediate host not being necessary for transmission. The eggs (*a, × 400 app.*) are expelled with the feces (*h*) of man and may be transferred directly to the mouth of man (*r*) by means of the fingers (*d*) or upon fecal-contaminated ground and vegetables (*i*).

In the villi of the small intestine the liberated embryos encyst as cysticercoids (*b*) which rupture the villi, become attached to the mucosa and grow to adult size (*c, g*). The terminal gravid proglottids (*g*) of the adult liberate eggs which are transported to the external environment with the feces to begin again the cycle.

[It was formerly believed that this parasite, known as the dwarf tapeworm of man, is transmitted by direct means only, i.e., without passing through an intermediate host. However, Bacigalupo (Evolution de l'*Hymenolepis fraterna* (Stiles) chez Pulex irritans L., Xenopsylla cheopsis (Rothchild) et Ctenocephalus canis (Curtis). *Ann. de Parasitol.*, 9:339–343, 1931) was able to infect several species of fleas and mealworms experimentally with this murine strain and observe development of the cysticercoid in the body-cavity of these insects. He reasoned, therefore, that an intermediate host, thus infected, might fall into a glass of milk or plate of soup and be ingested by a child as in the case of *Dipylidium caninum*.]

(After Kouri. In Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical.)
rectly from person to person. Children more commonly harbor *H. nana* than do adults, and this is particularly true in family and institutional groups. It is reported that 1 per cent of microscopic fecal examinations in the southern states show eggs of this tapeworm. The variety commonly occurring in rats is thought to be infective for man only in unusual circumstances.

**Pathology and Symptomatology.** *H. nana* is sometimes present in the small intestine in large numbers, causing abdominal pain with or without diarrhea, dizziness, insomnia, and occasionally convulsions and epileptiform attacks. A definite eosinophilia commonly occurs in infection caused by this tapeworm.

**Diagnosis and Treatment.** A specific diagnosis depends on finding the characteristic eggs in the feces or on identification of adult worms. With proper administration of drugs, attention to diet, and good sanitary habits, the prognosis is good.

Any of the common anthelmintics may be employed in treating *H. nana* infections, those of low toxicity being preferable. Hexylresorcinol, as used for *Ascaris infections* (page 148), is fairly satisfactory and reasonably nontoxic when given as directed.

**Prophylaxis.** Since the optimum viability of the eggs is immediately after their discharge from the bowel, soiled linen, toilet conveniences, and hands become the most likely media for transfer of egg-laden feces from anus to mouth. Personal hygiene, therefore, is a prophylactic measure of primary importance, especially in large families and institutions. The possibility of infection with murine strains should be considered, although opinions differ considerably as to the importance of this source of infection for man.

**IV. Man as Primary Host through Ingestion of Insect Intermediate Host**

*Hymenolepis diminuta* (Rudolphi, 1819) Blanchard, 1891

In contrast to *H. nana*, the common cosmopolitan murine parasite *Hymenolepis diminuta* seems to require an intermediate host. The infection is occasionally found in man (mostly in chil-
dren under three years of age). The intermediate hosts are various species of insects including the rat flea, mealworm, beetles, and myriapods. Infection occurs as the result of swallowing an infected intermediate insect host.

**Morphology.** The adult *H. diminuta* is much larger than that of *H. nana*, measuring from 20 to 60 cm. in length and from 3 to 5 mm. or less in width. The small, unarmed scolex is club-shaped with four small suckers and a depression at the apex in which there is a rudimentary rostellum. The number of proglottids may vary from 800 to 1000. The length of the mature proglottids is greater than the breadth. The eggs resemble those of *H. nana* but are somewhat larger, measuring from 50 to 80 μ in diameter. They also have a thicker, yellowish outer membrane or shell, and although the inner, colorless oncosphere membrane or embryophore is somewhat pointed at the poles, there are no filaments attached to them. The diameter of the outer membrane or shell varies from about 50 to 80 μ in its longest diameter, and the inner membrane, from 25 to 40 μ.

**Life Cycle.** When the infected intermediate host (Fig. 57, 2, 2') is swallowed by a suitable primary host (*i, i'*, the cysticercoid (b) escapes into the lumen of the small intestine, and the scolex attaches itself to the superficial mucosa. Subsequent growth is rapid, and the adult worms (c) begin to appear in about three weeks. The legend to this figure gives complete details of the cycle.

**Symptomatology, Treatment, and Prophylaxis.** In addition to symptoms ordinarily caused by tapeworm infection, cachexia is said to be common in children harboring the parasite in considerable numbers. Catharsis alone may be sufficient to expel most of the worms. However, one of the safer antihelmintics will ensure their elimination.

The most frequent source of infection in man is believed to be unbaked bread and poorly cooked cereals containing the infected intermediate insect host. Obviously prophylaxis against infection consists of avoiding the ingestion of any of the various intermediate hosts which still contain the living cysticercoid forms.
This parasitic worm requires both definitive and intermediate hosts, its usual definitive host being the rat. The adult inhabits the small intestine of its usual host, the rat or its occasional host, man. Its detached hexacanth embryonic egg-containing proglottids are passed with the feces and the eggs liberated at their disintegration.

The embryo-bearing eggs may be ingested by one of the rat fleas, meal-beetle, and so on, in which they transform into cisticercoids in the body cavity of the insect. If the definitive host rat, man in food ingests the insect which harbors the cisticercoids, the latter grow to be adults in the small intestine of the particular definitive host or man. Together with the feces, mature proglottids containing the eggs are expelled to begin again the cycle.

(After Kouri. In Kouri and Basnuevo: Lecciones de Parasitología y Medicina Tropical.)
**Dipylidium caninum** (Linnaeus, 1758) Railliet, 1892

The double-pored tapeworm, *Dipylidium caninum*, is one of the commonest intestinal parasites of dogs and cats, and occasionally it infects man.

**Morphology.** The scolex of *D. caninum* is small, roughly rhomboid in shape, has four unarmed suckers, and a diameter ordinarily of about 0.5 mm. The rostellum is club-shaped, retractile into the scolex, and armed with three or four circles of hooklets (about 60 in all) which are readily detached. The neck is short and slender and the "cucumber-seed-shaped" proglottids usually number less than 200 in the entire strobila. Each proglottid has two sets of genital organs with lateral symmetrically located genital pores. The individual eggs are fairly round, and vary in diameter from 30 to 50 μ. They are grouped in the uterus in packets of 5 to 20, and are seldom seen singly in the feces. The diagnosis must, therefore, be made from the proglottids, since they do not ordinarily disintegrate until some time after being passed with the feces.

**Life Cycle.** The proglottids (Fig. 58, a) passed in feces (h) finally disintegrate, releasing the egg capsules (b). When these rupture, the oncospheres or hexacanth embryos (c) are scattered about. Some get into the hair of the dog or fur of the cat and are ingested by the animal louse or by flea larvae, the adult fleas being incapable of ingesting the eggs. The oncospheres undergo very little change in the tissues of the flea larvae until the latter become adults, at which time the oncospheres develop into pear-shaped cysticercoids (d). When a dog (r) or a cat licks itself and ingests infected lice or fleas (z), the cysticercoids are soon liberated and attach themselves to the intestinal wall, where they grow rapidly and attain sexual maturity in about twenty days.

**Epidemiology.** In so far as is known, infection of the primary or definitive host by *D. caninum* can occur only through ingestion of the infected adult lice or fleas. In man, the infection is purely accidental or aberrant and not very common. Most of the cases reported have been in European children three years of age or younger.
Symptomatology and Diagnosis. Infected children seldom harbor more than one worm, which usually causes only mild symptoms such as indigestion, loss of appetite, and perhaps mild toxic manifestations. The diagnosis is usually made on finding in the feces the characteristic motile "cucumber-seed" proglottids, or the mother egg capsules, or eggs from disintegrated segments.

Treatment and Prophylaxis. Treatment of *D. caninum* infections is the same as that for other tapeworm infections. Since some
individuals have a strong inclination to fondle dogs and cats, the periodic administration of taenifuges to these animals will tend to lessen the hazard of infection in man. The precautionary training of children as to the risk of fondling canine and feline pets, admittedly difficult, is an excellent prophylactic measure.

Closely Related Tapeworms Reported as Rare Parasites of Man

In this group of flatworms with invertebrate intermediate hosts, there are two families (Davaineidae and Anoplocephalidae). Each contains a number of genera, adult worms of which have been reported in man. Species of the genera Davainea and Raillietina of the Davaineidae are common enteric parasites of scratching birds, such as chickens, turkeys, guinea fowl, and pheasants, and also of certain small mammals, including the rat.

Genera of the family Anoplocephalidae are parasites of sheep and other ruminants, horses, monkeys, and some of the higher apes. While the life cycles of a few of these hookless headed flatworms are partially known, many are still entirely unknown. Until recently it was generally believed that development was direct, as in the case of H. nana, but it has not been possible thus far to infect the proper definitive host by feeding the specific eggs. Stunkard (1937, 1939) recently has reported raising infective cysticercoids of Bertiella studeri and, earlier, of Moniezia expansa in certain mites. He predicts that other tapeworms of this group may be found to have similar modes of development with regard to the intermediate host.
CHAPTER X

PARASITIC WORMS OF THE LYMPHATIC AND CONNECTIVE TISSUES AND CIRCULATORY SYSTEM

FILARIA AND THE FILARIASES

Filaria are threadlike parasitic roundworms, the adults of which inhabit mostly the lymphatic and connective tissues. Some species give rise to definite pathologic lesions, while others apparently cause little or no serious tissue damage. Table XI gives important data regarding the human adult filaria, their larvae or microfilariae, and the principal known specific means of transmission. Figure 59 shows the various microfilaria of man, and will be helpful in differentiation as regards size and structure. This is of considerable importance, since specific diagnosis must often be based on the character of the larval embryos.

Wuchereria bancrofti (COBBOLD, 1877) SEURAT, 1921

Geographical Distribution and Habitat. The filarial worm Wuchereria bancrofti is indigenous in various parts of the warmer regions of the world. In the American tropics, it has been reported from the West Indies and the coastal areas of the northern part of South America. The region around Charleston, South Carolina, is the only definitely known endemic area in the United States.

The adults of W. bancrofti live in the lymphatic glands and vessels, while the sheathed embryos, which because of their small size are called microfilaria, find their way into the general circulation. They appear in largest numbers at night, hence the earlier designation, Microfilaria nocturna.
<table>
<thead>
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<th>Species</th>
<th>Adults</th>
<th>Larvae (Microfilariae)</th>
<th>Remarks</th>
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</thead>
<tbody>
<tr>
<td><em>Wuchereria bancrofti</em> (Filaria bancrofti)</td>
<td><strong>Adults</strong>&lt;br&gt;Male 40 by 0.1 mm.&lt;br&gt;Female 90 by 0.28 mm. Smooth cuticle. Bulbous anterior extremity. Occupy lymphatic glands and vessels.</td>
<td>Graceful curves; tail rather straight. Sheathed. 300 by 7.5 μ. Distance from head to V spot 50 μ; to break in cells 50 μ. Cells in head end form a curved line. Terminal cells do not fill up tail end. Nocturnal periodicity in peripheral circulation.</td>
<td>Transmitted by mosquitoes: Culex, Aedes, Anopheles. Causes elephantiasis, lymph scrotum, chyluria, etc.</td>
</tr>
<tr>
<td><em>Loa loa</em></td>
<td><strong>Adults</strong>&lt;br&gt;Male 27 by 0.3 mm.&lt;br&gt;Female 55 by 0.4 mm. Cuticle tuberculated. Anterior extremity like truncated cone. Wanders in subcutaneous tissues.</td>
<td>Angular curves; acute bend at tail. Sheathed. 240 × 7 μ. Distance from head to V spot 65 μ; to break in cells 40 μ. Tail is completely filled up with terminal cells. Diurnal periodicity in peripheral circulation.</td>
<td>Transmitted by species of a biting fly — Chrysops. Causes Calabar swellings. Worms often visit ocular region.</td>
</tr>
<tr>
<td><em>Acanthocheilonema perstans</em></td>
<td><strong>Adults</strong>&lt;br&gt;Male 40 by 0.07 mm.&lt;br&gt;Female 75 by 0.1 mm. Cuticle smooth. Anterior extremity club-shaped. Tip of tail shows two triangular processes. Found about root of mesentery.</td>
<td>Without sheaths, 200 by 5 μ. Posterior two-thirds taper to blunt ending. Cells to end of tail. Distance from head to V spot 45 μ; to break in cells 34 μ. Persists in circulation both day and night.</td>
<td>Transmitted by midges: Culicoides. No pathogenicity.</td>
</tr>
<tr>
<td>* Mansonella ozzardi* (Filaria ozzardi)</td>
<td><strong>Female</strong> only is known; 85-80 mm. in length; a pair of fleshy papillae at tail end. Lives in retroperitoneal tissue.</td>
<td>Without sheaths, 200 by 5 μ. Tail sharp pointed; cells not to end of tail.</td>
<td>Transmitted by midges: Culicoides furcatus. No pathogenicity.</td>
</tr>
<tr>
<td><em>Onchocerca volvulus</em> (O. caecutiens)</td>
<td><strong>Adults</strong>&lt;br&gt;Males 90 by 0.15 mm.&lt;br&gt;Females usually fragmented, up to 35 cm. by 0.4 mm. Cuticle striated. Found called up in cystic-like tumors under the skin.</td>
<td>Without sheaths. 250 by 7.5 μ. Found in cystic spaces in nodules and in cutaneous lymph spaces, very rarely in blood.</td>
<td>Transmitted by flies; Simulium and Eusimulium. Causes small subcutaneous cystic tumors, fever, skin eruptions, punctate keratitis, blindness.</td>
</tr>
<tr>
<td><em>Dracunculus medinensis</em></td>
<td><strong>Male</strong> from Leiper's monkey 22 mm.&lt;br&gt;Female 89 to 90 cm. long by 1.5 mm. wide. Smooth white body. Anchoring hook at tail end. Female lives in subcutaneous tissue of lower extremity.</td>
<td>Without sheaths. 600 by 20 μ. Tapering outline; gut present. Long slender tail. Cuticle striated. Extruded from break in skin of patient. Not present in blood or tissues.</td>
<td>Larvae develop in Cyclops. Man infected by drinking water containing Cyclops. Causes &quot;Dracunculiasis.&quot;</td>
</tr>
</tbody>
</table>
The Adult Worm. The mature worms are small, creamy white, threadlike nematodes with a smooth cuticle, unarmed mouth, and tapering, somewhat rounded ends. The smaller male worm (averaging 40 mm. long by 0.1 mm. wide) has about fifteen pairs of perianal and postanal papillae, and its tail has a tendency to curve ventrally. The female is twice the size, or even larger, than the male. The two uterine tubes of the pregnant female, which contain numerous larvae in various stages of development, can be clearly seen when held up to the light. The vulva is near the anterior end, and the anus near the posterior end. The females outnumber the males and in both sexes the life span is believed to be several years. When the embryos are deposited in the lymph channels, they tend to straighten out, eventually elongating the external membrane into a closed ensheathing tube.

Microfilarial Periodicity. The free, sheathed microfilaria are swept periodically into the general circulation. The time of their appearance, however, is peculiarly cyclical. Various explanations of this phenomenon have been advanced, the most probable one being that the periodicity is an evolutionary correlation with the nocturnal biting habits of the insect vector. In localities where *Culex quinquefasciatus* (a night feeder) is the transmitting agent, the optimal time of appearance of the microfilaria in the peripheral circulation is several hours after sundown; where *Aedes pseudoscuteellaris* (a day feeder) is the transmitting agent, the microfilaria appear peripherally during the day. In some of the Pacific islands, it is reported that there is no microfilarial periodicity, which would seem to indicate a ubiquitous biting insect vector.

Development of the Embryo in the Mosquito. Within two hours after reaching the stomach of the mosquito, the active embryo usually manages to escape from its sheath, aided probably by the relative fixity of the enveloping sheath in the surrounding viscid blood. The free embryo then bores its way through the stomach wall, usually reaching the thoracic muscles within twenty-four hours. During the course of another two days, it is observed to grow rapidly in length, the so-called anterior and posterior...
"V-spots" becoming more pronounced. By the end of the first week of development, the growing embryo has attained a length of approximately 250 μ and a width of from 15 to 25 μ. (The anlage of the alimentary canal is said at this time to be visible by transmitted light.)

The first ecdysis (shedding of the sheath) now occurs, and the larvae show three subterminal caudal papillae. They shed their skins again in a few days, becoming the longer filiform mature larvae, and soon appear in the fleshy proboscis of the transmitting mosquito where they attain an ultimate average length of 1.5 mm. and a breadth of approximately 20 μ. Having completed their development—which requires about three weeks more—the larvae move down toward the terminus of the labium, where "Dutton's membrane" intervenes between the labium and the two hinged labellae.

**Development of the Adult Worm in Man.** When the infested mosquito bites, the waiting larvae succeed in breaking through Dutton's membrane when it is stretched by the wide separation of the two hinged labellae. Formerly it was believed that the escaping larvae entered directly through the puncture wound, but apparently it is not necessary for them to do this, since they are able to penetrate the unbroken skin. Having passed through the skin of the definitive host (man), the larvae eventually reach the lymphatics where they slowly grow to maturity. It has been shown experimentally that the new generation of microfilaria appear in the peripheral blood of man about one year from the time of the original inoculation.

**Pathology.** In *W. bancrofti* infections, the pathologic changes are associated directly or indirectly with the presence of adult forms, which cause cellular reaction. According to O'Connor (1932), the specific type of tissue reaction depends largely on the kind of tissue involved, and on whether the worms are living or dead. They are frequently found closely coiled up in nodular dilatations of the lymphatics, in lymph varices, in lymph nodes or tissues, in lymphatic trunks, and even in the thoracic duct. In lymphatic channels of compact tissues, the living adult worms
merely produce dilatation of the vessels; in lymphatic channels of connective tissues, varying degrees of hypertrophy occur, associated frequently with lymph stasis and edema.

The worms are more and more confined by proliferative processes (in and around the lymph vessels), or by thrombi, to the formation of which embryonated eggs and larvae may contribute. Thus harassed, they soon die and ultimately become impregnated with deposits of calcium, having previously been subject to various cellular infiltrations somewhat similar to trichina encystment in muscle. In this way encapsulation and calcification of the dead and dying worms are brought about. Some investigators believe that toxic products discharged from the degenerating worms are responsible for the inflammatory processes which frequently develop around them. The urticaria which occurs in filarial patients who are not sensitive to food proteins is an indication of the possible allergic nature of this reaction.

Following the death of adult worms, the microfilaria soon disappear from the peripheral blood. However, degenerated microfilaria may also provoke cellular reactions, thus increasing the foci where obstruction occurs. Such blockage of lymph channels and vessels—primarily by degenerating adult worms, and secondarily by embryos—accounts, it is thought, for the later manifestations of filariasis known as "elephantiasis." Some workers, notably Grace (1931), believe that the lymphangitis leading to this regional enlargement is due to hypersensitivity to certain strains of hemolytic streptococci; other workers have found no evidence of such allergic phenomenon in focal centers of inflammation. If bacteria are present, it is believed by many that they are probably secondary invaders through the low-resistant skin covering the involved area. Descriptions of the character and the enormous size which elephantoid lesions often attain may be found in the more detailed texts dealing with *W. bancrofti* infections.

**Symptomatology.** During the long incubation period—about one year from the time of infection to the appearance of the infective larvae (microfilariae) in the peripheral blood—no clearly related symptoms have been observed. Following this phase, a
second comparatively symptomless one usually ensues, varying from a few months to many years; during this period, tissue changes may be in progress around the adult worms. The third or clinical phase of *W. bancrofti* infection is usually acute inflammatory. Not infrequently the acute attack is ushered in by malaise, frontal headache, mental depression, and urticaria suggestive of a toxic origin. Gradually these symptoms become less severe and the involved lymph channel areas are less painful. Finally, the fourth or chronic phase develops, with regional enlargement of the involved area, or the development of lymphocele, frequently accompanied by rupture in the less fibrosed types.

**Diagnosis.** The diagnosis of *W. bancrofti* infection rests on finding the specific microfilaria in the peripheral blood, in the chyle, or in other similar fluid. The optimum period to search for these larvae is ordinarily between 10:00 p.m. and 2:00 a.m. A thick blood film made and stained as described for the diagnosis of malaria parasites (Appendix IV) gives fairly satisfactory results. However, where feasible, it is advisable to look first for living forms. Later, when microfilaria are no longer being produced, owing to the death and ultimate calcification of the adult worms, roentgenography is a valuable aid in diagnosis. Finally, an intradermal group reaction may be tried. The source of antigen most readily obtained for this test is *Dirofilaria immitis* (the dog heartworm).

**Treatment.** There is no specific treatment for *W. bancrofti* infections. Arsenic compounds are reported to produce a favorable action in chyluria, and O'Connor (1935) stated that he had successfully treated the acute lymphangitis with local injections of sulpharsphenamine directed against the adult worms. Clinical manifestations such as lymphatic varices, chylous hydroceles, and elephantiasis are conditions requiring surgical intervention to effect a cure, or at least to alleviate the handicaps. In elephantiasis of the lower extremities, beneficial results have been obtained with the Knott (1938) pressure bandage technique.

**Prophylaxis.** The prevention of *W. bancrofti* infection depends primarily on individual protection and on the destruction of
larvae and adults of the transmitting culicine mosquitoes. Since the usual vector is a domestic mosquito frequently abundant in houses, both individual and general community house screening are important preventive aids. Logical community control measures require identification of the particular mosquitoes concerned in local transmission of the infection. Equipped with this information, local health authorities may intelligently direct their energies against the specific vector.

**Wuchereria malaya** (Brug, 1927) Rao and Maplestone, 1940

A sheathed microfilaria of man resembling somewhat that of *W. bancrofti* has been reported from southeast Asia and the Malay Archipelago. Since the adults have not been definitely known until recently, the larval form has been referred to as *Microfilaria malayi* (Brug, 1927) Faust, 1929. They are said to closely resemble *W. bancrofti* and have been named *W. malayi* (Brug, 1927) Rao and Maplestone, 1940. Elephantiasis occurs frequently in persons harboring this microfilaria, but thus far a specific relationship has not been definitely established. A nocturnal periodicity of this microfilaria in the peripheral circulation has been reported but it is not so clear-cut as in *W. bancrofti*. Several species of *Mansonoides* mosquitoes, it is believed, may serve as intermediate hosts.

**Loa loa** (Cobbold, 1864) Castellani, 1913

**Geographical Distribution and Habitat.** Infection due to the filarial worm *Loa loa*—often referred to as the loa, or eye-worm—occurs commonly in west and central Africa (particularly the Congo region). Cases which have been reported from the West Indies were probably not indigenous. The adult worms are about the same size as those of *W. bancrofti* and live in the connective tissues, where they move about freely. They often appear close to the surface, frequently about the eyelids or beneath the conjunctiva. They cause considerable local irritation in the form of areas of edema about the eyes and on the arms and legs. These so-called fugitive or "Calabar swellings," which are often as large
as a hen's egg, appear suddenly, are painless, do not pit on pressure, and usually subside after a few days. Some workers regard these swellings as allergic phenomena.

**Characteristic Features of Infection.** In man, development of *Loa loa* is extremely slow; from three to six years may elapse before the microfilaria appear in the blood. An outstanding feature of this condition is the frequent absence of symptoms related to the wanderings of the worms in connective tissues throughout the body, except when they come close to the surface. Microfilaria are present in the peripheral blood during the day and were formerly designated *Microfilaria diurna* in contradistinction to the nocturnally appearing, morphologically similar microfilaria of *W. bancrofti*. They are likewise sheathed, and are about the same size as those of *W. bancrofti*. In dried stained preparations they are differentiated by their angularity from the graceful sweeping curves of the microfilaria of *W. bancrofti*. Furthermore, the nuclei forming the central column in the microfilaria of *Loa loa* are larger than those of *W. bancrofti* and stain less deeply.

**Transmission.** Several species of the genus *Chrysops*—tabanid flies with spotted wings (Fig. 70)—are now considered to be the natural transmitters of *Loa loa* in Africa. Like the culicine transmitter of *W. bancrofti*, the microfilaria are sucked into the stomach with the fly's blood meal, where they develop and become infective in about ten days. They make their way from the thoracic muscles to the base of the proboscis, preparatory to escaping to a new host when the infected fly feeds. As many as 4 or 5 per cent of wild *Chrysops* caught in Calabar, West Africa, have been found to harbor infective forms of *Loa loa*.

**Pathology and Symptomatology.** The *Loa loa* adult is essentially a subcutaneous parasite, frequently appearing so near to the surface that its movements may be readily followed. It has been observed to travel as much as an inch in two minutes. Aside from itching, irritation, and edema produced during its migrations, *Loa loa* apparently causes comparatively little permanent damage to tissues, and produces no significant symptoms.

**Diagnosis.** Identification of the microfilaria when they occur in
the peripheral blood and recognition of the adult worms removed from their burrows beneath the conjunctiva or skin, constitute the most reliable means of diagnosis. In addition, a group filarial skin test similar to that mentioned in the diagnosis of *W. bancrofti* may be employed.

**Treatment, Prognosis, and Prophylaxis.** Relief from the recurrent annoyance of these parasitic worms when they come close to the surface of the body can be secured only by their surgical removal; otherwise a favorable prognosis should not be ventured. Prevention consists largely in avoiding the *Chrysops* bites when in endemic areas. Efforts at community control against *Chrysops* would be useful, but the habits and wide range of this insect make such a measure difficult to carry out in practice.

*Acanthocheilonema perstans* (Manson, 1891) Railliet, Henry and Langeron, 1912

**Distribution.** The small roundworm, *Acanthocheilonema perstans*, often called the “persistent filaria,” occurs extensively throughout the tropical regions of both hemispheres. In Africa, it is frequently associated with *W. bancrofti* and *Loa loa*, the ocular filaria. *A perstans* is found in some of the primates as well as in man. The adults probably localize chiefly in the mesentery and the retroperitoneal connective tissues.

**Morphology and Transmission.** In a general way the adults of *A. perstans* resemble the adults of *W. bancrofti* and *Loa loa*, but the sheathless microfilaria are considerably smaller than the sheathed microfilaria of *W. bancrofti* and *Loa loa*; furthermore they show no definite periodicity. In Africa, intermediate hosts have been found to be certain species of *Culicoides*, but the detailed epidemiology is not known.

**Pathology and Symptomatology.** Little can be said regarding the pathology and symptomatology of this filarial worm in man. However, in a related species in New World monkeys, the adult worms are found localized in the mesentery and other similar tissues, where they cause considerable irritation and fibrinous exudation.
Diagnosis and Prophylaxis. Diagnosis of *A. perstans* infection depends upon identification of the specific microfilaria and their differentiation from those of *Mansonella ozzardi*, which are also sheathless and likewise smaller than the sheathed microfilaria already considered.

Control or eradication of the transmitting midge is scarcely feasible, because of its habit of breeding in jungles and swamps. When exposed to these extremely small insects, avoidance of their bites is exceedingly difficult.

*Mansonella ozzardi* (Manson, 1897) Faust, 1929

*Mansonella ozzardi* is a filarial worm of the New World. Its microfilaria have been reported from the northern countries of South America, northern Argentina, and several foci in the West Indies. According to the meager data available, the adult worms are about the size of the other filarial worms already described.

Like those of *A. perstans* the microfilaria (Fig. 59) are sheathless, but they are somewhat larger and have pointed instead of blunt tails. The insect vector is thought to be a species of *Culicoides* native to the endemic areas. The epidemiology has not been fully investigated. Adult worms apparently have little pathologic effect and cause few symptoms.

*Onchocerca volvulus* (Leuckart, 1893) Railliet and Henry, 1910

Distribution. The filarial worm *Onchocerca volvulus* has been found widely scattered in equatorial Africa, most commonly on the west coast and in the French Congo. In the Western Hemisphere a similar filaria worm, believed to be the same species, has been reported from southern Mexico and western Guatemala. In the American variety of onchocerciasis, according to Strong (1934), the site of infection is more often the head (scalp and vicinity of orbit) than other parts of the body. Involvement of the orbit sometimes leads to blindness, and accounts for the popular designation of the parasite as the "blinding filaria."
Morphology, Biology, and Life Cycle. The adults of *O. volvulus* are found in the subcutaneous tissues, usually enclosed in fibrocystic nodules varying from 2 to 30 mm. in diameter. Several male and female worms are usually found in each nodule, intertwined in meshes of connective tissue. Hence the name "convoluted filaria." The viviparous females average about 30 cm. in length and from 0.3 to 0.4 mm. in diameter, and the males, from 2 to 4 cm. in length and from 0.15 to 0.2 mm. in diameter.

The larvae, which are approximately the same size as those of *W. bancroftii*, may be quite numerous in the cystic fluid and adjacent subcutaneous tissues, but are seldom found in the blood. These microfilaria, after escaping from the mother worm, promptly leave their sheaths and enter the lymphatic and subcutaneous tissues in the near vicinity. Because of a positive phototropic tendency, these embryos migrate into the adjacent cutaneous layers and, in Central America, not infrequently invade...
the cornea, where they produce a punctate, vascular, or interstitial keratitis.

Transmission. Studies by Blacklock (1926) and others indicate that certain species of Simulium serve as important intermediate hosts of O. volvulus. The developing embryos undergo two ecdyses in the thoracic muscles of the insect in the course of six days or more. The resultant “filiform” infective larvae soon migrate to the labium of the vector, prepared to enter a fresh host at the time of the insect’s next blood meal. In man, the incubation period may vary from four months to one year.

Pathology and Symptomatology. In the majority of cases of O. volvulus infection, the presence of the adult worms causes local cellular reactions, which result in fibrous encapsulation. In endemic areas in southern Mexico and Guatemala the lesions thus formed occur very frequently on the scalp or near the eye; whereas in Africa, the nodules are usually found on other parts of the body. The reasons for this difference in localization of the lesions are not known. Involvement of the orbit often leads to serious complications due, it is believed, to migrations of the microfilaria. One of these complications—keratitis—has already been mentioned. Total blindness is sometimes the ultimate sequel to extensive involvement of the eyeball itself.

Diagnosis. The diagnosis of O. volvulus infection may sometimes be made by finding the microfilaria in the tumor aspirate; at other times this aspirate may contain no microfilaria. Biopsy specimens of surrounding tissues, such as the skin and the conjunctiva, when placed in warm, physiologic saline solution, often afford positive evidence of the presence of microfilaria. Only in exceptional cases are the microfilaria found in the blood stream. Complement fixation tests, employing alcoholic extracts of the adult worm, have been found of value in diagnosis.

Treatment and Prognosis. Treatment of infection caused by O. volvulus varies with the site of the lesion. Early enucleation of the nodules is a comparatively simple surgical procedure in most instances. For tumors in the vicinity of the orbit, radical removal is imperative. It is believed that the injection of anthelmintics into
the center of the nodule kills the worms, which may then be absorbed. To kill the microfilaria, the general administration of tartar emetic and other similar drugs have also been tried.

For involvement of the interior of the eye, injection locally of 0.1 per cent solution of plasmochin has been suggested. If the lesions do not endanger the eye, the prognosis is believed to be good. Involvement of the orbit must always be regarded with grave apprehension.

**Prophylaxis.** Because *Simulium* breeds in rapidly flowing streams, destruction of the larvae of this vector is a difficult and expensive procedure. The use of larvicides, however, may be practicable on a limited scale where conditions permit. Strong, *et al.* (1934) have shown that surgical removal of the tumors as soon as they appear diminishes not only ophthalmic complications but also larval infection of the transmitting insects.

*Dracunculus medinensis* (Linnaeus, 1758) Gallandant, 1773

*Dracunculus medinensis* is variously called guinea, Medina, serpent, or dragon worm. Unlike the other human filaria worms, *except O. volvulus*, the females of *D. medinensis* are much larger than the males. The vulva of the gravid female is somewhat atrophied and the anus absent.

**Distribution.** The guinea worm occurs in some parts of Africa, Arabia, India, the Guianas, and Brazil. It was well known in ancient times, and it is thought that the troublesome “fiery serpent” mentioned by Moses in the account of the trek of the Israelites from Egypt back to Palestine was *D. medinensis*. Various wild and domestic animals, including carnivora such as the fox, raccoon, and mink, have been found to harbor similar filarial worms, but no cases in man from such sources of infection have thus far been reported. Dogs have been found infected naturally and may be readily infected artificially.

**Morphology.** The female worm is round, threadlike, and may reach a length of 100 cm. and a diameter of 1.5 mm. It lives in the subcutaneous and intermuscular tissues, particularly those of the lower extremities. The uterus of the pregnant worm is a fairly
straight tube, occupying the greater portion of the body and is filled with sharp-tailed, transversely striated, unsheathed embryos averaging 600 μ (0.6 mm.) by 20 μ in size. The tip of the tail of the mother worm is bent, and apparently serves to keep her anchored during discharge of the larvae which are expressed as a result of the gradual prolapse of the uterus when the head of the worm is ruptured after protrusion from the skin.

The male worm is comparatively small, usually from 1 to 3 cm. long and less than 0.5 mm. in diameter. Copulation apparently takes place early in the life cycle, since males are not found later than six months after the initial infection with larvae.

The gravid females are said to develop in retro-esophageal tissues and body cavities, migrating later to the subcutaneous tissues, frequently to the distal extremities where they are usually first detected. First a papule appears; this terminates after a day or two in a blister which soon ruptures, revealing the worm's head. If the affected area is immersed in fresh water at this time, a loop of the uterus will prolapse through the worm's mouth, or the body wall will burst open, discharging swarms of motile first-stage larvae. This process may be repeated several times, under similar conditions, until all the stored larvae have been discharged, and the mother worm dies.

**Life Cycle and Transmission.** If suitable species of the crustacean cyclops are present in water containing the active, wiry larvae, they ingest these larvae which promptly break through the intestinal wall into the body cavity, where one or two ecdyses or molts take place. In from ten to twelve days this stage of larval development is completed; then ingestion of the infected cyclops by the proper definitive host (man) makes possible the gradual evolution of the adult worms. The young larval worms, freed from the cyclops by digestion, migrate through the intestinal wall of the host, and in suitable loci such as loose connective tissue reach maturity in from six months to a year.

**Pathology and Symptomatology.** During the long incubation period, practically no related symptoms are observed. However, a few hours prior to the development of the local cutaneous
lesion, there may arise marked prodromal symptoms such as erythema, urticarial rash with intense itching, nausea, vomiting, dyspnea—all believed to be the result of the activities of the pregnant worm.

**Diagnosis.** The diagnosis of *D. medinensis* infection is seldom, if ever, made before the adult female worm appears at the surface to larviposit. At this time the local and toxic symptoms become manifest. In fact, in most cases the patient applies for medical aid only after the pregnant worms have begun to discharge the larvae.

**Treatment.** The natives of the countries in which *D. medinensis* occurs have long employed a simple method for removal of the pregnant worm. They gradually extract the worm by carefully rolling it onto a small twig, a little at a time each day, until it has been completely removed from its burrow. This method is somewhat hazardous because of the chance that the embedded part of the worm may be broken off within its tunnel. Such an accident would be likely to result in inflammation and abscess formation.

Some clinicians report good results from injecting the worm with 0.1 per cent solution of acriflavine or bichloride of mercury, and extracting it—usually without difficulty—after about twenty-four hours. If the worm is injected with a 10 per cent solution of collargol, it may be located by x-ray and then dissected out. Repeated cold water douching of the ulcer and of the area containing the worm stimulates the expulsion of larvae, after which it is said the worm may emerge spontaneously, or it may be extracted without risk of breaking it. The prognosis is usually good unless secondary infection intervenes, and pyemia or septicemia develop.

**Prophylaxis.** Control and prevention of *D. medinensis* infection consist essentially in (a) prohibiting afflicted persons from bathing, wading, or swimming in water that is used for drinking and domestic purposes; and (b) instructing all individuals not to drink water from suspected sources. In endemic areas, various means of protecting isolated, as well as community, water sources have been more or less effectively carried out. It must be said, however, that primitive, superstitious, and custom-fast peoples
generally prove to be somewhat insensible both to prohibitions and to attempted education along hygienic and sanitary lines. The introduction of certain species of fish into pools, ponds, and reservoirs, under favorable circumstances, may serve a useful purpose in the control of the infective transmitting cyclops.

SCHISTOSOMA (BLOOD FLukes) AND THE SCHISTOSOMIASES

Schistosoma (Weinland, 1858)

Distinctive Features. Unlike the other parasitic flatworms, the sexes of the Schistosoma are separate, although closely associated during the mating period. Other differing characteristics are (1) an absent or suppressed redial stage in the molluscan phase of the life cycle; (2) the forked tails of the cercariae; (3) absence of encystment of cercariae, which are free-swimming and enter the skin or mucosa of the definitive host by direct penetration.

Morphology and Life Cycle. Since the three species of this genus which are found in man (Schistosoma haematobium, mansoni, and japonicum) are somewhat similar in morphology and life cycle, only a composite description will be given.

Because the sexes are separate, it may be well to begin with the eggs as they appear when passed in the urine or in the feces (Fig. 60, h). The egg (a), subterminal spined in this instance, contains a ciliated miracidium (b). In water, under suitable conditions, the eggs hatch in a few hours. The miracidia swim about rapidly in search of a satisfactory molluscan host (2), the tentacles of which they are able to penetrate by means of their cilia, aided by a lytic substance from glands situated near the pointed anterior end. Miracidia which fail to find a suitable host within a few hours die.

After the initial penetration, the miracidia lose their cilia and migrate to the viscera of the intermediate host (the snail) where they encyst. Within each of these so-called sporocysts (Fig. 60, c), daughter sporocysts develop, and in the course of a few weeks the visceral mass (d) of the snail contains many small, tubelike daugh-
Life cycle of *Schistosoma mansoni*. The adult parasites, male and female (f, × 4 app.), inhabit principally the smaller ramifications of the mesenteric veins of man (z). To deposit her eggs (a, × 130 app.) the gravid female migrates to the veins of the sigmoid and rectum (sr). The eggs are forced through the veins and intestinal wall into the lumen of the intestine aided by their sharp subterminal spines and peristaltic muscular contractions of the intestines. Mixed with feces (h), the eggs (a) reach the external environment in an embryonate state. The ciliated embryos (b), called miracidia, soon escape from the eggs upon contact with water under suitable conditions.

Miracidia (b, × 130 app.) swim about rapidly by means of their many cilia in search of a suitable intermediate snail host (s) in which to undergo primary (c) and secondary (d) sporocyst formation. In the latter the cercariae (e) are formed, which abandon the snail and swim about freely (e', × 90 app.) until they encounter the definitive host (z) whose skin (p) they (e”) penetrate and finally reach the portal vein where they become adult males and females (f), localizing in the mesenteric veins. The gravid females produce the characteristic subterminal spined eggs (a) which are forced through the veins and intestinal wall to the gut lumen and are transported in the feces (h) to the external environment to begin again the cycle.

(After Kouri. In Kouri and Basnuevo: Lecciones de Parasitolología y Medicina Tropical.)
ter sporocyst larvae (e). Five to seven weeks after the entrance of
the miracidia into the mollusc, sporocyst development ceases and
rapid transformation into infective “forked tail” cercariae takes
place.

The actively motile cercariae (Fig. 60, e', x 90 app.) escape
from the snail host and swim about, attracted through agitation
of the water, perhaps by the specific definitive human host (r). They
(e") readily penetrate the skin of this host (p), shed their tails,
and after gaining access to the circulation eventually reach the
portal system where they develop to maturity within a period of
about three weeks. The adult male worms average 1 cm. in length;
the females are considerably longer but much more slender. Indivi-
dual differences between the males and females, and between
the eggs in the three species, together with common snail hosts,
are summarized in Table XII.

The mature female enters the “gynecophoric” canal of the male
(a channel formed by the infolding of the lateral margins of his
broad, flat body) and usually remains there in copulation until
she is ready to deposit her eggs. Figure 61 shows the relationship
of male and female during the period of fertilization of eggs.

Preparatory to laying her eggs, the pregnant female leaves her
consort and works her way against the portal current until she
reaches a small venule in the vesical or hemorrhoidal plexus.
Then she forces her way still further by expanding the vessel
that is slightly narrower than her own transverse diameter. When
further progress is not possible, she deposits her eggs and with-
draws. This unique procedure is illustrated in Figure 62 for the
lateral spined eggs of S. mansoni and for the terminal spined eggs
of S. haematobium.

It should be noted that the eggs of both species are deposited
with the spines pointing in the direction of blood flow. This fa-
cilitates penetration and escape of the eggs into the bladder or
rectum, as the case may be. The spineless eggs of S. japonicum are
deposited en masse, blocking the venule, followed by thrombosis,
necrosis, ulceration, and with discharge of the eggs into the intes-
tine.
Fig. 61. *Schistosoma japonicum* (× 15 app.), showing the female (♀) partially hidden in the gynecophoric canal formed by the male (♂). (After Mense. In Stitt: Diagnostics and Treatment of Tropical Diseases. Edition 5. Copyright the Blakiston Company, Publishers.)

Fig. 62. Diagram representing deposition of eggs (× 20 app.) by *S. mansoni* (A) and *S. haematobium* (B) in blood vessels, and their passage to the exterior. 1, Anterior sucker; 2, posterior sucker (acetabulum); 3, vaginal orifice; 4, uterus containing eggs. (After Manson-Bahr. Manson's Tropical Diseases. Edition 11. Courtesy of Cassell & Company.)
### Table XII

**DISTINGUISHING FEATURES OF HUMAN SCHISTOSOMES**

(From Hegner, Augustine, and Root)

<table>
<thead>
<tr>
<th></th>
<th><em>S. haematobium</em></th>
<th><em>S. mansoni</em> (Sambon, 1907)</th>
<th><em>S. japonicum</em></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adult male</strong></td>
<td>Size, 12–14 mm. long. Cuticula finely tuberculated. Intestinal crura unite late so that united region of intestine is short. Testes large, 4 in number.</td>
<td>12 mm. long. Cuticula grossly tuberculated. Intestinal crura unite early so that united region of intestine is long. Testes small, 8 in number.</td>
<td>9–22 mm. long. Cuticula non-tuberculated. Intestinal crura unite far back, united region being one-fifth to one-sixth of body length. Testes slightly lobate, 7–8 in number.</td>
</tr>
<tr>
<td><strong>Adult female</strong></td>
<td>Size, 20 mm. long. Uterus long and voluminous and contains a number of eggs. Ovary in posterior half of body.</td>
<td>14–15 mm. long. Uterus short, contains usually from 1 to 3 eggs at a time. Ovary in anterior half of body.</td>
<td>12–26 mm. long. Uterus well developed, occupies about half of postacetabular region, contains 50–300 eggs. Ovary about the middle of the body.</td>
</tr>
<tr>
<td><strong>Eggs</strong></td>
<td>Terminal spine, 150 by 60 μ, usually deposited in veins of bladder; escape with urine.</td>
<td>Lateral spine, 150 by 60 μ, usually deposited in veins of rectum; escape with feces.</td>
<td>Abbreviated spine, 80 by 65 μ, deposited in portal system, enter intestine higher than eggs of <em>S. mansoni</em>.</td>
</tr>
</tbody>
</table>

### The Schistosomiases

**Geographical Distribution.** The three schistosome flukes give rise to somewhat different clinical manifestations in man, two of which have been recognized since ancient times. Their distribution, geographically and regionally, is interesting (Fig. 63).

The endemic hematuria of the lower Nile, known as "bilhar-
BLOOD FLUKES

zia,” * occurs also in other parts of Africa but not to the same extent as in the Nile delta. It has been reported in islands adjacent to Africa, parts of the Mediterranean littoral, and the countries of the Near and Middle East. Apparently it is not found endemically east of the Indus River in Asia or in the Western Hemisphere.

Intestinal schistosomiasis caused by *S. mansoni* and its subterminal spined eggs occurs quite extensively in various parts of Africa, but is not found so commonly in adjacent parts of southern Europe and the Near East as is *S. haematobium*. On the other hand, *S. mansoni* infections have been reported as commonly occurring in the Lesser Antilles and northern South America east of the Andes. This infection is believed to have been introduced into the Western Hemisphere by African slaves who were infected with *S. mansoni* upon arrival in the West Indies, the adjacent countries, and Brazil.

Epidemiology. As many of the African slaves must have been infected with *S. haematobium* as well as *S. mansoni*, the most plausible explanation for the absence of *S. haematobium* infection

* After Bilharz (1851), who first recovered the adult worms.
from the Western Hemisphere is that the *S. mansoni* miracidia found suitable snail hosts, whereas those of *S. haematobium* did not. Japanese schistosomiasis, caused by *S. japonicum* and its practically spineless eggs, is limited to the Far East as an endemic disease, being prevalent in some parts of Japan, Eastern China, Formosa, the Philippines, and probably some of the islands south and southwest of the Philippines.

The transmission of all the schistosome infections depends essentially on the interplay of three factors, without any one of which schistosomiasis would cease to exist. The first factor is man, who acts as a contributory agent when the embryonated eggs are passed in urine or feces. These eggs, if they are to develop, must somehow reach fresh water in a viable condition, where the ciliated embryos (miracidia) may escape from their shells preparatory to finding a suitable snail host—the second factor. If the miracidia fail to reach this host, they soon die; but if they succeed and penetrate the snail's tentacles, they have excellent chances of undergoing further development and reaching the cercarial stage (infective for man)—the third factor.

It is essential, however, that these actively motile, fork-tailed cercariae, upon expulsion from the snail, should come within a comparatively short time in contact with the skin of the mammalian host. If they do so, and conditions are favorable, they may penetrate the epithelium often producing a dermatitis * and subsequently develop into mature adult male and female schistosome worms. The drinking of water containing the infective larvae may conceivably also afford favorable opportunities for these cercaria to penetrate the mucosa of the alimentary canal, particularly the upper portion.

Pathology and Symptomatology. The tissue changes resulting from the schistosome infections are similar fundamentally, although the clinical manifestations may vary considerably, depending upon the individual characteristics of the specific organisms

* The local immune response to repeated infections is said to be more severe. "Kabure" has been applied to this condition but it is now believed that schistosomes abnormal to man are involved here.
concerned and the locus of egg accumulation. The clinical effects of the presence of schistosomes in the body may be divided into three periods: (a) the prepatent period; (b) the period of egg deposition and extrusion; (c) the period of tissue proliferation and repair.

The prepatent period. This period comprises the time elapsing between exposure to the cercariae and the time of beginning egg-laying in the portal venules. The earliest clinical and pathologic manifestations are associated with entry of the cercariae into the skin. There is likely to be an itching or stinging sensation, depending on individual sensitivity and the intensity of exposure. Within a few hours, small petechiae may be seen at the sites of penetration of blood vessels. These usually disappear without local inflammation, although an urticarial rash may be manifest later. As migration of the cercariae and their evolution to adult male and female schistosomes occurs, toxic symptoms of variable intensity arise, such as anorexia, headache, malaise, pain in the back and the extremities, afternoon rise in temperature followed by chills and sweating at night. The blood may show leucocytosis with a relative increase in the eosinophile count. The abdomen may be distended, secondary to enlargement of the liver and possibly the spleen. With repeated infections, precordial pain and labored breathing may be noted.

The period of egg deposition and extrusion. The relative numbers of eggs produced by these three schistosomes, the presence or absence of spines, and the anatomic areas of distribution are some of the factors involved in the pathologic changes as well as in the symptomatology of schistomiasis. In S. haematobium infection, various degrees of cystitis occur with proliferative changes; in S. mansoni and S. japonicum infections the lodgment of eggs in the intestinal wall and their escape into the lumen are accompanied by analogous pathologic processes. The varying ultimate results in the type and intensity of signs and symptoms in the latter infections are probably due, in part at least, to the presence or absence of spines on the eggs. Another important factor is the number of eggs deposited by each female worm. In S. mansoni
infection, eggs with subterminal spines are produced in comparatively small numbers, whereas in *S. japonicum* the spineless eggs are produced in abundance. The reaction of the intestinal wall to such factors as these would obviously account for specific differences in the respective pathologic pictures associated with the extrusion of eggs of these schistosomes.

The first direct sign usually noted in *S. haematobium* infection is the passage of a small amount of blood at the end of micturition, accompanied at first by few or no significant symptoms. Sooner or later, however, local symptoms arise indicative of definite pathologic changes in the bladder. Cystoscopic examination reveals inflammation and hyperplasia of the bladder and urethral mucosa, the result of the deposition of the terminal spined eggs in the local perivascular tissues. Reactive cellular infiltration, followed by abscess or pseudotubercle formation and their accompanying sequelae, indicates the nature of the histopathologic picture and, to some extent, the associated clinical manifestations.

In both *S. mansoni* and *S. japonicum* infections the initial extrusion of eggs is accompanied by diarrhea and later by dysentery, which often becomes severe as the disease progresses and constitutes the "schistosome dysentery" referred to in connection with the discussion of protozoal dysenteries. The underlying early pathology and symptomatology of *S. mansoni* and *S. japonicum* are similar to those briefly indicated in infections caused by *S. haematobium* in the urinary tract. A frequent symptom is abdominal pain (often localized) associated with blood, mucus, and the characteristic subterminal spined eggs of *S. mansoni*, or the spineless eggs of *S. japonicum*, in the stool. Depending on the extent and nature of the initial infection, the pathologic changes and related symptoms continue to evolve and tend to become chronic. Other organs become involved at times, due principally to the lodgment of eggs and the pathologic processes caused by their presence. The liver and spleen tend to become larger and firmer.

The period of egg deposition and extrusion—the acute phase of the disease—usually lasts from a few weeks to several months. During this time secondary anemia and leucopenia tend to dominate
the blood picture, particularly in *S. japonicum* infections. In the bladder or the intestinal wall, as the case may be, abscesses may form around the eggs and break through into the lumen, discharging their contents. Repair by granulation and formation of scar tissue then follows. When egg deposition is not followed by abscess formation, the eggs become encapsulated by fibrous tissue and calcification eventually takes place. Fibrous thickening of the bladder and intestinal walls, and fibrous enlargement of organs involved, are probably aided by thrombosis of various local vessels—lymph channels, bile passages, and blood vessels.

**The period of tissue proliferation and repair.** The fibrous thickening of the bladder and the intestinal walls continues, and papillomata frequently develop. When the bladder is involved, the pathologic changes and symptoms are like those of chronic cystitis, and cystoscopy becomes increasingly difficult. In infections from *S. mansoni* and *S. japonicum*, intestinal involvement is accompanied by loss of tonicity and the development of fibrous constrictions which may greatly impede the passage of food and feces. Secondary pyogenic infections, leading to fistulae and malignant growths, may readily develop as complications or sequelae. A frequent late complication is obstruction of the portal circulation which, if uncompensated, results in troublesome ascites. Thrombosis of the mesenteric and portal vessels are significant complications in *S. japonicum* infections.

This stage of the disease may last for years, or the patient may succumb to an intercurrent infection. Of the three schistosomiases, that caused by *S. japonicum* is usually associated with more rapidly evolving pathology and more adverse and varied symptomatology, due probably in part to intrinsic factors inherent in the nature of the specific organism at present not well understood.

**Diagnosis.** In endemic areas, hematuria is suggestive of vesical schistosomiasis or bilharzia. Cystoscopic and sigmoidoscopic examination are valuable aids in diagnosis. However, certainty of the specific nature of the disease demands the finding of the characteristic terminal spined eggs in the urine or the feces. The in-
tradermal and complement fixation tests (group positive) de­
vised by Fairley (1927) may be helpful in the latter part of the
prepatent period and in cases in which eggs cannot be demon­
strated. Differentiation must be made from various other condi-
tions which may give rise to similar symptoms.

The diagnosis of schistosomiasis caused by S. mansoni must be
based primarily upon the finding of characteristic subterminal
spined eggs in the feces. The complement fixation test may be
useful, particularly during the prepatent period. In addition to
a high eosinophilia, increased euglobulin in the blood is said to
be significant during the period of deposition and extrusion of
the eggs. Differentiation must be made from various other dis-
eases of the intestinal tract, liver, and spleen.

The diagnostic procedure in S. japonicum infections is essen-
tially the same as that outlined for the other two human schis-
tosome infections. Additional laboratory confirmation in a sus-
ppected case of either S. mansoni or S. japonicum may be obtained
by applying the Faust-Meleney hatching technique. This proce-
dure is carried out as follows: After washing and sedimentation of
the feces, the sediment so obtained is diluted with clear water
and allowed to stand for a few hours to permit the eggs to hatch.
Since the free-swimming miracidia tend to collect at the surface
of the water, they may be seen readily with the aid of a hand

Treatment. Although symptomatic treatment of schistosomiasis
is helpful, permanent amelioration of the underlying condition
requires suitable chemotherapeutic as early as possible. The two
chemicals especially recommended are sodium antimony tar-
trate and “fouadin” (neoantimosan). These drugs should be ad-
ministered as follows:

Sodium antimony tartrate. From 60 to 100 mg. in 1 per cent
solution are given intravenously, on alternate days, until a total
of from 1.5 to 1.8 gm. has been given. It must be remembered
that this drug depresses the circulation, respiration, and tonicity
of the central nervous system. It is contraindicated in cardiac
block, pneumonia, nephritis, or advanced hepatic cirrhosis.
Fouadin (neoantimosan) is given intramuscularly in 7 per cent solution, as follows: first day, 1.5 cc.; second day, 3.5 cc.; on eight alternate days from the third day through the seventeenth day, 5 cc. each day.

The administration of either of these chemicals as directed is said to result in early symptomatic improvement. As treatment proceeds, the eggs passed in the feces become sterile and are finally absent, indicating the death of the parent worms.

Prophylaxis. Control and prevention of the schistosome infections must be based on: (a) successful curtailment of infection of the specific snail hosts; (b) destruction of infected snail hosts; or (c) killing the infective cercariae or preventing them from penetrating the skin of man. In Egypt, where irrigation is practiced on an extensive scale, periodic drying of the irrigation canals and ditches has been found to be partially effective, but this is said to be insufficient for total eradication of the infection among snails. Doubtless, the most reliable means of both individual and community prophylaxis is education in hygiene and sanitation of man himself, who is primarily responsible for the spread of this infection.
PART THREE

ARTHROPODS AND DISEASE
TRANSMISSION
CHAPTER XI

INTRODUCTION*

The close relationship which arthropods bear to disease and injury in human beings has become evident as the life cycles of the animal parasites that cause disease have been unravelled. Long before their unique roles in the biologic transmission of diseases such as filaria and malaria were known, it was suspected that insects are linked to the prevalence and spread of these and various similar diseases. Gradually the true epidemiologic significance of the relationship has been clearly and dramatically demonstrated. The economic importance of this scientific knowledge was readily recognized as it became available, and attempts were made to apply it by forward-looking governments and other progressive organizations. In some instances the results achieved have been phenomenal and epoch-making, while in others the effective application of data already available leaves much to be desired. However, a great deal of essential detailed knowledge regarding the less obvious aspects of the epidemiology, pathogenesis, and inter-relationships of many of these parasites still awaits investigation and study.

The passive role played by insects in disease transmission, although less specific and more difficult to evaluate, is nevertheless often important. The habits of the common housefly and other familiar and annoying insects are too well known to require more than passing reference. Poor hygiene and sanitation are often concomitant factors in the transfer of disease by insects, particularly when bacteria are the causative agents. This is well illustrated in fly-borne typhoid fever and dysentery, many examples

* This constitutes a brief general introduction to all subsequent chapters which comprise Parts Three and Four.

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<table>
<thead>
<tr>
<th>Disease</th>
<th>Insect</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria (Laveran, 1880)</td>
<td><em>Anopheles</em> species</td>
<td><em>Plasmodium falciparum</em></td>
</tr>
<tr>
<td>Yellow fever (Finlay, 1900-1902)</td>
<td><em>Aedes aegypti</em></td>
<td>Filterable virus</td>
</tr>
<tr>
<td>Dengue (Australian Commission, 1928)</td>
<td><em>Aedes aegypti</em></td>
<td>Filterable virus</td>
</tr>
<tr>
<td>Filariasis (Bancroft's)</td>
<td><em>Culex fatigans</em>, etc.</td>
<td><em>Filaria bancrofti</em></td>
</tr>
<tr>
<td>Filariasis (sheathless microfilaria)</td>
<td><em>Calicoides species</em></td>
<td><em>Acanthochelomonema perstans, Mansonella ozzardi</em></td>
</tr>
<tr>
<td>Cholera, typhoid, dysentery, etc.</td>
<td><em>Musca domestica</em> and other flies (mechanical)</td>
<td><em>Vibrio comma</em>, etc.</td>
</tr>
<tr>
<td>Anthrax, relapsing fever, poliomyelitis (?)</td>
<td><em>Stomoxys calcitrans</em>, etc.</td>
<td><em>Bacillus anthracis</em>, etc.</td>
</tr>
<tr>
<td>Sleeping sickness (African) (Dutton, 1901)</td>
<td><em>Glossina palpalis morsitans</em> (tsetse fly)</td>
<td><em>Trypanosoma gambiensis</em>, <em>T. rhodesiense</em></td>
</tr>
<tr>
<td>Kala-azar (Leishman, Donovan, Manson, 1903)</td>
<td><em>Phlebotomus species</em> (biting moth fly or sandfly)</td>
<td><em>Leishmania donovani</em></td>
</tr>
<tr>
<td>Oriental sore (Cunningham, 1885)</td>
<td><em>Phlebotomus species</em></td>
<td><em>Leishmania tropica</em></td>
</tr>
<tr>
<td>Forest yaws (espundia)</td>
<td><em>Phlebotomus species</em></td>
<td><em>Leishmania braziliensis</em></td>
</tr>
<tr>
<td>Onchocerciasis (blindling filaria)</td>
<td><em>Simulium species</em> (buffalo gnat or black fly)</td>
<td><em>Onchocerca volvulus</em></td>
</tr>
<tr>
<td>Filariasis (ocular)</td>
<td><em>Chrysops species</em></td>
<td><em>Loa loa</em></td>
</tr>
<tr>
<td>Plague</td>
<td><em>Xenopsylla cheopis</em></td>
<td><em>Bacillus pestis</em></td>
</tr>
<tr>
<td>Endemic typhus</td>
<td><em>Ceratophyllum fasciatus</em></td>
<td><em>Rickettsia prowazeki</em></td>
</tr>
</tbody>
</table>

*From chart arranged by Dr. John C. Torrey.
### INTRODUCTION

<table>
<thead>
<tr>
<th>Disease</th>
<th>Insect</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidemic typhus (Nicolle, 1909)</td>
<td><em>Pediculus vestimenti</em></td>
<td><em>Rickettsia prowazeki</em></td>
</tr>
<tr>
<td>Relapsing fever (European)</td>
<td><em>Pediculus vestimenti</em></td>
<td><em>Spirochaeta (Borellia) recurrentis</em> (Vel obermeieri)</td>
</tr>
<tr>
<td>Trench fever</td>
<td><em>Pediculus vestimenti</em></td>
<td><em>Rickettsia pediculi</em></td>
</tr>
<tr>
<td>Plague, typhus fever, relapsing fever, kala-azar</td>
<td><em>Cimex species</em></td>
<td>(See under individual items)</td>
</tr>
<tr>
<td>Chagas' disease (Cruz-Chagas, 1909)</td>
<td><em>Triatoma species</em></td>
<td><em>Trypanosoma cruzi</em></td>
</tr>
<tr>
<td>Texas cattle fever (T. Smith-Kilbourne, 1893)</td>
<td><em>Margaropus annulatus</em></td>
<td><em>Babesia lipemina</em> (Piroplasma)</td>
</tr>
<tr>
<td>Rocky Mountain spotted fever (Ricketts, 1906)</td>
<td><em>Dermacentor andersoni</em></td>
<td><em>Rickettsia rickettsii</em></td>
</tr>
<tr>
<td>African relapsing fever</td>
<td><em>Ornithodorus moubata</em></td>
<td><em>Spirochaeta (Borellia) dutton</em></td>
</tr>
</tbody>
</table>

of which have been reported in the literature from time to time. Milk, which furnishes an excellent culture medium for bacterial growth, may be an accessory transmitting agent.

Injury to man through the bite or sting of insects, arachnids, snakes, and various water-inhabiting animals is particularly serious when poisonous or toxic substances are introduced at the same time. Hence the value to clinicians of a general knowledge of the habits of the specific animal agent, the character of substances likely to be introduced and the ways and means of dealing effectively and promptly with their poisonous effects. The black widow spider—an arachnid—and venomous fish and snakes are examples in point. Pertinent facts regarding the various ani-
mal groups alluded to in this and the preceding introductory paragraphs are briefly presented in Parts Three and Four of this treatise. Such information, it is believed, should constitute an essential element of instruction given to undergraduate medical students.

Table XIII comprises a tabulation of the principal insect vectors of disease, the diseases which are transmitted and the specific etiologic agents. These data are arranged under the natural groups to which the various insects belong.
Control and prevention of disease transmitted by mosquitoes is dependent on a knowledge of the insects themselves. Hence a brief description of their life cycles and the salient points in their specific habits and morphology is essential as a basis for a discussion of diseases associated with their activities. Unless intelligently and specifically directed against the particular transmitting mosquito, community control efforts to protect man against the mosquito-borne diseases will accomplish comparatively little, although amelioration of conditions caused by their presence as a pest may result.

**Distinguishing Features of Mosquitoes**

Mosquitoes differ from most other dipterous insects of medical interest by the possession of scales along the veins and the posterior margins of the wings. Other outstanding characteristics are the prominent proboscis, which projects forward beneath the anterior, inferior aspect of the head (clypeus), and the antennae, which are bushy in the male and rather unadorned in the female. Insects most likely to be mistaken for mosquitoes are gnats or midges, which generally appear in swarms or clouds dancing in mid-air (mosquitoes rarely do this). Furthermore, when folded over the back, the wings of mosquitoes are as long as the abdomen or longer, whereas those of gnats are usually shorter.

Figure 64 shows the relation of a female *Anopheles quadrimaculatus* to the development of *Plasmosium falciparum*, and the mechanism whereby transmission is accomplished. The mandibles, according to Fox (1925), "lie on either side of the hypopharynx..."
Fig. 64, Top: Graphic summary of the mosquito's role in transmission of malaria. (Modified from Thompson and Robertson. Protozoology, 1928. Courtesy of Baillièrè, Tindall & Cox.)

1–8. Asexual phase (schizogony) of *Plasmodium falciparum* in man (right center).  
1–4. Forms in the peripheral blood.  
5–8. Forms in the capillaries of the internal organs.  

11–17 and 17a. Formation of gametocytes in man (left center).  
14, 17, and 17a. Development of male and female crescents (gametogony) from apparently asexual forms in the capillaries of the internal organs of man.  
18–32. Sporogony in the female anopheline mosquito.  
18 and 18a. Male and female gametocyte, respectively, which have recently entered the stomach of the mosquito.  

19. Exflagellation of the male crescent, which has assumed a spherical outline and has extruded the so-called "polar bodies."  
19a. Female gametocyte, which has rounded up and has also extruded the polar bodies.  

20. Fertilization of the female or macrogamete by the male or microgamete (syngamy).  
21. Zygote or motile ookinete (sometimes also called the traveling vermicle). The motile ookinete makes its way to the outside of the stomach wall.  
22. Oocyst. The motile ookinete rounds up and encysts on the outer surface of the stomach wall.  
23. Oocyst. The nucleus has divided and the daughter nuclei have taken up positions at the nodes formed by the vacuolation of the cytoplasm.  
24. Oocyst. Nuclear divisions continue, and from each of the many nuclei spicules of protoplasm project into vacuoles on either side. These are immature sporozoites.  
25. Oocyst. The sporozoites are now fully formed.  
26. Rupture of the oocyst, with liberation of the sporozoites into the body cavity of the mosquito.  
27. Sporozoites free in the body cavity.  
28. Sporozoites entering the muscles of the thorax.  
29. Sporozoites invading the salivary glands.  
30. Sporozoites in the salivary glands.  
31. Sporozoites being inoculated into man through proboscis.  
32. Sporozoites initiating the asexual cycle in man (according to Schaudinn).  
33. Suggesting an intrinsic extra erythrocyte developmental phase according to recent investigators.  


A, palpi; B, labrum-epipharynx (cross-section on left); C, mandibles (cross-section on left); D, hypopharynx (cross-section on left); E, maxillae (cross-section on left); F, labium showing hinged labella (cross-section of shaft on left showing relative positions of muscles and trachea); G, salivary duct in cross-section of hypopharynx (D) on left; H, muscles in labium; I, trachea in labium.
CYCLES OF THE MALARIAL PARASITE

Figure 64

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above and the maxillae on either side of the hypopharynx below." Both mandibles and maxillae are serrated. In the act of biting these two pairs of lancets move up and down rasping and sawing a small channel through the skin. Together with the needle-like hypopharynx and incomplete tubelike labrum-epipharynx, they constitute a bundle of six piercing parts. As this so-called "stiletto bundle," steadied by the two labellae (attached to the tip of the labium) enters the skin, the gutter-like labium beneath bends or bows as these piercing parts are pushed into the skin.

The hypopharynx contains the venenosalivary duct through which saliva flows, and it also serves to close the longitudinal groove in the lower surface of the labrum-epipharynx, thus forming a tube through which blood is sucked into the stomach of the mosquito. Lying a little above and on either side of the proboscis are a pair of sensory organs known as palpi, and above these on either side of the frons or forehead are two other somewhat similar sense organs, the antennae, which are plumose in the males only. Both palps and antennae are important in differentiating mosquitoes from other insects.

**LIFE CYCLE**

The transformational changes undergone by mosquitoes comprise four principal stages: (1) the egg; (2) the larva, or wiggle-tail; (3) the pupa, or tumbler; and (4) the winged adult, or imago. The eggs are usually laid on water, or close to the edge of water, or sometimes on mud. Some species may elect to lay their eggs in shallow cavities likely to be filled later with water. The "wiggle tails," which hatch from the eggs within several days under favorable conditions, are entirely aquatic and cannot live long out of water or without an abundance of oxygen and microscopic life in the water. The larval stage lasts from five to ten days or more, depending largely on temperature and availability of food; the pupal stage usually lasts from forty-eight to seventy-two hours, also varying with temperature.

The longevity of adults varies greatly, some species hibernat-
Ordinary house mosquitoes probably do not live more than from two to three weeks in midsummer. The females of many species seek a blood meal before depositing their eggs and because of this habit may serve as carriers of disease not only passively, but also as biologic transmitters of certain specific diseases, such as filaria, malaria, and some virus diseases.

**Classification**

From the standpoint of hygiene and preventive medicine, adult mosquitoes may be divided into two groups: (1) *anophelines* and (2) *non-anophelines* or *culicines*. Under natural conditions certain species of the anophelines transmit the malarias of man; other anopheline species appear not to be important as vectors of malaria. Non-anophelines are known to transmit bird malaria and certain other diseases of both man and lower animals.

There have been described throughout the world some 1400 species of mosquitoes; about eighty of these occur in North America. The most important differential characteristics of those principally concerned in disease transmission have been conveniently epitomized under tribes by Herms (1939) as follows:

**Culicini**

Adults—Palpi of female one-half as long as the proboscis; scutellum trilobed; pulvilli present. Rest with proboscis at an angle to abdomen, body appearing humped usually; wings not generally spotted or dappled; genus *Culex* brownish or grayish with banded legs or distinct thoracic markings.

Eggs—Laid in rafts.

Larvae—Well-developed siphon, usually lying quite oblique or almost vertical to water surface when breathing.

Pupae—Breathing trumpets long and tubular.

**Anophelini**

Palpi of both female and male as long as proboscis; scutellum rounded and without lobes. Most species rest with proboscis in straight line with abdomen and at a sharp angle to the resting surface. Wings usually spotted or dappled.

Laid singly.

No true siphon; lie parallel to water surface when breathing or feeding, with head rotated 180° on body axis; possess palmate float-hairs on certain anterior abdominal segments (1–7).

Breathing trumpets short and broad at margin; paddles at abdominal tip have an accessory hair.
Aedini
Adults—Palpi and trilobed scutellum as in culicini; abdomen pointed; post-spiracular bristles present; pulvilli absent or hairlike.

Eggs—Laid singly or in small groups in water or mud where water is likely to appear later.

Larvae—Siphon as in culicini but shorter and thicker, barrel-shaped in genus *Aedes*.

Pupae—Trumpets short and truncated; paddles at abdominal tip have an accessory hair.

Megarhini
Large, tropical, and usually highly colored. Basal one-half of proboscis rigid, distal one-half flexible, non-blood-sucking.

Laid singly.

Large, predaceous mouth-parts particularly adapted for capturing prey. Often found in tree holes and similar locations.

Large, trumpets short and broad.

The adult anopheline or malaria-carrying mosquitoes may be readily distinguished from the non-anophelines by the way they stand when resting (Fig. 65). The whole body (proboscis, head, thorax, and abdomen) is held in a straight line inclined at an angle of from 45 to 90 degrees to the surface. Non-anophelines, on the other hand, are definitely humped as to head and thorax, the abdomen being approximately parallel with the surface on which it is resting. In both male and female, the palpi are almost as long as the proboscis, whereas in non-anophelines the palpi, while almost as long as the proboscis in males (or even longer), are much shorter than the proboscis in females. Furthermore, the wings of anophelines are frequently marked or spotted with black, black and white, or black, white and yellow, while the wings of non-anophelines are generally unspotted.

Of the anophelines there have been described about 180 species, approximately 70 of which are considered to be potential carriers of human malaria. Information bearing on this point has been derived from epidemiological and experimental evidence as well as direct observation in nature. However, only about 20 of these species are significantly important in this connection.

Based on the character of the "hypopygium" (external genitalia) of the male anopheline, the various species are classed in three subgenera. These in turn are further divided into groups,
FIG. 65. The life histories of mosquitoes. Morphological details and life histories of three genera of mosquitoes: Culex, Anopheles, and Aedes. Eggs: above, \( \times 5 \) app.; below, \( \times 25 \) app. Larvae, \( \times 2.5 \) app. Pupae, \( \times 5 \) app. Adults, \( \times 2.5 \) app.

An. Pl., anal plate; An. Pron., anterior pronotal setae; Anl. Sdl., anal saddle (dorsal plate); Ant., antenna; A. T., antenna tuft; Cmb. Sc., comb scale; D. Br., dorsal brush; D. H. H., dorsal head hairs; Hal., halter; Hr. Tft., siphon hair tuft; I. C. H., inner clypeal hair; L. A. T., lateral abdominal tuft; Mesemp., mesepimeral setae; Mn., mesonotum (tergum); O. C. H., outer clypeal hair; Pal. Hrs., palmate or float hairs (tuft); Pec. Sc., pecten scale; Pn., postnotum; Po. Bron., posterior pronotal setae; Prea., prealar setae; Proeps., proepisternal setae; P. Spr., postspiracular setae; Resp. Trump., respiratory trumpet; Scl., scutellum; Sp., spiracle; St. Pl., sternopleural setae; V. Br., ventral brush.

(After Herms, adapted from various authors. Herms: Medical Entomology. Edition 3. Courtesy of the Macmillan Company.)
depending also on hypopygial features. The subgenera, with the exception of species of two small genera (*Chagasia* and *Bironella*), now accepted are as follows:

1. **Subgenus *Anopheles*** which includes the majority of American species (together with a few species of the Eastern Hemisphere) with the exception of the following subgenus (*Nysso rhynchus*) and *A. gambiae* of the subgenus *Myzomyia*, a recent importation from West Africa to the region of Natal, Brazil.

*A. maculipennis*, a medium-sized mosquito, is probably the most widely distributed species of the subgenus *Anopheles*. It occurs in the northwestern United States, western Canada, and Alaska; Europe, North Africa, Asia Minor, and the Orient. It bites both day and night but its role as a vector of malaria varies greatly with different races or varieties, which often may be identified by the eggs. Relatively, its importance in transmitting malaria depends largely on its feeding preferences. If the blood of domestic animals is the food of choice of *A. maculipennis* (zoophilism), its importance as a carrier is not great unless there is a scarcity of such animals; on the other hand, if human blood is preferred (androphilism), its potential role as a carrier of malaria must be seriously reckoned with.

*A. quadrimaculatus*, a medium-sized black mosquito with significantly spotted wings, is found in southern Canada, much of the United States east of the Rocky Mountains, and in Mexico. Wherever this anopheline is found, it constitutes an important potential vector of malaria. It breeds in quiet pools and puddles in well-shaded places and bites chiefly at dusk or night but may also do so in darkened rooms during the daytime.

*A. hyrcanus* ranges from southern Europe eastward to China and Japan. It is considered an important malaria carrier in southeastern Asia and several races have been found to serve as vectors of the filarias *Wuchereria bancrofti* and *malayi*.

*A. pseudopunctipennis*, a medium-sized black mosquito with white spotted wings, is found in the southwestern United States,
Mexico, Central America, the West Indies, west coast of South America, and northern Argentina where it is said to be the most important vector of malaria. In other parts of its range, it is not considered an important vector of malaria except in southern Mexico. It breeds in pools, springs, and streams.

A. crucians and A. punctipennis, both medium-sized dark-colored anophelines (the former with mottled wings), are considered relatively less important as vectors of malaria in the United States.

A. crucians is found in the south Atlantic and Gulf states of the United States and gulf coastal areas of Mexico and Central America. There are two principal varieties, one breeding in fresh and the other in brackish water.

A. punctipennis is widely distributed in Canada, the United States, and Mexico. It breeds in pools, ponds, and small streams and prefers to feed upon the blood of large animals.

The principal members of an essentially American tropical group of the subgenus Anopheles which breed principally in pools are the following:

A. punctimaculata, quite common in Panama and other Central American countries, Colombia, Venezuela, and Trinidad. It is thought to be a common carrier of malaria in the Panama Canal Zone.

A. opimacula in Venezuela, A. intermedius and A. pseudomaculipes in Brazil are three other tropical species belonging to this group of the subgenus Anopheles. They breed mainly in jungle pools.

2. Subgenus Nyssorhynchus, in which are classed a few species which constitute the most abundant and important malaria carriers of tropical America.

The Nyssorhynchus group which comprises the principal species of this subgenus consists of "white-hind-footed" anophelines which are found in Central America, northern South America, and the West Indies. The principal members of this group are:
A. *albimanus*, a medium-sized black mosquito with yellow wing spots. It breeds in marshes, swamps, and streams and is an important vector of malaria in humid areas.

A. *argyritarsus*, a medium-sized mosquito with white spots on wing-costa which is recognized as a malaria carrier.

A. *darlingi*, common in British Guiana and Brazil, which is a domestic species breeding in swamps, small pools, and ditches on sugar and rice plantations. It is androphilous and an important malaria carrier.

A. *albitarsus* and A. *tarsimaculatus* found abundantly in the same regions as A. *darlingi*. They are potential vectors of malaria but fortunately are zoophilous.

3. Subgenus *Myzomyia* includes species restricted to various parts of the tropics and subtropics of the Eastern Hemisphere with the recent exception of *A. gambiae* previously mentioned.

Geographically, this group is highly specialized. It contains some of the most dangerous malaria vectors in the world as instanced by the recent history of *A. gambiae* in Brazil. The introduction of this species to the Western Hemisphere may have been brought about through the agency of fast Navy destroyers (four days from Dakar to Natal) or aircraft which cover the same distance in six to eight hours.

Typical species of this subgenus may be briefly grouped according to continent as follows:

**Europe**

*A. hispaniola* and *A. superpictus* which are also found in northern Africa (the latter also in Asia). *A. superpictus* appears to be zoophilous by preference and ordinarily is thought to play a minor role in transmission of human malaria.

**Africa**

*A. funestus* which breeds in clear sluggish streams and is an important vector of malaria in tropical Africa.

*A. gambiae* in tropical Africa and southern Arabia which breeds in stagnant pools and sluggish streams and frequents human habi-
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It is also an important vector of malaria in Africa as well as in parts of Brazil closest to Africa.

*A. pharoensis* is considered a good vector of malaria in Egypt and possibly other parts of the Orient and Eastern Mediterranean region.

**ASIA AND EAST INDIES**

*A. culifacies* which is found in Arabia, India, and Siam and is considered an important malaria vector in India.

*A. maculatus* which is found in India, southeastern Asia, and the East Indies. It breeds in clear streams and is a recognized malaria carrier in Malaya and the Dutch East Indies.

*A. ludowi* and *A. stephensi* outstanding vectors of malaria in southern Asia and Malaya. *A. stephensi* is found in Arabia, Mesopotamia, India, and French Indo-China. It is urban in habitat, breeding in cisterns, wells, and small pools.

**OCEANIA AND AUSTRALIA**

*A. annulipes* and *A. punctulatus*, the former breeding in small swamps and stagnant pools in Australia and the New Hebrides and suspected of being a malaria carrier.

Among the culicines (non-anophelines) there are two practically distinct groups belonging principally to the genera *Culex* and *Aedes*, respectively.

Species of *Culex* usually have beaks and legs of a somewhat uniform blackish brown color and generally are not ringed or spotted. The thorax is reddish or grayish brown, the abdomen, dark brown with narrow bands of yellowish scales at bases of segments.

*Aedes* mosquitoes, on the other hand, are characterized primarily by banding of the legs and other parts of the body, depending on the species. *Aedes aegypti* (the yellow fever mosquito), formerly known as *Stegomyia fasciata*, is most striking in appearance. The ground color is black, marked with brilliant silvery white. The proboscis is plain, the palpi are white tipped, and the thorax is marked medio-dorsally by two anteroposterior
parallel white lines with two other curved white lines on either side. These markings as a whole suggest a lyre in design. The abdomen is ringed with white bands and further ornamented with rows of triangular white spots on either side. Finally, the tarsi of the legs are banded silvery white at the bases of the articulations, and the last joint of the hind legs is entirely white.

**Deposition and Characteristics of Eggs**

Most female mosquitoes, after partaking of a blood meal, deposit their eggs upon water, generally at night, although certain species of the genus *Aedes* often deposit their eggs on mud or a dry place where water is likely to accumulate later. Species with a single brood a year (the spring species in particular) lay the eggs above the water line so that spring rains will carry them into temporary pools. Then, if excessive rains in summer or fall cause flooding, the eggs hatch. This suggests a "hibernating" adaptation. Individual eggs are longish ovals, roughly cigar-shaped and less than 1 mm. long. Those of three common species representing the genera *Culex*, *Anopheles*, and *Aedes* are shown, as laid, in Figure 65, those in the first row being magnified considerably, while those in the second row are greatly magnified. It should be noted that the eggs of *Culex* are more numerous (about 150 to 250) than the eggs of the other two species and are cemented together in the form of a hollow boatlike mass known as the "egg-raft"; the eggs of *Anopheles* and *Aedes* are laid singly, but adhere more or less to one another. Only anopheline eggs have laterally placed air bladders or "floats" which serve to buoy them up. Individual eggs of both *Culex* and *Aedes* will sink when support by surface tension is disturbed in any way.

**Distinguishing Features of Larvae**

The larvae or wiggle-tails, which generally hatch under favorable conditions in from one to three days, also have certain individual characteristics (Fig. 65). Unlike the larvae of *Culex* and *Aedes*, which have a distinct breathing tube or "siphon" on the
eighth abdominal segment, *Anopheline* larvae have merely a pore or tracheal orifice through which to breathe. The parallel alignment of the larvae with the surface of the water which this feature makes essential is facilitated by "palmate hairs" on certain abdominal segments. With this mechanism of support and their ability, while feeding, to rotate the head through 180 degrees against the surface film which is rich in plankton flora and fauna, the anopheline larvae are well adapted to surface feeding.

When larvae emerge from the egg shells, they are small, but they feed voraciously and grow very rapidly. They moult about three times during the period of larval growth, which lasts from one to two weeks under average conditions. They have a characteristic head, thorax, and segmented abdomen. As already intimated, in order to obtain atmospheric oxygen directly, they assume characteristic positions with reference to the surface film of the water, as illustrated (with certain specific identifying details) in Figure 65. The so-called "anal gills" also aid the young larvae in utilizing oxygen dissolved in the water. The body length of full grown larvae is often greater than that of the adult mosquito.

**Characteristics of Pupae**

Preparatory to pupal development, the mature larva doubles up somewhat, ceases to feed, and shortly escapes from its larval skin as a comma-shaped pupa, or "tumbler." It does not feed further and is inactive most of the time, although it darts about excitedly at times and frequently remains at the surface long enough to take in air through its respiratory "trumpets," which project upward from the dorsum of the thorax. The latter is relatively larger than the more slender abdomen which is made up of seven segments, the terminal one of which is equipped with two fins or "paddles." (Fig. 65.)

After from one to five days, the mature pupa with its tail extended lies quietly in alignment with the water's surface, the dorsum of the thorax being uppermost. The pupal skin soon splits longitudinally and slightly transversely over the thorax; through
this opening the thorax of the adult shortly emerges, followed by the head and then the abdomen. Employing this last pupal skin as a means of support, the adult mosquito or “imago” first expands its wings * and then frees and extends its legs, the abdomen finally being released. After perhaps five to ten minutes under favorable circumstances, the adult mosquito is nearly ready for flight. As a general rule, and perhaps always, the adult is not fully dried at this time. When it flies away, it is rather limp and a few hours are usually necessary in order to complete drying.

MOSQUITO-BORNE VIRUS DISEASES **

Dengue

Dengue (breakbone or “dandy” fever) is a widespread disease of warm climates. Particular areas of endemicity are the West Indies, the Near East, southeastern Asia and Malaysia. The disease is likely to occur in epidemics. In the United States its local occurrence is associated with the prevalence of *A. aegypti*, particularly in the south Atlantic and Gulf states. Because of its clinical and epidemiologic similarity to yellow fever, dengue was formerly suspected of being a closely related disease. However, the results of cross-immunity experiments seem not to lend support to the likelihood of any direct relationship.

The immunity following an attack of dengue is variable and largely individual, while that following yellow fever is absolute and fairly permanent. As in yellow fever, *A. aegypti* is the usual transmitting mosquito of dengue, although species of certain other genera have also been under suspicion. Also, as in yellow fever, epidemics of dengue usually appear after comparative local freedom from the disease for a considerable period; the explosive outbreak usually follows a less dramatic previous one. When *A. aegypti* breeding is at its maximum, the major outbreak suddenly appears.

* The wings arise from pads which expand in response to pumping of air and blood through the mosquito's body.

** The nonvirus mosquito-borne diseases, the malarias and *W. bancrofti* infection, are discussed in Chapters VI and X, respectively.
Pathology and Symptomatology. Postmortem records of dengue are very few, as the mortality is low. Localized inflammation in the lungs and cranium and of the crucial ligaments of the knee have been noted, as well as serous effusions in the neighborhood of the involved joints.

The incubation period of dengue is usually from four to seven days, and the onset is very sudden. It is generally preceded by malaise, with perhaps twinges in one or more fingers, toes, joints, or limbs. When the temperature begins to rise, these initial symptoms are likely to be intensified, and headache and other related subjective symptoms appear. Leucopenia and slow pulse are characteristic features. About the third or fourth day, the temperature usually drops to nearly normal and tends to remain there for from twelve hours to three days at the end of which time the patient feels much better.

Following this variable intermission, the temperature rises again, attended with pain and possibly greater depression than before. This period of temperature is, however, usually of shorter duration and is accompanied by a terminal, measles-like rash, which generally begins on the hands and feet. The rash is not so dusky red in appearance as that of measles, and it may be somewhat punctiform like the rash of scarlet fever. Profuse sweating and diarrhea may be associated with the rash. Desquamation is furfuraceous in character and is frequently accompanied by marked itching. Convalescence is likely to be slow and is frequently associated with malaise and nervous depression.

Diagnosis. The diagnosis of dengue is purely clinical. Particularly suggestive are the "saddle-back" type of fever, terminal rash, severe pains in the back, joints, and postorbital muscles, suggesting the name "breakbone fever." As expressed by an entomologic acquaintance, who suffered from an attack, "during the violent chill, the bones seem to be parted and then banged together, causing most excruciating pain followed by a feeling of great weakness."

Leucopenia during the first period of fever is a particularly suggestive symptom. Differentiation must be made from the vari-
ous other fevers associated with rashes. Confusion with articular rheumatism may arise if the pains about the knees and ankles are mistaken for joint involvement.

Treatment and Prophylaxis. The treatment for dengue is supportive and symptomatic. Control and prevention depend primarily on destruction of the transmitting mosquitoes and their larvae, and in protection of both sick and well persons from the bites of adult mosquitoes. Since the virus is apparently in the peripheral blood only during the first three or four days of the fever, it is most important to thoroughly shield the patient from mosquitoes at this time.

Yellow Fever (Yellow Jack)

Historical. Prior to 1900 there was much speculation as to the nature of the specific etiologic agent of yellow fever and the manner or means of its transmission. In a report of the American Army Commission (1911), published two years later, the following statements appear: (a) *Bacillus icteriodes* of Sanarelli has no etiologic relationship to yellow fever. (b) Fomites are a negligible factor in the transmission. (c) *Aedes aegypti,* when fed on the blood of a yellow fever patient in the first three days of the disease, is capable of transmitting the infection, after a period of twelve days, to a susceptible person; and that a mosquito thus infected remains so for the remainder of its life. (d) The blood of a yellow fever patient in the first three days of the disease, which is sterile for *B. icteriodes,* is infective for yellow fever when injected subcutaneously into a susceptible person. In this connection they found also that heating of infectious blood to 55° C. for ten minutes, destroys the virus, and when passed through a Berkefeld filter, the virus remains infectious thus showing that it is filterable.

Two other notable contributions to our knowledge of the epi-

*As early as 1881, Finlay (1912) of Havana believed that yellow fever was transmitted by *Culex* (Stegomyia) fasciatus (now *A. aegypti*). Carter (1900) concluded in 1898 that a period of about two weeks generally elapsed between the appearance of the first case and of secondary cases resulting from this one.

**In 1919 Noguchi (1925) reported the cause of yellow fever to be *Leptospira icterioides* but the organism which he isolated was probably *L. icterohemorrhagiae* of infectious jaundice (Weil's disease).
demiology and immunology of yellow fever are the demonstration by Stokes, Bauer, and Hudson, in 1928, that the rhesus monkey is susceptible to the disease, and the discovery by Max Theiler (1930) that yellow fever may be transmitted to mice by intracerebral inoculation, producing in these animals meningo-encephalitis. After a sufficient number of passages in mice, the virus becomes fixed, and when reinoculated into the brain of rhesus monkeys causes a fatal encephalitis. The form of yellow fever virus fixed for mice is now known as the "neurotropic strain" in contrast to the ordinary or "viscerotropic strain." These advances have opened the way for active immunization, and for testing for the presence of immune bodies in persons who have previously had yellow fever (the so-called mouse-protection test).

**Geographical Distribution.** The history of colonization of the Western Hemisphere is paralleled by records of repeated epidemics of yellow fever. The earliest clear descriptions of this disease were made in connection with outbreaks in the Americas following their discovery and early exploration. However, both historical and epidemiologic evidence indicate that yellow fever was probably originally carried from Africa to the New World through slave trading.

Our knowledge of the geographical distribution of yellow fever has been greatly extended by the recent work of the International Health Division of the Rockefeller Foundation in Africa and various countries of South America. Extensive surveys conducted by members of their scientific staff are still in progress. In Africa the disease is known to be endemic in a wide belt of territory extending from about 15 degrees north to 5 degrees south latitude, and from the west coast to the Bahr-el-Ghazel province of the Anglo-Egyptian Sudan. Likewise, the disease is endemic in wide areas in Brazil, and recently limited epidemics have occurred in Colombia, Bolivia, and other South American countries.

In the past, yellow fever has been imported on ships into various eastern North American cities, southern European countries, and even into England, but these limited epidemics have been
brief in duration. With the present greatly accelerated means of transport, there is danger that yellow fever may be introduced by automobile or aeroplane into India or even China, since local


strains of *A. aegypti* are known to be as susceptible to yellow fever virus infection as are the African Aedes.

In Figure 66 is shown the distribution of yellow fever as known at the present time. Contrasted with the much wider distribution of *A. aegypti*, depicted in Figure 67, the possibility of spread of epidemic yellow fever to new areas becomes clearly apparent. South Africa, India, southeastern Asia, and adjacent islands, appear particularly, to be regions open to the spread of this disease.

**Epidemiology.** Yellow fever, occurs mostly in low-lying, hot, squalid, insanitary districts, particularly in the vicinity of docks and wharves of the larger seaport towns and cities where *A. aegypti* abounds. However, it has been shown recently that this disease may also be endemic in inland districts where *A. aegypti* is entirely absent, for instance in northern Nigeria and in South America, notably in Brazil. In districts in which *Aedes* is absent, persons contracting yellow fever are laborers in or near the jungle; women and children are usually spared when they are not living in or
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adjacent to such areas. Hence the name "jungle yellow fever," which is applied to this type of the disease.

Soper (1938) showed that certain species of monkeys, which are plentiful in such localities as those just mentioned, are sus-

Fig. 67. Geographical distribution of Aedes aegypti. (After Patton and Evans. Insects, Ticks, Mites, and Venomous Animals of Medical and Veterinary Importance. Grubh, Croyden, 1929.)

ceptible to infection and that a considerable proportion of the animals examined had immune bodies in their blood. It is thought also that a highly susceptible hedgehog may possibly play a similar reservoir role in Africa. Certain other animal species, which are supposedly not susceptible to yellow fever under natural conditions, if subjected to the infection experimentally, may actually harbor the virus in their blood for some days until antibodies develop. Even the possibility that arthropods may serve as permanent reservoirs for this virus has been suggested.

The most important epidemiologic factors concerning the yellow fever virus are the following:

(1) The virus may be transported from place to place.

(2) Development of the virus in epidemic form usually re-
quires a mean atmospheric temperature of over 75° F. (24° to 25° C.) and it is said that the endemic centers have never extended beyond 40 degrees north latitude and 40 degrees south latitude, where the isotherm is not below 20° C. The disease ceases to spread when the thermometer drops below this point, and it stops abruptly at the freezing point.

(3) Dampness favors the development of the virus, particularly when *A. aegypti* is the vector.

(4) Rarely is there evidence of the existence of the virus far inland or on very high ground. Extension inland is usually along lines of communication, such as navigable rivers, canals, and railroads.

(5) Immunity may be acquired only by a previous attack of yellow fever or by artificial immunization. Natives who escape yellow fever at the time of an epidemic usually do so by reason of an acquired immunity from a previous attack which frequently occurs in childhood. Hence, the native epigram, "White man, big fever; dark man, little fever."

(6) The incubation period of the virus in man during epidemics rarely exceeds four or five days, with an upper limit of thirteen days.

(7) A period of at least twelve days elapses in a community which is free of yellow fever before secondary cases arise from a newly introduced, active case of the disease. This is known as the "extrinsic" incubation period of the virus, in contradistinction to that of four or five days in man, the "intrinsic" incubation period.

In summing, it may be briefly stated that in order for yellow fever to continue in a community, three essential factors are required, namely: (1) the virus; (2) the vector, usually *A. aegypti*; (3) susceptible human beings, living together under circumstances and conditions in which infected insect vectors and man are freely accessible to one another.

Elimination of any one of these three factors would soon result in the disappearance of yellow fever. The insect vector must be present in sufficient numbers for yellow fever to continue permanently or be endemic. Under these circumstances, it is thought
that native children have yellow fever in a very mild, generally unrecognized form, and that the disease is only recognized as such when it attacks nonimmune immigrants.

Pathology. The virus of yellow fever has a particularly destructive action on liver cells, the histopathology of which is characteristic. The most marked changes are to be found in the midzonal region of the liver lobules and to a lesser degree near the periphery. The liver cells are often separated from one another, tending to assume roundness in shape, and fatty degeneration is quite marked. The cytoplasm frequently undergoes hyaline degeneration and the nuclei show peripheral distribution of chromatin and acidophilic intranuclear inclusions similar to those found in other virus diseases.

Hyperplasia of the endothelial cells of the intralobular sinuses occurs, accompanied by infiltration of large mononuclear cells and a few polymorphonuclear leucocytes. In the experimentally infected rhesus monkey, the infiltrating cells are mostly polymorphonuclear, while the great majority of parenchymatous cells contain acidophilic intranuclear inclusions, but inclusions are rare in the cytoplasm, and fatty degeneration is less marked than in man.

An outstanding characteristic of yellow fever is the yellowish staining imparted to the skin, sclera, meninges, and cartilages. Petechiae are common in mucous membranes and skin, while larger extravasations of blood may be found in the muscles. There is a generalized fatty degeneration of the capillaries and smaller blood vessels. The stomach and intestines usually contain recognizable fluid blood or its decomposition products. If death has occurred during the later stages of the infection, the liver is likely to be friable and may present a yellow color, likened to that of boxwood, indicative of profound degenerative changes.

In the spleen there may be some hyperplasia of the sinus endothelium and atrophic changes in the malpighian corpuscles. More definite signs of toxic damage, however, are found in the kidneys. Focal hemorrhages under the capsule and in the cortex are common. The renal epithelium shows cloudy swelling, merging
into fatty degeneration which affects the tubules more than the
glomeruli. The tubules contain irregularly scattered casts of al­
buminoid material or desquamated epithelium, much like ordi­
nary albuminous casts.

Symptomatology. The initial or prodromal symptoms of yellow
fever are similar to those occurring in other specific fevers, such
as malaise, chilliness, and headache. Uncomplicated cases of
yellow fever pass through three phases: (a) the initial fever, (b)
the period of calm or stasis, (c) the period of reaction.

The initial fever. Usually this is sudden in onset and lasts three
or four days. The temperature generally reaches its maximum
within the first twenty-four hours. In cases of medium severity,
it may rise to 103° or 104° F. During the three or four succeeding
days, the temperature slowly subsides to 99° F., or below, the
daily fluctuations seldom being more than one-half to one degree.
Frequently associated symptoms are chill, severe headache
(frontal and orbital), pain in the back, loins, calves, knees, ankles,
and epigastrium.

The face is flushed and swollen, the skin is dry, and the pa­
tient is most miserable and restless. At first the pulse rate ranges
from 100 to 120 and is full and strong, but as the disease progresses,
it tends to lose these characteristics, gradually falling in force
and frequency until the heart often beats only 30 or 40 times a
minute. By the third day the sclerae usually show a yellowish
tinge and frequently the skin also is yellow, hence the name "yel­
low fever." Although various kinds of eruption may occur in
individual cases, there is not a characteristic eruption in this
disease.

Albumin in the urine, with a tendency to suppression of the
urine, constitutes a most important diagnostic, as well as prog­
nostic, feature of yellow fever. The albumin usually begins to
appear about the second day of the fever and tends to decrease
as the fever falls. The urine is almost invariably acid and shows
granular casts with evidence of hemoglobin spectroscopically. The
appearance of bile pigment and bile-stained casts toward the end
of the acute attack is a favorable sign.
Nausea and vomiting are more common in yellow fever than in most other fevers. The so-called "black vomit" is a striking feature and when it occurs constitutes a grave sign. In the earlier stages, vomiting of bilious matter is a common occurrence. This may subside or give place to "coffee ground" vomit, which in turn may be followed by the typical black vomit. The majority of deaths during the febrile phase occur on the fifth or the sixth day. Death is frequently preceded by a rapid rise and then sudden fall in temperature.

The period of calm. In mild cases this period usually sets in after subsidence of the initial fever and may last several days before convalescence begins. Recovery, once begun, is usually rapid, and the patient is able to be up and about in a week from the beginning of the initial fever. In severe cases, however, the period of calm is followed by a period of reaction (the third stage) associated with a secondary rise in temperature.

The period of reaction is ushered in by a return of the fever, which does not rise so high as in the initial fever, but assumes an adynamic, remittent character, which may continue for several days or weeks. Complications such as abscess, parotitis or hepatitis tend to prolong this secondary fever. The jaundice is now very marked and black vomit may recur or appear for the first time; or the urine may be suppressed, and stupor, coma, and other nervous symptoms follow, very often terminating in death. In some instances the secondary fever ends in a crisis accompanied by sweating, followed by a prolonged convalescence.

Diagnosis. There is no clinical feature of mild yellow fever which will serve to distinguish it from many other fevers, including infectious jaundice (Weil's disease) and remittent malarial fever. Dengue is particularly difficult to differentiate from mild yellow fever and the same is true of Rift Valley fever and other similar rarer conditions. The appearance of the characteristic eruption on the fourth day in dengue is highly significant differentially.

Severe yellow fever may be easily confused with infectious jaundice, bilious remittent fever, and blackwater fever. Early in
an epidemic of yellow fever, a differential diagnosis may be most
difficult to make. Several deaths in quick succession within a
limited area, preceded by fever and black vomit, make the diag­
nosis reasonably certain. When a diagnosis is based on clinical
grounds alone, the probabilities must be most carefully and criti­
cally weighed in the light of the best clinical judgment ob­
tainable.

Four laboratory procedures are now available for specific di­
agnosis:

(1) During the first three days of the initial fever, the patient's
blood may be inoculated intracerebrally into rhesus monkeys; if
the specific virus is present, these animals die after an interval
of at least four or five days, showing suggestive symptoms and
characteristic lesions at autopsy. If the blood of such animals is
inoculated directly into the brains of mice, encephalitis should
develop in from seven to fifteen days if the disease is yellow fever.

(2) To confirm or establish the diagnosis in recovered cases,
the monkey-protection test or, preferably, the mouse-protection
test may be advantageously employed.

(3) Fatal cases of yellow fever may be diagnosed post mor­
tem from a histologic examination of the liver. The essential
features are mid-zonal necrosis, infiltration with mononuclear
cells, and the presence of inclusions in the cytoplasm and nuclei
of liver cells. The use of the "visceratome" to obtain specimens
of liver tissue obviates resort to a general autopsy.

(4) Application of the complement fixation principle, employ­
ing as antigen the plasma, serum, or liver of monkeys which have
been previously infected with yellow fever virus. This test has
not thus far given constant results with human sera.

Treatment. Although, formerly, drugs designed to destroy the
etiologic agent were employed in the treatment of yellow fever,
it is now recognized that proper nursing care is of more value.
Once in bed, the patient should not be allowed to get up. Im­
une serum is of little use except possibly during the incuba­
tion period. By the fourth or fifth day of the initial fever, a suf­
ficient quantity of immune bodies to neutralize the virus is
usually produced. If death occurs, it is probably caused by the effect of metabolic destructive toxins on liver, heart, and kidneys.

An efficient purgative given at the onset of the disease (castor oil, 2 ounces, or even more) is most beneficial. Free ingestion of water tends greatly to obviate the failure of renal function, which is the usual immediate cause of death. The various symptoms arising during the course of this disease should be treated in accordance with accepted clinical practice. Proper stimulation when indicated and careful feeding are important. To combat the acidosis, alkaline water should be given. The Sternberg sodium bicarbonate mercury perchloride (bichloride) treatment* to counteract hyperacidity of the stomach and intestines was formerly quite popular.

**Prophylaxis.** Control, as well as prevention, of yellow fever should be based on our present considerable knowledge of its epidemiology. When an epidemic is threatened, it is most important to recognize the earliest cases and to prevent the infection of *A. aegypti* mosquitoes. This can be accomplished by screening the patient, destroying all adult mosquitoes within the premises and effectively screening the premises from subsequent entry of these insects, and instituting a general campaign designed to prevent further breeding of *Aedes*, centering in and about the premises in which cases of yellow fever are housed or hospitalized.

As a general preventive measure, extermination of all mosquitoes on an extended scale is desirable, since it has been found that rapid reduction of mosquitoes generally results in corresponding diminution of all mosquito-borne diseases. However, since the preferred breeding places of *A. aegypti* are stagnant water in the vicinity of man’s habitation, it is extremely necessary first to eliminate, in so far as possible, all conceivable ways and means conducive to the hatching of *Aedes* eggs and the development of the larvae and pupae.

Those in authority in both endemic and epidemic areas should exert every effort to have the following measures carried out:

* The prescription consisted of 150 grains of sodium bicarbonate and 1/4 grain of mercury bichloride dissolved in a quart of water; of this 1 1/2 oz. may be given every hour.
(1) Mosquito nets should be used generally unless houses are effectively screened.

(2) Nonimmune persons should be prevented from coming in contact with patients with yellow fever and from frequenting areas considered specially dangerous.

(3) All cisterns, water tanks, and containers of every description holding water, even though only temporarily, should be effectively screened or drained.

(4) Reports of all cases, even suspicious cases, should be obtained for immediate investigation by a fully qualified health officer.

(5) Clearance of ships from infected ports or their entry into noninfected ports without adequate inspection and quarantine should be prohibited.

(6) All travel to and from endemic as well as epidemic yellow fever areas should be properly regulated. This should include particularly the rapidly expanding and extending means of transportation by bus, truck, and aeroplane.

An effective means of prophylactic vaccination was worked out in 1931 by Sawyer, Kitchen, and Lloyd (1932), and quite successfully employed in a limited way for several years. The vaccination consisted in the subcutaneous inoculation of enough immune human serum to give a passive immunity, followed by a single subcutaneous injection of the attenuated neurotropic living yellow fever virus. The success of this method depends on the fact that once the yellow fever virus has become fixed for the mouse brain by many repeated intracerebral inoculations, it fails to produce viscerotropic lesions in normal mice when inoculated subcutaneously. With this approximately fixed virus, the majority of mice die from encephalitis on the third or fourth day after intracerebral inoculation.

Subsequent studies by means of chick embryo culture of a strain of yellow fever virus of greatly diminished viscerotropic and neurotropic virulence has led to a greatly modified strain known as “17D.” This is considered safe for subcutaneous injection without accompanying serum and is the strain which has
been extensively employed in yellow fever prophylaxis. In preparation of the vaccine, nine-day-old developing chick embryos are inoculated with this attenuated living yellow fever virus. After four days' incubation, the embryos are removed aseptically from the eggs and finely ground mechanically, centrifuged to remove coarse particles and mixed with heat inactivated normal human serum in the proportion of one to four. The material is then placed in ampoules, rapidly desiccated, frozen, sealed, and stored at a temperature not higher than 4°C. The individual dose of the vaccine is 0.5 cc. of a 1:10 dilution in normal saline given subcutaneously.

**Rift Valley Fever**

This is primarily an epidemic disease of sheep and cattle, first studied in Kenya Colony, East Africa. It is caused by a filterable virus which is transmissible to man under certain circumstances. Experimental investigation of the virus thus far seems to indicate that certain mosquitoes of the genus *Mansonia* (culicines) may be responsible for transfer of the virus in nature. The incubation period in man is said to vary from four and one-half to six days.

**Characteristics of the Virus.** The active virus may remain in the blood for about ten days. It is found also in the liver, spleen, and other internal organs, and may pass through the placenta of pregnant animals. A fixed neurotropic strain has been produced by injecting the specific immune serum into a susceptible animal prior to an intracerebral inoculation of the virus. There is no cross immunity with the viruses of dengue or yellow fever. Immunity of immunized animals to reinfection has been found to last for six months or more, and apparently for as long as three years in one instance in man.

**Pathology and Symptomatology.** The characteristic pathologic lesion of Rift Valley fever in inoculated mice is a marked focal necrosis of the liver, essentially similar to that produced by yellow fever. The clinical picture of active disease, as well as prodromata, resemble those of influenza, dengue, and yellow fever.
Diagnosis, Treatment, and Prophylaxis. The virus of Rift Valley fever may be distinguished from the virus of dengue and yellow fever by its direct pathogenicity for mice and rats when inoculated intraperitoneally. Both monkeys and man (immune to yellow fever) are susceptible to this virus. There is no relationship between this disease and psittacosis, a virus disease of birds which is directly communicable to man.

Treatment and prophylaxis, at present based on inadequate knowledge, is empirical, and rational methods await more complete laboratory and therapeutic studies.

Other Probably Related Virus Diseases

According to reports from Africa, there are probably several more virus diseases of lower animals which occasionally may be transmitted to man by intermediate insect hosts, presumably mosquitoes.

Recent work on the etiology and epidemiology of equine encephalomyelitis, a virus disease, indicates also that this disease likewise may be transmitted by mosquitoes. The western type has been transmitted by *A. aegypti* experimentally. Finally, it appears that the virus (neutropic) of herpes encephalomyelitis, a somewhat similar condition, also may be transmitted by *A. aegypti*. 
CHAPTER XIII

FLIES AND DISEASE TRANSMISSION

Among the lower diptera—to which the mosquitoes belong—there are several genera of small size which are of considerable medical importance. These are diverse in form, some resembling small mosquitoes, others being more like flies such as the related horse-flies. These various lower diptera and the disease conditions with which they are associated will be described briefly before discussing the higher diptera, which are exemplified by the houseflies and stableflies.

THE LOWER DIPTERA EXCLUSIVE OF MOSQUITOES

Genus Culicoides (Biting Midge or "Punkies")

There are many species of the Culicoides genus (Fig. 68, 5, 6, 7) which occur in most parts of the world. Rarely, however, do more than half a dozen species occur in one locality. These flies are also known as "no-see-ums" and sometimes as sandflies. The adults are small, measuring from 1 to 3 mm. in length. The females are vicious biters, and often become veritable pests to man and to beasts. In their biting habits, they resemble the black flies (Simulidae) and are sometimes erroneously mistaken for them. The larvae are aquatic or semiaquatic, being found in small accumulations of water, particularly in water containing vegetation, and in tree holes and decaying vegetation. They are frequently called "blood worms" because of their color.

One species has been found to serve as intermediate host for the filarial roundworm Acanthocheilonema (Filaria) perstans of equatorial Africa and in limited secondary foci in British Guiana and New Guinea. Another species has been reported to be a trans-
mitter of a related filarial worm. *Mansonella (Filaria) ozzardi*, which occurs in northern South America and some of the West Indies. (See Chapter X for brief descriptions of these and related parasitic worms.)

Fig. 68. Mosquito-like insects. 1, *Phlebotomus papatasii*; 2, *P. papatasii* (natural size); 3, *P. papatasii* larva; 4, *P. papatasii* larva (natural size); 5, *Culicoides* sp.; 6, *Culicoides* sp. (natural size); 7, *Culicoides* sp. larva, X 15 app.; 8, *Simulium* sp. in attitude often observed, X 4.5 app.; 9, *Simulium reptans*, X 4.5 app.; 10, *Simulium* larvae, X 3 app. (From Stitt, Clough and Clough. Practical Bacteriology. Haematology and Parasitology. Edition 9. Copyright the Blakiston Company, Publishers.)

**Genus Phlebotomus (Sandflies)**

There are a number of species of *Phlebotomus*, all of small size, which are of importance medically. The adults measure from 3 to 5 mm. in length. They are tawny in color, hunchbacked, and have conspicuous black eyes, hairy body, wings, and legs. The mouth-parts of the female resemble those of the female mosquito, but are considerably shorter. The bite produces a local inflammatory indurated lesion with wheal formation from 1 to 2 cm. in diameter. Species of this genus have been shown to transmit three diseases, as follows:
PHLEBOTOMUS, SANDFLY, OR PAPPATACI FEVER

Phlebotomus fever is a widely distributed dengue-like fever, which is transmitted principally by the moth midge or sandfly, Phlebotomus papatasii (Fig. 68, 1, 2, 3, 4). Clinically this fever resembles somewhat dengue, influenza, and similar febrile infections, but the fever lasts only a few days (usually three, hence also the name “three-day fever”). The causative agent is known to be a filterable virus which is present in the blood during the first twenty-four to forty-eight hours. The developmental period of the virus in the insect is from six to eight days, and it may be passed on to the next generation of sandflies. Squashing the fly while it is biting seems to be necessary for successful conveyance of the infection.

KALA-AZAR OR “DUM-DUM” FEVER
(Leishmania donovani infection)

Kala-azar occurs in the Mediterranean littoral, southern Russia, Iraq, India, North China, and other parts of the world. Opinions vary as to whether transmission is direct through the bite of the fly or from the crushing of the fly and subsequent rubbing into the wound of the crushed remains of the infected fly (Chapter IV). In the local manifestation of oriental leishmania infection, the causative organisms (L. tropica) inhabit the skin only; they do not invade the viscera.

VERRUGA PERUVIANA, CARRION’S DISEASE, OR OROYA FEVER
(BARTONELLIASIS)

Verruga peruviana occurs in certain narrow canyons on the western slopes of the Andes at altitudes between 3000 and 10,000 feet. The local lesions and the preceding febrile condition are phases of the same disease. These were only suspected of being related before the so-called “Bartonia” bodies (Bartonella bacilliformis, Strong) were found to be the true etiologic agent of both conditions.

After an undetermined but probably short incubation period,
a fever of from two to four weeks' duration develops, characterized by prostration, marked anemia, and high mortality. If the patient survives, the fever subsides and the eruptive or verruga (wart) stage begins.

**Genus Simulium** (Buffalo Gnats or Black Flies)

The adults of the genus *Simulium* are rather small (1–5 mm. long), of robust build, with strong legs and short, broad, delicate wings. The larvae and pupae live in running streams, attached to stones, vegetation, and the like. *Simulium* larvae possess silk glands, the secretion of which enables them to cement themselves fast and spin a cocoon-like wall pocket for protection during pupation. The adult is released from the pupal case by gas pressure within, which splits the pupal skin; and on reaching the surface of the water it takes flight almost immediately.

Diseases which species of *Simulium* are known to transmit are onchocerciasis and a leucocytozoan infection of certain birds.

**Onchocerca volvulus and Onchocerciasis**

Although these small black flies (Fig. 68, 8, 9, 10) have long been suspected of transmitting disease, it is only recently that definite incriminating evidence has been revealed in the case of man. A study of onchocerciasis * (a conspicuous, subcutaneous nodular tumor of the trunk and head, caused by *O. volvulus*) has resulted in establishing the carrier relationship of *Simulium* species to this disease both on the west coast of Africa, and in southern Mexico and Guatemala.

The adult female worms of *O. volvulus* measure from 35 to 55 cm. long; the males much less. Several adult worms and numerous viviparous larvae are generally found in each tumor. The eye is often seriously involved, with a resulting keratitis, evidently due to migration of the larvae. A bovine form of this disease, of significant economic importance, has been reported from Queensland, Australia.

* A brief clinical description of this condition has already been given in Chapter X.
LEUCOCYTOZOOON INFECTION

Of considerable comparative interest is this sporozoon disease of domestic turkeys and wild and domestic ducks, which is known to be transmissible through the agency of certain species of Simulium in Michigan and Nebraska, respectively. The life cycle of the parasite, which infects leucocytes, is similar to that of malaria plasmodia, the asexual cycle occurring in the avian host and the sexual cycle in the transmitting insect.

Since effective control of the transmitting agent appears to be impracticable, procedures designed to minimize its opportunities for biting and the use of suitable repellants in case of emergency are probably all that may be attempted practically. Effective prevention of spread of infection requires that active cases of the disease be shielded from the bites of Simulium.

GADFLIES OR HORSEFLIES AND DEERFLIES

Gadflies are comparatively large, stocky, usually dark-colored flies, among which the Tabanidae are of medical importance. They vary in size; some are as small as the common blowfly or even smaller, while many are much larger. As a rule, it is only the smaller species that attack man regularly. The head is usually as wide as the thorax, with very large, often brilliantly colored compound eyes which meet in the midline in the males, but are more or less separated in the females. The antennae are superficially three-jointed, but the terminal ones are usually made up of several somewhat smaller segments. The length of the proboscis varies greatly among different genera. Only the females have mouth-parts adapted to piercing the skin and drawing blood.

Life Cycle. The eggs are deposited in groups of from 100 to 700 or 800 and are usually attached to the leaves or stems of plants or to rocks overhanging the water or close to it. Under favorable conditions, the eggs hatch in from five to seven days, drop into the water and the larvae then burrow into the mud. They feed voraciously on all kinds of other soft-bodied larvae, moulting from time to time. As they grow older, they seek drier places and
finally pupate just below the surface of the ground, often some distance away from water. Development is rather slow, the time required for completing the entire life cycle varying from about four months in the tropics to a year or more in the temperate zones.

**The Genera Tabanus and Chrysops**

Of the two principal genera, *Tabanus* (horseflies, usually with unicolorous wings, Fig. 69) and *Chrysops* (deerflies, usually with brown spotted wings, Fig. 70), the former has been shown to be capable of transmitting mechanically anthrax, and likewise surra, a trypanosome infection of horses in southern Asia. Species of *Chrysops*, on the other hand, are known to be intermediate hosts of the ocular filarial worm *Loa loa* of west Africa, and a common insect transmitter of *Pasteurella tularense*, causative organism of tularemia, a plaguelike disease of rodents and man in California.

**The Higher Diptera**

The higher diptera of medical importance may be conveniently subdivided into the blood-sucking muscid and pupiparous flies, and the non-blood-sucking, filth, and myiasis-producing flies.

**The Blood-Sucking Muscid and Pupiparous Flies**

Of the muscid flies, only a few genera are blood-sucking. Among these the stablefly (*Stomoxys calcitrans*) and the hornfly (*Haematobia irritans*) are widely distributed. Species of the genus *Glossina*, which are in reality larviparous, are related both to the true muscid flies (which are oviparous, most of them falling under the non-blood-sucking group) and the true pupiparous flies of the family Hippoboscidae. The Glossininae or "tsetse flies" are confined to Africa south of the Sahara Desert, except one species which has a wide distribution in that continent well north of the equator, and has also been found in the southern part of Arabia. While *Stomoxys calcitrans* and various species of *Glossina* frequently attack man, the hornfly rarely does.

*For brief description, see Chapter X.*
Fig. 69. *Tabanus punctifer*. The inset shows the fly natural size (From Cameron. The Parasites of Man in Temperate Climates, 1940. Courtesy of the University of Toronto Press.)

Fig. 70. *Chrysops discalis*. The inset shows the fly natural size (From Cameron. The Parasites of Man in Temperate Climates, 1940. Courtesy of the University of Toronto Press.)
Stomoxys calcitrans (THE BITING STABLEFLY)

Morphology. In general appearance and size, *Stomoxys calcitrans* (Fig. 71, A, B) resembles the common housefly. It may be recognized readily by its prominent, blackish, piercing proboscis which projects forward from the under side of the head. Both male and female are vicious biters. The mouth-parts are fewer in number and are more highly specialized than those of the mosquito or horsefly. The component parts are shown in Figure 72.
The labium is long and rigid, being well chitinized and equipped with rasping teeth just inside the edge of the rudimentary labellum. Preparatory to biting, *S. calcitrans* quickly rasps through the skin by means of its exerted toothed labellum, all the remaining mouth-parts following quickly, accompanied by a sharp stabbing pain. This fly draws blood quickly and if undisturbed fills to capacity in from three to four minutes; when disturbed, it merely changes position or flies to another animal to continue its exclusive blood meal.

**Life Cycle, Epidemiology, and Control.** *S. calcitrans* breeds in decaying hay, straw, grass, rotting grain, or stable manure. The small, ovoid eggs, varying from about forty to seventy-five, are laid one at a time. After two to five days, the larvae hatch and begin to feed on decaying vegetable matter. At the end of from two to three weeks, the larvae pupate. The pupae are elongate-ovoid,
chestnut brown in color, and measure from 5 to 7 mm. in length. They are found in the ground, beneath or around the edges of the breeding place where drier conditions exist. After from five to ten days or longer, the adult flies emerge from the pupal cases.

Like the Tabanidae, the biting stablefly appears to serve only as a mechanical transmitter of disease. If present in unusual numbers, however, it may become a serious pest, resulting in economic loss manifested by debility and even death of large farm animals, and serious curtailment of milk production.

Control and prevention of the breeding of S. calcitrans is difficult because the eggs and larvae are scattered and not readily attacked by natural enemies. Piles of hay, straw, and vegetation should not be allowed to rot, and manure should be kept on a screened-in platform with a slatted bottom above a concrete basin containing a few inches of water.

Haematobia irritans (the hornfly)

Before passing on to a description of the "tsetse" flies (Glossinae), a brief description of the hornfly should be given. This is a small cousin of the stablefly (Fig. 71, A), which it resembles in general appearance, but it is only about half the size, being approximately 4 mm. long. Its life cycle is completed in proportionately less time than is that of the stablefly.

The hornfly is also known as the "Texas fly," and has been comparatively recently introduced into the United States from Europe, where it has been a significant cattle pest for many years. The female deposits her eggs in small numbers, most often in freshly passed cow manure. These eggs are relatively larger than those of S. calcitrans and are reddish brown in color, hence not easily seen on the cow droppings.

Large economic loss may result from the presence of this fly. Control may be most effectively accomplished by promptly scattering the cow droppings to dry or handling them in such a way as to prevent the maturing of larvae. This fly may serve as a mechanical transmitter of disease. Fortunately, however, it is not a frequent household visitor.
FLIES AND DISEASE TRANSMISSION

THE LARVIPAROUS *Glossina palpalis* AND *morsitans*

*Glossina palpalis* and *morsitans*, two human trypanosome transmitters, are considerably larger than the common stable fly, *S. calcitrans* (Fig. 71, A, B). Whereas the antennal arista of *S. calcitrans* have simple hairs on one side only, those of the *Glossinae* are feather-like as shown in Figure 71, C, D, E. They are brownish in color and have a formidable-looking proboscis, projecting rigidly forward from the head, which appears strikingly bayonet-like and is composed of parts comparable to those of *Stomoxys*.

The venation of the wing is characteristic of the genus *Glossina*; that is, the fourth longitudinal vein bends suddenly upward before it meets the anterior cross vein which is very oblique. This feature is unique among diptera of the present time. In fact, some authors have gone so far as to suggest that fossil imprints of strikingly similar wing venation from the Miocene of Colorado may be, phylogenetically, those of a closely related genus or a close equivalent of the present one.

**Life Cycle.** As in *S. calcitrans*, both sexes of *Glossina* are blood-suckers. The female gives birth to full grown larvae which are extruded singly at intervals of from ten to twelve days throughout her life. During the intra-uterine state, there are three moltings, the larvae feeding on fluid from special uterine "milk" glands. The newly extruded larvae are creamy white to pale yellow in color and have a pair of shining black lobes (breathing spiracles) at the posterior extremity. The larvae, being unable to crawl as do most other muscid larvae, move and burrow by a sort of peristaltic action of the whole body.

The necessary conditions of pupation, which usually takes place within an hour of deposition, are a moist soil and adequate protection from drying winds and the sun. The pupal stage lasts from three to four weeks or longer, depending largely on the temperature. The imago or adult fly emerges by pushing off the pupal case cap by means of a temporary bladder-like structure called the "ptilinum." This mechanism is common to *Stomoxys,*
Glossina, and other higher diptera. In the Glossinae, although both sexes feed on blood, only females are capable of transmitting trypanosomiasis biologically. They rarely feed at night.

Glossina palpalis is found generally in the moist jungle country of west and central Africa as far east as lakes Albert and Victoria. It inhabits areas near water where the undergrowth is thick. The flies are unusually abundant at fording places, boat landings, and along native trails. For larval deposition, the female prefers rather heavily shaded sandy beach or loose soil near water.

Glossina morsitans. The bite of Glossina morsitans was known to be fatal to domestic animals long before the causative organism was discovered. This species was the “tsetse” fly associated with the cattle disease known as “Nagana.” Its preferred habitat is savannah or parkland bordering on the wet jungle where G. palpalis abounds. Its range extends from Rhodesia up through the great lake region of central Africa to the Anglo-Egyptian Sudan and thence west to Senegal on the west coast. It is said not to be uniformly distributed but to occur in so-called “fly belts” which expand and contract with the changing seasons. For larval deposition the females prefer loose soil under fallen trees or limbs which do not quite touch the ground.

Control and Prevention. Measures of control and prevention directed against the larvae or breeding places are frequently ineffective. Successful application of control efforts against the adult tsetse flies is likewise difficult because of their peculiar habits. The following measures have been found helpful in varying degrees, depending on local circumstances and conditions: (a) equipping of native carriers with special dark colored fly-paper back shields; (b) clearing away of undergrowth, which furnishes the dense shade necessary for safe deposition of larvae and pupation; (c) destruction of game animals on which the adult flies feed, particularly in areas in which Trypanosoma rhodesiense infection occurs and G. morsitans abounds; (d) use of specially designed mechanical trapping devices.

The human trypanosome diseases, whose transfer is associated biologically with these tsetse flies, are described briefly in Part I.
ECTOPARASITIC PUPIPAROUS FLIES

These are commonly known as "tick flies" or "louse flies." Like the Glossinae, they produce but one offspring at a time. They rarely attack man, but some of them serve as vectors of blood-inhabiting protozoa of birds and mammals on which they are ectoparasites.

Certain genera of these flies are known to transmit species of the protozoan genus Haemoproteus to nesting pigeons in the tropics and subtropics, and to the mourning dove and quail in California. Horses, cattle, dogs, camels, and sheep are among the mammals on which other genera of these flies are ectoparasitic and are possible transmitters of nonpathogenic trypanosomes.

THE NON-BLOOD-SUCKING, FILTH, AND MYIASIS-PRODUCING FLIES

In relation to diseases of man, these flies, in addition to their role as simple mechanical transfer agents of various parasitic infections, may transport eggs of other parasitic insects or deposit their own eggs or larvae in the vicinity of natural body orifices, discharging sinuses, or wounds. Thus they contribute directly or indirectly to parasitic invasion of tissue by insect larval forms. The term "myiasis" is applied to diseases or syndromes produced through tissue invasion by such fly-larvae.

Mechanical transmission of infectious material by flies may be accomplished (a) by means of soiled external appendages such as hairs, bristles, foot-pads, and mouth-parts; or (b) by ingestion and passage into the digestive tract of the fly, to be deposited later in vomit or fecal droppings.

EYE OR FRUIT FLIES

Eye flies (sometimes also called eye gnats) are similar in appearance but smaller than the common fruit or vinegar fly, Drosophila. They are referred to as "eye gnats" because they seem to prefer eye secretions and may at times cause a variety of epidemic conjunctivitis known as "pink-eye." Recent evidence from Jamaica seems to indicate that certain species of this fly may be
instrumental also in the transmission of yaws, a tropical treponemal skin eruption.

Although these flies are classed as nonbiting, the simple labellum is provided with spines, which apparently serve as cutting or rasping instruments and are capable of producing small abrasions of the conjunctiva or skin conducive to the passage of pathogenic micro-organisms harbored by the insect.

The larvae of many of these flies, often called stem maggots (family Chloropidae), live in grass and other plants. Those of the genus Hippelates, however, develop in decaying animal and vegetable matter and even in feces of various animals. Species of Hippelates found in the southern states and in California are known as "pink-eye flies" or "sore eye flies." Siphunculina funicola is known as the "eye fly" in India, Ceylon, and Java.

THE COMMON HOUSEFLY AND OTHER HOUSE-INVADING FLIES

The true housefly, Musca domestica, according to counts of collections obtained from dining-rooms in different parts of the United States, comprises ordinarily about 95 per cent of the flies found in dwelling houses. Counts for the lesser housefly, it is estimated, rarely exceed one quarter of this figure. The male housefly is somewhat smaller than the female, its length averaging about 6 mm. as contrasted with 7.5 mm. for the female. Further general description appears unnecessary except to refer to certain features of the proboscis and to call attention to the hairiness both of body and legs. All of these contribute only too well to the transfer of filth and infection to eating utensils, food, and the person of man himself.

The prominent proboscis of the housefly is fleshy, consisting mainly of a grooved labium which terminates in a pair of abrading labella. It is highly muscular and may be partially drawn up to be out of the way when the fly is not feeding. Upon the upper (grooved) surface of the labium lies the inconspicuous blade-like labrum, which forms a food channel when superimposed upon the hypopharynx, supported by the labium. The hypo-
pharynx contains the salivary canal. The general appearance and relationship of these structures are shown in Figure 73. The maxillae appear to be fused with the fleshy basal part of the proboscis, the maxillary palps remaining as separate appendages.

**Life Cycle.** Familiarity with the development of the housefly is essential in planning effective control measures. The adult female (Fig. 74) lays from 75 to 150 eggs singly, but piles them up in masses quite rapidly. One female may lay a dozen or more such batches of eggs at intervals of several days throughout her life, the length of which may be two months or more. She may begin to lay eggs in from nine to twelve days after emerging from the pupal case. The elongated pearly white eggs (about 1 mm. in length) frequently hatch in from twelve to twenty-four hours in
summer when deposited in manure, the locus of preference.

The temperature of an average manure pile to which material is added daily has been found to vary from $18^\circ$ to $66^\circ$ C. The young, growing larvae are most numerous where temperatures are between $45^\circ$ and $55^\circ$ C. Larvae feed voraciously and under favorable conditions pupate in about five days, the pupal stage lasting another four or five days when the outside temperature is around $18^\circ$ C. Flies hatched from eggs laid in manure have been found to go through the complete life cycle in as short a time as twelve to fourteen days. At $30^\circ$ C. the minimum time for complete metamorphosis is reported to be nine and one-third days. The late stage pupal cases are darkish brown, smooth ovoids about 6 to 8 mm. long, and may be identified readily.

**Epidemiology, Control, and Prevention.** The role of houseflies in the transmission of disease is almost exclusively passive, but none the less effective. This is perhaps best illustrated in fecal-borne bacterial diseases such as the typhoid fevers and dysenteries. Other body wastes containing infectious material may also be effectively transported by houseflies.

Proper fly control requires the co-operation of the whole community, since a single neglected manure pile may produce enough flies to annoy a whole village. Control measures which are more
or less effective against houseflies are (a) proper screening of houses; (b) minimizing the breeding of flies in manure and rotting organic wastes; (c) swatting; (d) trapping flies by use of sticky fly-paper and poisoning with a slightly alkaline 2 per cent solution of formalin in milk or sweetened water placed in open dishes or pans; (e) destruction of adult flies in confined spaces by fumigation (burning insect powder such as pyrethrum), or by spraying with approved commercial preparations.

Of all these methods, destruction of larvae at the source, as described on page 286 for the stablefly, is to be preferred where effective community co-operation is feasible. Lacking this co-operation, however, individuals and small groups may achieve relative security and comfort by intelligent application of a combination of the methods of control briefly set forth in the preceding paragraph.

THE LESSER HOUSEFLY AND THE LATRINE FLY *

Among other flies not infrequently found in houses are species of the genus Fannia (Homalomyia) known as the "lesser housefly" and the "latrine fly." The former (Fannia canicularis) is the more common frequenter of houses, hovering in mid-air or flying here and there about the room. In size this species (Fig. 75, io, ii) is somewhat smaller than the male housefly, which it resembles closely.

The eggs are deposited on decaying vegetable matter and excrement, particularly that of man, horse, and cow. The larvae emerge in twenty-four hours or less and may be recognized as flat, spiny grubs about 6 mm. long when full grown. The larval stage lasts five or six days and the pupal stage requires about seven days for its completion under favorable conditions.

The latrine fly (Fannia scalaris) is very similar in appearance to the lesser housefly just described, but is slightly larger. It frequents insanitary latrines mostly, and comes into houses less often.

* These flies apparently are not attracted to foodstuffs in houses and for this reason are of much less importance medically than is the common housefly.
The larvae are distinguished from those of the lesser housefly by the lateral spindlesike appendages which are single and feather-like in appearance, instead of being double as in the larvae of the latter.

![Fig. 75. Myiasis-producing flies (in which the larval stage is important). 1, Cochliomia (Chrysomyia) americana; 2, C. larva; 3, Dermatobia hominis larva, early stage (ver macaque); 4, D. hominis larva, later stage (torcel or berne); 5, D. hominis; 6, Auchmeromyia lutecola; 7, A. lutecola larva, (Congo floor maggot); 8, Sarcophaga (Wohlfahrtia) magnifica; 9, S. magnifica larva; 10, Fannia (Homalomyia) canicularis; 11, F. canicularis larva. 1-9, X 1 app.; 10, 11, X 2.5 app. (From Stitt, Clough and Clough. Practical Bacteriology, Haematology, and Parasitology. Copyright the Blakiston Company, Publishers.)

OTHER FLIES RESEMBLING THE HOUSEFLY

Certain other flies which resemble the housefly in color, pattern, and approximate size are Musca autumnalis (Europe and Asia); Muscina stabulans* (the non-biting stablefly), which is somewhat larger than the female housefly and is said to occur more generally about stables; Pollenia rudis, the "cluster fly," may be distinguished readily from all other houseflies in that the

* According to some authorities, this species is seldom found in either houses or stables, being a scavenger as larva and perhaps eating other insect larvae. With the exception of Musca autumnalis, these flies are not generally considered medically important. They may be annoying at times, but that is all.
FLIES AND DISEASE TRANSMISSION

thorax is clothed with short, silky yellow hairs. It is usually a larger insect than the common housefly, is more stockily built, and is slower in its movements.

MYIASIS-PRODUCING FLIES AND MYIASES

This assembly of non-biting flies, in addition to their capabilities as passive transmitters of disease, may serve more specifically through their larvae to cause disease directly or indirectly. The various conditions arising are conveniently classified as the "myiases." Clinically, these may be subdivided into cutaneous, cavity and wound, intestinal, and external myiasis.

The myiasis-producing flies themselves and the lesions produced by their larvae may be classified and discussed more logically under the following familiar biologic terms: (a) Obligatory myiasis-producing flies. The pregnant female deposits eggs or larvae in or near living tissues, which regularly become parasitized. (b) Facultative myiasis-producing flies. The pregnant female ordinarily oviposits on decaying animal tissues, but may also occasionally select neglected or unprotected wounds. (c) Accidental myiasis-producing flies. Flies of widely different habits which normally oviposit in animal excrement, meat, or decaying vegetable or animal matter, but whose larvae are at times ingested by man.

OBLIGATORY MYIASIS-PRODUCING FLIES

These non-biting flies may be divided into three categories as follows:

Group 1 comprises flies not attracted directly to the animals which are to serve as hosts for their larvae, but which may deposit eggs or larvae in places frequented by these hosts. The larvae are very often capable of penetrating the unbroken skin. Data regarding these flies and the lesions their larvae cause may be summarized as follows:

The American Tropical Warble Fly (*Dermatobia hominis*). This fly (Fig. 75, 5) frequently lays its eggs upon the body of some other insect which visits animals or man. The eggs are sometimes
found glued to the bodies and legs of certain species of mosquitoes as well as to other insects that are attracted to wounds.

Eggs transported to a suitable warm-blooded animal incubate and hatch almost immediately after they come in contact with the animal. The larvae (Fig. 75, 3) then penetrate the skin of their new host. Growth of the larvae (Fig. 75, 4) and the accompanying irritation lead to the formation of local lesions beneath the skin called “warbles.” (Death or killing of the larvae in situ in case of man often leads to serious and most troublesome complications.) When fully grown, the larvae emerge and drop to the ground to pupate.

In this connection mention should also be made of certain blood-sucking fly larvae of nestlings of the temperate zone, and also the “Congo floor maggot” (Fig. 75, 7) of Africa. The former are responsible for the death of many young birds, while the latter is a troublesome pest in infested native huts.

The Tumbu Fly (Cordylobia anthropophaga) of Africa is thought to be attracted by the feces of rats to the places these animals frequent. Here the pregnant female deposits the larvae in moist soil. The first stage larvae attack not only the rat but often also the dog and man. Presence of larvae in the skin causes irritation and pain, the resulting lesion resembling a small boil.

Group 2 comprises flies, the females of which are attracted to animals by the odor of blood or necrotic tissue. Eggs or larvae are deposited by the female in or near a wound or diseased tissue, the larvae not being able to penetrate the unbroken skin. The larvae cause cutaneous, cavity, and wound myiasis. In contrast to the single larval invasions of Group 1, the lesions of this group are generally multileral. Notable examples are the “screw worm flies,” Cochliomyia (Chrysomyia) americana and C. bezziana of the Old World (Fig. 75, 1) and the “flesh flies” (sarcophaga, Fig. 75, 8, 9) found everywhere.

Group 3 comprises genera most of which belong in the family OESTRIDAE, a heterogenous assemblage. Common to all the genera is the inability of the larvae to live except as parasites of animals, and the rudimentary mouth-parts of the adults. The larvae are
found in both wild and domestic animals, and only accidentally in man. The usual locations in their hosts are the skin, nasal passages, and stomach. The female fly lays her eggs or larvae on hair or other parts of the body of an animal. The larvae attack first the adjacent tissues, and after migration or transfer, other parts of the body.

One of the most interesting and important genera of this group of flies is *Hypoderma*, whose larvae cause warbles in cattle. The adult fly is large, stocky, hairy, and bee-like in appearance. The females attach their eggs (about 100) to the hairs of cattle. The tubercle-spined larvae soon hatch, penetrate the skin, usually through a hair follicle, and migrate extensively throughout the body. After from three to six months the mature larvae (approximately 25 mm. long) congregate in the subcutaneous tissues of the back, where the characteristic indurated boils or warbles may be seen. During the course of another month, the larvae work their way to the surface of the skin, drop to the ground, and pupate.

In certain cattle-raising areas, man is also occasionally parasitized. The larvae, migrating under the skin, produce symptoms known in popular phraseology as "creeping eruption." This is not, however, the only condition of this nature in man to which this expression has been applied. (See page 163 for a brief description of a similar condition caused by aberrant hookworm larvae in man, and page 223 regarding microfilaria of *Loa loa*.)

Larvae of other genera cause the so-called nasal "bats" in sheep and antelope, and stomach bots in horse, rhinoceros, and elephant. In horse bots the eggs are attached to hairs. They hatch under the influence of friction and moisture. When the animal licks its own body or that of another infested horse, the larvae cling to the tongue and are subsequently swallowed. Attaching themselves to the stomach wall, they remain there until mature, when they become detached, pass out with the feces, and pupate in the earth. Rare instances of suspected bot infection in man have been reported.
FACULTATIVE MYIASIS-PRODUCING FLIES

These flies, commonly called "blowflies," deposit their eggs principally on dead animals, where the larvae feed on the decaying tissues of the carcass. Larvae of some species are occasionally found in neglected wounds of animals and man (some of them occurring rather frequently). In a few instances, an obligatory parasitism has developed. This type of myiasis is probably the most dangerous for man and is frequently of serious import in sheep, cattle, and horses because of the extensive tissue involvement. New World "screw worms," the larvae of Cochliomyia (Chrysomyia) americana, frequently infest the wounds of animals and sometimes the nose of human beings in the southern United States and tropical America. This genus has developed such a degree of parasitism that it is generally classed under the obligatory parasites (page 295).

ACCIDENTAL MYIASIS-PRODUCING FLIES

These are flies of widely differing habits, which ordinarily oviposit in excrement or decaying vegetable matter and are only accidentally ingested by man. Although most of the eggs and larvae ingested do not survive passage through the stomach, there is sufficient clinical evidence to show that the presence of larvae of various flies in the stomach or intestines has given rise to definite irritation with related symptoms. The "rat-tailed" maggots of the genus Eristalis, a syrphid or hover fly have been reported to cause severe gastric myiasis. In some instances, larvae of other flies have been reported to cause unusual manifestations. Ordinarily, however, intestinal myiases are transient and comparatively unimportant.

IDENTIFICATION OF FLY LARVAE

Accurate identification of the larvae of the various species of flies requires detailed knowledge of the morphology. Excellent keys for this purpose are available. In brief, identification is based on the general shape and external appearance; the character of the stigmal areas; the nature of the spiracles, both anterior and
posterior with reference to their particular settings; and the structure of the cephalothoracic sclerite or plate.

**BLOWFLY "MAGGOTS" IN SURGERY**

Blowfly maggots have been utilized in surgery, to some extent, for centuries; and recently they have been employed rather widely, particularly in chronic bone infections. The flies most commonly used as sources of the larvae are the well-known blowflies, *Lucilia sericata* and *Phormia regina*.

It is important to use maggots which feed only on necrotic tissue. Even in these species, however, there is some danger that the larvae may attack normal tissue. For this reason, use of the fly maggots in surgery has been largely discontinued. In treating chronic infections, non-living materials having similar effects have been largely substituted.
CHAPTER XIV
OTHER ARTHropods in Disease Transmission

In addition to flies and mosquitoes, there are a number of other groups of arthropods involved in the transmission of disease. These groups will be discussed in this chapter with brief reference only to systematic classification. Several species definitely associated with transmission of disease have been referred to already in connection with worm parasites.

Lice, Genera Pediculus and Phthirius

In general, lice (Anoplura) are of two different types, namely, those which chew like the beetle or grasshopper, known as bird lice or Mallophaga; and those which bite and suck, having mouthparts adapted for both piercing and sucking, known as Siphunculata, or true lice. The latter are of considerable medical importance. There are two distinct species—Pediculus humanus (varieties capitis and vestimenti) and Phthirius pubis—which vary considerably both in general morphology and in regional location on the body.

Pediculus humanus (vars. capitis and vestimenti)

Pediculus humanus presents two slightly different forms, depending on the locations in which they generally occur. The essential morphology is similar in both varieties, but there are certain minor differences which aid in identification. P. capitis is smaller in size, shows more pronounced “festooning” of the abdomen, and greater hairiness than does P. vestimenti. Some authors deem these superficial differences adequate evidence on which to assign each to the status of a distinct species.
The body of this louse is ventrodorsally flattened, pyriform in shape, and about 1.5 to 2 mm. long. Palps and wings are not present as in most insects (Fig. 76, 1, 2). The mouth-parts consist of a kind of labrum-epipharynx, sometimes referred to as the “haustellum.” This bears six pairs of minute teeth distally, which are everted when the louse feeds and serve to anchor it to the skin of the host. Within the haustellum there is a dorsally situated, lightly chitinized food canal. Ventral to this is a sheath containing the protrucible piercing or stabbing apparatus, which consists of a pair of closely approximated dorsal stylets and a slender, tubular, single, median-ventral one representing, respectively, the maxillae and hypopharynx. Through the latter the salivary duct passes. The stylets are protected and supported by a grooved structure, the labium. Preparatory to feeding, the louse applies the haustellum to the skin, inserting its everted teeth and forc-
ing the three stylets down into the dermis. At the same time droplets of saliva exude from the salivary duct in the hypopharynx, and blood is drawn up through the funnel-like food canal to and through the pharynx and esophagus into the stomach.

*Pediculus humanus capitis* (De Greer, 1778)

The habitat of *P. capitis* (Fig. 76, 1) is the head, where the pregnant female attaches her eggs (called “nits”) to hairs; that of *P. vestimenti* (Fig. 76, 2) is the clothing, where the female attaches her eggs to fibers of cloth. The eggs hatch in from five to nine days, depending on the temperature of the environment. If the egg-infested hair is cut or the egg-infested clothing not worn or exposed to cold, the eggs may lie dormant without hatching for thirty-five days or longer. There are several larval stages; in all these, the larvae closely resemble the adults except for size. If the growing larva is able to feed on blood regularly, it reaches the adult stage in from twelve to fourteen days after hatching.

*Pediculus humanus vestimenti* (De Greer, 1778)

The body louse is an important transmitter of communicable diseases. It serves as the common vector of epidemic typhus fever,* trench fever, and European relapsing fever, the two former caused by *Rickettsia prowazeki* and *pediculi*, respectively, and the latter by *Borellia recurrentis* (*Spirochaeta obermeieri*).

*R. prowazeki* lives in the epithelial cells of the gut of the insect, causing many insect deaths, while *R. pediculi* occurs only in the lumen of the louse’s gut. *B. recurrentis* inhabits the blood of the louse. It is transmitted, not directly by bite or through contamination with feces, but through crushing of the louse when it is attempting to bite or in the act of doing so.

*Typhus fever, endemic in type, has long been known in Mexico under the name of tabardillo, and scattered cases of a similar nature have been reported in the United States from time to time. Owing to a close resemblance of the skin eruptions in tabardillo to those in Rocky Mountain spotted fever, the two diseases were formerly thought to be the same. Ricketts proved this belief to be incorrect, but unfortunately he died from tabardillo during the course of his investigations.*
**Phthirius pubis** (Linnaeus, 1758)

*Phthirius pubis*, popularly known as the “crab louse” (Fig. 76, 3), is usually found among the hairs of the pubic region, but in badly infested individuals, it may spread to adjacent parts of the body or even to the head. It attaches its eggs to hairs as does *P. capitis*, giving rise similarly to “nits.” Its smaller size and different shape make it readily distinguishable from *P. humanus*, vars., *capitis* and *vestimenti*.

The crab louse is not important as a specific transmitter of disease. The infestation is generally considered somewhat difficult to control in those usually afflicted. However, local application of blue ointment gives prompt relief if there is satisfactory cooperation by the patient. If this ointment irritates the skin excessively, Curran advocates a pyrethrum wash of about three times the strength of “flit,” which he has found effective.

**Louse-Borne Diseases**

Irritation of the skin due to infestation by lice, known as “pediculosis,” is of itself often of sufficient intensity and significance to warrant concern on the part of clinicians, sanitary and school authorities. However, it is the specific role which pediculi play as disease carriers that may have far reaching effects when louse infestation is not controlled. This is particularly true where human beings are crowded together in jails, in times of disaster and famine, and in army barracks or trenches. Epidemic typhus, trench, and European relapsing fevers are diseases transmitted through the agency of lice infected with the specific causative agents. Among these, epidemic typhus fever warrants special brief clinical consideration here. Detailed information on all of these conditions is readily available in the literature.

The *Rickettsia* bodies of typhus fever require an incubation period of five to six days in the louse before infection can be transmitted through its bite, but fecal-borne infection may occur several days earlier. In man the incubation period averages from eight to twelve days, the onset symptoms of the disease often being...
chills, rigors, severe frontal headache. Epigastric pain and vomiting are also frequent early manifestations. On the third day the fever rises, frequently somewhat suddenly, to 103° or 104° F. and remains at this level, with slight morning remissions, for from twelve to fourteen days. The drop in temperature toward the end of this febrile period is often rather abrupt. A foul mouth, offensive breath, epistaxis, severe vomiting, apathy, and stupor are frequent accompanying symptoms.

A characteristic pleomorphic rash appears, usually on the fifth day, first showing upon the abdomen and inner aspects of the arms, spreading over the chest, back, and trunk but rarely involving the face. Manson-Bahr (1940) describes this rash as consisting of "roseolar macules, together with a fine irregular dusky mottling underlying the epidermis," which is generally termed subcuticular mottling. The rash fades away gradually although it may persist for some days. Rickettsia attack the cytoplasm of endothelial cells lining the smaller blood vessels, especially those of the skin and brain. This fact underlies the pathologic and clinical manifestations of typhus fever. In diagnosis, the Weil-Felix agglutination reaction is most important. In this test the patient's serum is mixed with Proteus vulgaris cultures designated as OX1, OX19, OXK, and OXR. (See page 327.) Epidemic typhus fever may resemble such diseases as typhoid fever, malaria, relapsing, and Rocky Mountain spotted fever. Differentiation from endemic (murine) typhus or Brill's disease may be made on the fact that epidemic typhus does not cause scrotal lesions in the guinea pig or febrile disease in the rat with apparent scrotal lesions.

Although Rickettsia pediculi has been accepted by some authorities as the causative agent of trench fever, this etiological relationship is largely based on circumstantial evidence, since these organisms have never been demonstrated in man suffering from trench fever. This fact in connection with the absence of vascular skin lesions so characteristic of Rickettsia infection and failure to transmit trench fever infection to any animal constitute adequate grounds for doubt of the validity of the etiological relationship. Trench fever is relapsing in type and is rarely if ever fatal. In this
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Respect it resembles dengue, which is caused by a filterable virus. The causative agent of trench fever, however, is not filterable as it occurs in the blood.

Relapsing (recurrent) fever, generally referred to as European relapsing fever, is transmitted by Pediculus humanus. It is endemic and frequently epidemic in Europe, Asia, and the northern part of Africa. Sporadic cases have been reported in the United States from time to time. The etiological agent, Borellia (Spirochaeta) recurrentis or obermeieri, is indistinguishable morphologically from Borellia duttoni of African or tick-borne relapsing fever. Clinically the disease is characterized by sudden onset with chills or rigor, giddiness, epistaxis, vomiting, photophobia and intense headache. The incubation period may vary from two to about twelve days. The temperature usually rises rapidly to 104° or 105°F or higher. Quite commonly an erythematous rash occurs during this first period of fever, which may last from five to seven days. The spleen is invariably enlarged and tender. The temperature then drops rather abruptly and a period of apyrexia and an apparent recovery sets in, followed in seven to nine days by a relapse. This first relapse is generally milder than the original attack and rarely lasts as long. With subsidence of this temperature convalescence usually sets in, but in severe cases a second relapse may occur and convalescence be prolonged, accompanied by various complications.

Prophylaxis of Louse-Borne Diseases

Control and prevention of louse-borne diseases, while comparatively simple in principle, require the exercise of extreme care and vigilance in carrying out the necessary details. Prophylaxis in the individual, the family, and the community become increasingly difficult for obvious reasons. Head lice may be eradicated by rubbing into the hair a mixture of equal parts of kerosene and vinegar, covering the head tightly with a towel for half an hour and then washing the hair with plenty of soap and warm water. Ordinary laundry and dry-cleaning processes are effective in destroying lice and their much more resistant eggs.
on the hair or clothing. A bath with cresol or kerosene soap will kill body lice on the individual. Further details on group and mass "delousing" are beyond the scope of this book. Needless to state, the minutiae must be carefully planned and carried out with reference to the particular circumstances and conditions to be dealt with.

THE BEDBUGS AND REDUVIID BUGS
(FAMILIES CIMICIDAE AND REDUVIIDAE)

Bugs have been variously classified as HEMIPTERA, HETEROPTERA, and RHYNCHOTA. The order HEMIPTERA, which normally have two pairs of wings, is now divided into two distinct suborders: (1) HETEROPTERA, in which the first pair of wings is thickened at the bases, while the distal overlapping portions are membranous; (2) HOMOPTERA, in which the first pair of wings is of about the same thickness throughout.

To the HETEROPTERA belong the true bugs, characterized by a jointed suctorial proboscis which, when not in use, particularly in case of bedbugs, is commonly flexed ventrally beneath the thorax; to the HOMOPTERA belong a wide variety of plant-eating bugs of great agricultural importance, both ecologically and economically. Some of these, under unusual circumstances, may bite man and lap or suck up the exuding blood.

CIMICIDAE OR BEDBUGS * (CHINCHES, MAHOGANY FLATS)

The CIMICIDAE have flattened bodies (adults 4 to 5 mm. long and 3 mm. wide), a three-jointed labium or rostrum, and four-jointed, conspicuous, freely movable antennae. The wings are vestigial; the fore-wings are reduced to mere pads and the hind-wings are entirely absent.

There are two species which have become preferential parasites of man and are commonly found in houses. These are known as Cimex lectularius, the common bedbug, and Cimex hemipterus,

* Other species which closely resemble bedbugs in general appearance are parasitic on poultry, pigeons, swallows, and bats. While these may attack man, they do not remain on him very long.
the oriental bedbug (Fig. 76, 4, 5). In the former, the lateral aspects of the convex dorsum of the prothorax appear as wing-like extensions. *C. lectularius* is world-wide in distribution. *C. hemipterus* has largely replaced *C. lectularius* in the tropics of the Old World, but is comparatively rare in the American tropics.

**Morphology of Mouth Parts.** The hinged proboscis is made up of four stylets representing maxillae and mandibles. The maxillae stylets are somewhat grooved and form a sucking tube when approximated to the grooved, three-jointed labium. In biting, *Cimex* brings the proboscis from its folded position against the ventrum to a vertical position, inserts the toothed ends of the bladelike mandible stylets together with the maxilla stylets, and proceeds to suck up blood through the channel formed by the maxilla stylets and the labium.

**Life Cycle.** The female lays 150 or more small but distinctly visible, oval, white eggs in a number of batches. The eggs hatch in ten days or more, depending on temperature. Just as the female requires a blood meal before she can mature her eggs, so the larvae (which resemble adults quite closely in form, but are smaller and paler in color) require in each of the five larval phases at least one blood meal in order to moult and pass into the next stage.

Without feeding, bedbugs may survive for several months, or perhaps a year, and in lieu of man will feed readily on rats and other animals such as the chicken. The average period of active life of bedbugs is from three to six months, but in cool places they may survive much longer. They live in cracks or crevices, especially about beds, feeding at night, and have a tendency to migrate from room to room or even from house to house. Soiled linen is a prolific means of transportation of bedbugs. Their foul, penetrating odor when crushed is characteristic.

**Epidemiology.** While bedbugs may transmit disease passively (experimentally, they have been proved able to do so), there is at present no convincing evidence that they serve as true intermediate hosts in nature for any known parasite. Although they have long been suspected of playing such a role in kala-azar, and
experimentally have proved to be a good incubator for the *Leishmania* organisms, numerous attempts to transmit this disease naturally through their bites have been negative.

On the other hand, they have been shown not to be efficient incubators of *Borellia duttoni* or *recurrentis*, anthrax, tuberculosis or typhoid bacilli. It has been reported, however, that they may transfer *Pasteurella tularensis* from mouse to mouse up to seventy-one days after an infective feeding, and their feces, when they are fed on rats suffering from plague, may be plague-infective for forty-eight days.

**Control and Prevention.** Bedbugs are easily killed by most insecticides, but because of their secretive habits, it is frequently difficult to bring the lethal agent into contact with them. Kerosene, gasoline, various phenol and cresol mixtures, and mercuric chloride solution are effective if properly applied.* A feather, small brush, or spray-gun constitute practical means of getting the insecticide into crevices where these insects hide. A very successful practice in laboratory animal quarters is a weekly sterilization of all the cages with live steam. Thorough fumigation with hot sulphur gas or cyanide gas has been found useful for bedbug extermination in barracks and similar places.

**The Reduviid Bugs**

Many of these bugs, popularly known as cone noses, assassin bugs, corsairs, and kissing bugs, are vigorous fliers and runners. A rather long, narrow head, distinct neck, and a conelike nose characterize Reduviid bugs, of which there are many genera. They are much larger than bedbugs and possess well-developed wings; but they have similar long, four-jointed antennae and comparable mouth-parts. The sturdy three-jointed proboscis is usually curved beneath the head in repose, but it is thrust forward when the insect bites or attempts to protect itself.

*For best results in control, the use of "rotenone" is recommended or rotenone and pyrethrum spray (of about double the strength of flit). These must be generously applied to both beds and bedding, et cetera.*
Triatoma (Mestor, Panstrongylus) megista

**Life Cycle.** The conspicuous barrel-shaped eggs of *Triatoma megista* (relatively much larger than those of the bedbug) are to be found in places where the adults (Fig. 76, 6) occur. The female deposits her eggs singly or in small groups, which usually hatch in from ten to twenty days under favorable conditions.

The metamorphosis is incomplete, a wingless nymph emerging from the egg. The nymphs feed on the juices of other insects and the blood of small animals such as birds and rodents. They mature rather slowly. It is believed that in most cases there is only one generation a year. These insects may pass the winter in the egg, nymph, or adult stage.

**Epidemiology.** In addition to the ordinary biting habits of Reduviid bugs, alluded to on previous page, *T. megista*, known also as “barbeiro,” appears to be a common transmitter of *Trypanosoma cruzi*, the causative agent of Chagas’ disease (Brazilian trypanosomiasis). The life cycle and mechanism of infection of *T. cruzi*, as well as the disease it causes, are discussed under the trypanosomiases (pages 76 to 82).

**Prophylaxis.** Owing to the flying skill of Reduviid bugs, their effective control is often both difficult and expensive. Proper construction, screening, and sanitation of houses, hospitals, and other buildings are most helpful. Unfortunately, however, it is in the adobe, thatched native huts that *T. megista* commonly occurs. The intelligent employment of strong disinfectants and the liberal use of whitewash are helpful adjuncts in prevention.

**THE FLEAS (ORDER SIPHONAPTERA)**

About 800 species of fleas, divided into six families, have been described. Although fleas tend to be host-specific, they are found on unusual hosts much more frequently than are lice, which are much less mobile in this respect. The larvae of fleas are rarely parasitic.

**Classification.** On the basis of their relation to man and his diseases, fleas may be roughly classified in two groups, depending on
the absence or presence of comblike structures, the “ctenidia,” which may be present on the ventral edge of the forehead (frons), on the prothorax, or on both of these parts.

1. Fleas without combs or “ctenidia.”
   - *Pulex irritans*, the human flea.
   - *Xenopsylla (Pulex) cheopis*, the rat flea (transmitter of plague).
   - *Tunga (Dermatophilus, Sarcopsylla) penetrans*, “chigoe” or chigger of tropical countries, not to be confused with the harvest mites or so-called grass ticks of temperate countries.

2. Fleas with one or more combs or “ctenidia.”
   - *Ctenocephalus canis* and *felis*, the dog flea and the cat flea.
   - *Ceratophyllus (Nosopsyllus) fasciatus*, the temperate zone rat flea.
   - *constable (Leptopsylla musculi)*, the mouse flea.

**Distinguishing Features.** Fleas are small, wingless, medium to dark brown, shiny insects, flattened laterally. Their mouth-parts are adapted to puncturing the skin and sucking blood. They have three pairs of prominent legs, the hind pair of which is longer than the others and well designed for jumping.

The antennae (Fig. 77) lie in a fossa closely approximated to the head. The mouth-parts consist of a pair of anteriorly placed jointed mandibles and a fused, stylet-shaped labrum-epipharynx. The latter, in conjunction with the mandibles, serves to form a food channel through which blood is sucked up. On the dorsal aspect of the ninth abdominal segment, there is a saddle-like depression in which lies a hairy padlike structure (probably sensory), known as the “pygidium.”

Males may be readily distinguished by the presence of two pairs of small claspers on the ninth abdominal segment behind the pygidium and the coiled, chitinous “cirrus,” within the region of the fifth, sixth, and seventh abdominal segments. Females may be recognized by the conspicuous gourdlike “spermatheca” in the last three abdominal segments. These structures, except the latter, together with various other distinctive features of flea anatomy and morphology, are shown in Figure 77.
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Life Cycle. The comparatively large, glistening white, ovoid eggs (0.5 mm. long) are laid a few at a time in the dust of floors, on hair (loosely), or in the nests of their hosts. After several days,

small bristled wormlike larvae with biting mandibles emerge from the eggs and feed on available organic debris and dejecta of adult fleas containing an abundance of dried blood.

In from one to several weeks, during the third larval stage, the larva spins a cocoon around itself and pupates. In some species, this quiescent stage lasts for a week; in others, nearly a year. The adults may survive for a year or more under favorable conditions, living for months without feeding if the climate is cool and moist. If, however, the climate is hot and dry, they may die quickly.

![Diagram of a male flea](Image)
Fleas without Combs or "Ctenidia"

Pulex irritans (Linnaeus, 1758), the human flea. This species (Fig. 78, A) is cosmopolitan in distribution and may be found frequently on many domesticated animals, notably swine. This is the flea most frequently observed in houses in many parts of the world. (In the eastern and southeastern United States, however, the cat flea or dog flea is more commonly found in houses.) P. irritans is interesting principally because of its close resemblance to Xenopsylla cheopis, the Indian plague-carrying rat flea. It has been shown experimentally to transmit plague and Eskey (1938) believes that it may be the principal vector of an un-
usual form of plague reported from the mountains of Ecuador.

*Xenopsylla cheopis* (Rothchild, 1909), the Indian or tropical rat flea (Fig. 78, B), is probably the most important flea in transmitting plague from rat to man, although several other species of *Xenopsylla* are found on rats in certain localities. It is at present the most prevalent rat flea in all parts of the tropics and sub-tropics and is not infrequently encountered in temperate regions that have considerable trade with tropical countries. Rats harboring this flea have become established at times in some of the cities along the Gulf of Mexico and the west coast of the United States. The original host of *X. cheopis* was probably the Indian black rat (*Rattus rattus rattus*), but now it is found also on the brown rat (*Rattus norvegicus norvegicus*) and the roof rat (*Rattus rattus alexandrinis*). *X. cheopis* (particularly from dead rats) readily attacks man as well as other animals.

*Tunga penetrans* (Linnaeus, 1758), chigoe flea or chigger. Unlike the females of most other fleas, the female of *T. penetrans*, after impregnation, bores its way into the skin (particularly of the toes or soles of the feet) and develops rapidly, often to the size of a small pea. (Length before impregnation is less than 1/20 of an inch.) The mature female usually contains 100 or more eggs. Clinically, the female flea appears as a small, black speck in the center of a pale, indurated skin area. This is, in reality, the slightly protruding terminal segment of the female from which the eggs are expelled.

The adult tunga flea may be readily distinguished from other fleas by the proportionately larger, pointed, fishlike head, the upper and lower borders of which are somewhat curved. In neglected sick natives, lesions produced by this flea may become most loathsome in appearance and jeopardize life. The removal of the fleas is accomplished by gradually enlarging the orifice until the globular egg-filled body can be squeezed out.

**FLEAS WITH ONE OR MORE COMBS OR “CTENIDIA”**

*Ctenocephalus canis* and *felis* (Curtis, 1826), the common dog flea and cat flea, have both genal and pronotal combs (Fig. 78, D).
They are cosmopolitan in distribution and so closely resemble one another as to have been considered by some authors as varieties of the same species. They are the fleas usually found in house infestations in the eastern and southeastern United States. Although capable of transmitting plague experimentally, it seems doubtful whether they do so in nature. As intermediate hosts, they may serve as transmitters of the tapeworms *H. diminuta* (page 211) and *D. caninum* (page 213).

*Ceratophyllus (Nosopsyllus) fasciatus* (Bosc, 1801), commonly called the European rat flea (Figs. 77, 78, C), is widely distributed in both Europe and North America and less widely in other parts of the world. It is regarded as a negligible factor in causation of natural outbreaks of plague, but appears from experimental evidence to be important as a natural reservoir for the virus of endemic typhus or Brill’s disease. It has only a pronotal ctenidium, with about 18 to 20 spines.

*Ctenopsyllus segnis* (Schönherr, 1816), the cosmopolitan mouse flea, is also commonly found on rats. It rarely bites man, and although regarded as a possible vector of plague, it is considered negligible in human outbreaks. It has small genal or cheek combs and a pronotal comb.

**Flea-Borne Diseases**

The reaction of human skin to ordinary bites of fleas is quite variable; in some persons there follows a reddish raised papular-like lesion, frequently edematous and at times induration and pustular formation follows. In case of *Tunga penetrans* (chigoe or chiggers), the female flea burrows into the skin, engorges itself with blood, and produces many eggs which escape through the barely visible orifice marked by its dark colored posterior. The sites generally attacked are the creases in the soles of the feet and the borders of the toe nails. In the tropics, natives may be infected with dozens of these fleas, and often secondary infection, abscess and ulceration follow. Their early removal by gradual enlargement of the opening and expression of the egg-filled female is an art in which some natives are quite adept.
In addition to serving as intermediate hosts and transmitters of the tapeworms *Hymenolepis diminuta* and *Dipylidium caninum* (Chapter IX) and possibly transmitters of tularemia among rabbits, the chief medical interest in fleas is their transmission of bubonic and septicemic plague, and of endemic typhus fever or Brill's disease. Although a résumé of the medical history of plague and endemic typhus fever is most tempting at this juncture, it has seemed advisable to refrain so as not to exceed the limits of permissible space.

Plague is primarily a disease of rodents, particularly of rats, and only secondarily of man. Outbreaks of plague are most always preceded by an epidemic among rats, characterized by a high mortality. Infected fleas migrate from the sick or dead rats in search of a fresh blood meal.* If man is successfully attacked, plague almost invariably follows in a few days. Among early symptoms are vague pains, malaise, and mental apathy, followed by headache, drowsiness, chills and fever which in severe cases is high and remittent in type. A variety of other symptoms may and often do occur which cannot be entered into here. Generally within twenty-four hours the characteristic "bubo" or buboes occurs in connection with glands draining the area in which the bite occurs (about 70 per cent in the groin). These vary considerably in size, and pain is often severe but in some cases may be insignificant. After a time the bubo softens, soon bursts if not incised, and discharges pus and slough.

In septicemic plague, enlargement of lymphatic glands is rarely apparent during life. The patient is prostrated from the beginning, indicating a high degree of virulence of the causative organism, *Pasteurella pestis*, and entrance of large numbers into the blood where they can be demonstrated during life. Pallor and

* The incidence of plague depends on the species of fleas rather than on their number. It has been found that *Xenopsylla cheopis*, the Indian rat flea, is three times more susceptible to *Pasteurella pestis* than many other fleas. When infected, its life is short, averaging about two weeks, whereas other fleas similarly infected may live for some months. This is attributed to the ready blockage of the proventriculus of this flea with clumps of the rapidly growing bacilli. The resultant inability of the insect to fill its mid-gut leads to the flea's wandering from host to host in search of blood, without ability to satisfy its hunger.
apathy are early signs, and there is generally little febrile reaction. Great weakness, stupor, and coma are prominent later manifestations terminating in death on the first, second, or third day. Pneumonic plague may arise as a complication of the septicemic form or from a previous pneumonic case through infected droplet transfer. The patient usually dies on the fourth or fifth day of this form of the disease.

Endemic or murine typhus (Brill's disease) is now generally considered to be a mild form of the classic epidemic typhus due to passage of the *Rickettsia prowazeki* through reservoir murine hosts. This disease is transmitted by *Ceratophyllus (Nosopsyllus) fasciatus*, the temperate zone rat flea; several species of *Xenopsylla* have been experimentally infected. It usually occurs in persons handling food which is open to contamination by rats, is relatively common in the Southern states (U.S.A.), and has been reported from many other parts of the world. An epidemic form known as "tabardillo" occurs in the region of Mexico City. The disease is frequently mild in its clinical manifestations, and follows in general the pattern of true epidemic typhus fever.

**PROPHYLAXIS OF FLEA-BORNE DISEASES**

Control of fleas on the domestic dog and cat, as well as on rat and man, requires strict hygienic and sanitary precautions in and about the house. This involves airing and sterilization of pillows, bedding, coverings of furniture, vacuum cleaning and scrubbing of floors with soapy water containing 3 per cent cresol or other suitable insecticide and removal of rubbish and dry organic matter which foster the growth of flea larvae. The domestic cat and dog, as well, should be subjected to periodic defestation through the use of pyrethrum powder, kerosene soapsuds, or a mixture consisting of 3 per cent cresol and 10 per cent kerosene kept well mixed with its water base. A mixture consisting of 2 ounces of washing soap to 1 quart of water and 2.5 pints of kerosene well emulsified and added to 5 gallons of water is said to give excellent results in this respect.

In a practical way prophylaxis regarding rat flea transmitters of
plague and endemic typhus requires effective destruction of rats harboring the disease-carrying fleas. Such measures as trapping of rats, rat-proofing of buildings, and cyanide fumigation expertly conducted and controlled are among the most helpful measures. Many countries require a strict quarantine against ships arriving from known infected ports. In California and elsewhere other specific effective measures have been devised to deal with situations in which rodents other than rats have been found to be widely infected with the plague bacillus.

THE MITES AND TICKS (CLASS ARACHNIDA, ORDER ACARINA)

Mites and ticks are generally classed under several superfamilies. In the order Acarina, unlike other orders of the Arachnida, the cephalothorax is fused with the abdomen. The class Insecta, to which practically all the arthropods thus far discussed belong, includes only arthropods with distinctly separate head, thorax, and abdomen.

Mites

In general all species of mites are small, many of them being barely visible but a few attain a length of 1/4 inch. The adults possess four pairs of legs, as do other arachnids including the ticks (insects have only three pairs of legs). The larvae, during the first stage, have only three pairs of legs, but after one or more molts, a fourth pair appears. The mouth-parts are varied, following in general the pattern of those of ticks (page 322).

Life Cycle of Mites. The life cycle of many species of mites is completed in less than a month; in some, it is as short as eight days. The female of the human itch mite or scabies mite deposits her eggs (10 to 25) during a period of about two weeks and then dies at the end of her burrow. The hexapod larvae hatch in from three to four days and mature in from ten to twelve days, during which time the area involved enlarges rapidly. An infestation with mites is often referred to as “acariasis.”

A large percentage of mites are free living, but, when parasitic,
there is every degree of parasitism, ranging from incidental to obligate. They attack both plants and animals and are aquatic as well as terrestrial. Some produce disease, while others are transmitters of disease.

**Classification and Description.** A clinically convenient grouping of mites will be employed here, but only a few of these categories warrant more than brief comments.

**ITCH, MANGE, AND SCABIES MITES (FAMILY SARCOPTIDAE)**

These acarine mites burrow in the skin, and the pregnant female deposits her eggs in the small tunnels thus produced. The many varieties of the most common genus, *Sarcoptes*, differ only slightly. A number of them may interchange hosts; for example, *Sarcoptes scabiei*, var. *suis*, which is normally parasitic on swine, often shifts to man for a limited time and is then reported as *S. scabiei* var. *hominis*. The adult female (Fig. 79) is about $\frac{1}{8}$ mm. in length and the male slightly more than half this size.

The sarcoptes mite attacks preferably the skin between the fingers, the bends of elbows and knees, the shoulder blades, breasts, and the inguinal region, producing an intolerable itching caused
by its waste products. The minute tunnels (Fig. 80) resulting from burrowing in the epidermis may reach 3 cm. in length, and are associated with formation of small vesicles and papules.

Scratching causes oozing of serum, bleeding, and infection, conducive to further spread of the mites. Transmission is by direct contact, such as handshaking, exchange of infested towels, clothing, and bedding.

The essentials of treatment are as follows: (1) Thorough softening of the skin by massage with tincture of green soap and hot water. (2) Application of sulphur ointment (5 per cent in lanolin) at intervals of three to four days. (3) Thorough boiling, steam sterilizing, or baking of all linen coming in contact with the lesions.

**Scab mites (genus *Psoroptes*)**

Scab mites also belong to the family **Sarcoptidae**. They resemble the scabies mites, but have longer legs and more slender bodies. They do not burrow, but live at the bases of hairs, injuring the skin and causing an exudate, which partially hardens and piles up on the surface as a moist, loose crust, known as "scab" and some-
times also as scabies. The eggs, although fragile, are protected in these crusts, and the mite is thus able to withstand surprisingly adverse conditions. Transfer to other animals by contact is quite common. Sheep, cattle, and horses are mainly affected; man rarely.

**Follicle Mites (Family Demodicidae)**

Follicle mites (Fig. 81) inhabit the hair follicles and sebaceous glands of mammals, often causing areas of inflammation. In man, hair follicle lesions, known as blackheads or "comedones," may be associated with the presence of *Demodex folliculorum* (Simon). Opinions differ, however, as to its direct causal relationship to comedone formation in man, and also to its distribution in North America. It has been claimed that acne rosacea may in some way be associated with the presence of hair follicle mites. Follicular mange, a common affliction of the dog, is caused by one of these mites. Treatment is often difficult because of the deep-seated character of the lesions.

![Figure 81. Hair follicle mite, Demodex folliculorum, X 350 app. (After Mignon. In Chandler: Introduction to Parasitology. Edition 6. Courtesy of John Wiley & Sons).](image)

**Harvest Mites or "Chiggers" (Family Trombiculidae)**

Harvest mites, associated in this family with the largest of mites, are small and some of them brightly colored. *Trombicula irritans* (Riley), which attacks man, is barely visible, and were it not for the red color, its presence as causative agent of the irritation would rarely be suspected. This is the feared "bête rouge" of Mexico, Central, and South America.

The larvae attach themselves to the skin by means of their hooked chelicerae, causing severe dermatitis with intolerable itching. Red blotches appear first, followed after some hours by urticarial wheals. In temperate climates, persons usually acquire
chigger infestations during late summer and early autumn while walking through weeds and shrubbery where the mites abound.

*Trombicula akamushi* (Brumpt) in its larval form (0.4 mm. long) transmits a frequently fatal oriental disease, which symptomatically resembles typhus fever. This disease, known as Japanese river fever (tsutsugamuchi disease), is probably caused by a rickettsia. Since the mature mites, as well as the nymphs, are vegetable feeders, the infection is transmitted only by the parasitic larvae, which have the infection passed on to them through the egg.

**CHEESE, GRAIN, RAT, AND WEB-SPINNING MITES (BELONGING TO VARIOUS OTHER FAMILIES OF MITES)**

The small, so-called cheese mites are commonly found on various dry foods and similar substances. They are about 0.5 mm. or less in length and develop very rapidly. It is said that before the nymphs become fully mature, they are able to attach themselves to other insects for purpose of transport to fresh sources of food supply. Persons handling infested products may suffer from a temporary dermatitis often referred to as “grocer’s itch.”

The ordinary grain or straw itch-mite (*Pediculoides ventricosus*) is a predaceous louselike mite which attacks the larvae of a variety of insects. Although normally considered a beneficial mite, it sometimes attacks man, producing a very annoying dermatitis. Epidemics of dermatitis have been traced to these mites, the infection having been contracted through sleeping on infested straw mattresses or while working in grain fields at harvest time.

The tropical rat mite of the genus *Liponyssus* has been reported from many parts of the world as irritating to man. It is said to be widely distributed in the southern United States. Man experiences, at the time he is bitten, itching and some degree of pain, the latter sometimes continuing for several hours. In addition to rat control, Herms (1939) has recommended for prevention, rubbing furniture, woodwork, and so on, with kerosene. The common poultry or roost mites, often a serious pest in many parts of the world, belong to a closely related genus.
The web-spinning mites infest fruit trees and other vegetation. They are comparatively small and frequently reddish in color, hence the popular appellation “red spiders.” Persons engaged in harvesting certain crops, such as hops and almonds, often complain of itching purported to be produced by these red mites.

**Ticks (Superfamily Ixodoidea)**

Ticks, members of the order Acarina, differ primarily from insects in that the adults have four pairs of legs, a fused head, thorax and abdomen, only two pairs of mouth parts, and no antennae. They are distinguished from the mites and other acarines by a median, probe-shaped, anchoring organ (the dart or hypostome) beset with numerous backwardly projecting barbs, and by a pair of stigmal plates.

**The Mouth Parts.** The two piercing chitinous chelicerae which lie above or dorsally to the solitary hypostome and the hypostome itself arise directly from the so-called basis capituli. Serving as protectors for the sharp chelicerae and accessory steadying anchors to the hypostome, there are two modified palps, called pedipalps. The mouth consists of a slitlike opening between the bases of the chelicerae and hypostome. Together, these parts are known as the “capitulum” or rostrum. On reaching its intended host, the tick, employing the chelicerae, tears open the skin and forces the barbed hypostome deeply into the subcutaneous tissues until the chelicerae and the hypostome are completely buried and anchorage secure.

**Distinguishing Features.** Recognition of ticks that are medically significant is important and fortunately not difficult. There are two families, known as the Ixodidae and the Argasidae. They are differentiated on the basis of the presence or absence of a chitinous back-shield or “scutum.”

**The Ixodidae (Ticks with Scutum)**

These commonly called “hard ticks” have mouth-parts which project in front of the body when viewed dorsally (Fig. 82, r, 2, 5, 5'). The stigmal plates are posterior to the fourth pair
of legs, the skin is finely striated, and the anus lies behind the middle of the ventral surface. In the male the scutum is well developed. The female possesses depressed so-called "porose areas"

(probably sensory) on the dorsal surface of the basis capituli. Sexual dimorphism is marked.

Most animal ticks belong to this family. There are two main groups, determined principally on the absence or presence of marginal "festoons." They are subdivided into nine genera, based on the character of pedipalps, capitulum, ornamentation, presence or absence of eyes, and anal grooves in relation to anus. While species of a majority of these genera transmit piroplasma,
rickettsia, tularemia, and other parasitic diseases in animals, several species of the genus *Dermacentor* are the only ones of much importance in so far as human disease is concerned. These are usually ornate ticks, with eyes and marginal festoons. The *basis capituli* is rectangular when viewed dorsally, and the pedipalps are usually short and broad. The following brief description of the life cycle of *D. andersoni* will serve as a basis for discussion of specifically related disease.

**Dermacentor andersoni** and Rocky Mountain Spotted Fever. *D. andersoni*, called the "wood tick," occurs in northwestern United States and British Columbia, where it is the vector of Rocky Mountain spotted fever. It may transmit tularemia also, particularly among rodents, and may cause a tick fever. The adult ticks feed principally on large animals such as sheep, cattle, horses, and game animals (Fig. 83), but the larvae and nymphs prefer to feed on smaller animals, such as woodchucks and pine squirrels. All forms may feed on medium sized animals, notably jack rabbits.

**Life Cycle of D. andersoni.** Copulation of adults occurs on the host and when fully fed, the greatly distended female drops to the ground. In about a week, egg laying begins and continues over a period of about three weeks. Five thousand eggs (Fig. 84, A), or even more, may be laid by a single female, and if un-
disturbed, she will pile these up ahead of her in one large mass. After about thirty-five days' incubation, the young hexapod "seed tick" emerges (Fig. 84, B), and upon finding a suitable

Rodent host, feeds for three to five days. It then drops off, moults in from six to twenty-one days, and emerges as a nymph with eight legs (Fig. 84, C).

Nymphs which are usually the progeny of over-winter adults, generally hibernate until the following spring, when they seek a larger sized host to which they attach themselves for a period of from four to nine days. When fully engorged, these nymphs drop off, moult once, and in twelve to sixteen days moult again, finally emerging as adult ticks (Fig. 82, 5, 5'). Some of these adults may find hosts before the end of summer, but, as Cooley (1932) explains, the general development of drought usually necessitates their lying dormant under shelter until the following spring. Since it is usually only the adult tick which bites man, the danger from this source is confined to a few months, from early spring to about July 1. The life span between successive egg layings is about two years.

**Causative Organism of Rocky Mountain Spotted Fever.** Rocky Mountain spotted fever results from infection of man with the
very small "rickettsia bodies" known as *Dermacentronexus ricketttsi.* The usual transmitter in endemic areas has been found to be *D. andersoni.* The region of greatest virulence of the disease is the Bitter Root Valley of western Montana, the severity being considerably less in adjoining and nearby states and in British Columbia (in Montana 75 to 90 per cent mortality has been reported and in Idaho only about 5 per cent).

Since 1931, this disease (usually in mild form) has also been reported from numerous central and eastern states of the United States, being found most prevalent in parts of Maryland, Virginia, and North Carolina. Two interesting points in this connection are the greater prevalence among women and children and the fact that the vector is *Dermacentor variabilis,* a common eastern dog tick.

Transmission among rodents in nature is through the bite of the nymphs of *D. andersoni* and also of the common rabbit tick (*genus Haemaphysalis*), which is said to harbor regularly a mild type of the virus. Both males and females of *D. andersoni* may pass on the virus to offsprings through the eggs, and moultings of larvae do not prevent such transmission. Virus appears to be inactivated in the tick during periods of fasting or hibernating, but is reactivated after a blood meal.

**Symptomatology and Pathology of Rocky Mountain Spotted Fever.** The incubation period of Rocky Mountain spotted fever is from two to five days in severe cases, and from three to fourteen days in mild cases. The prodromal symptoms are malaise, headache, pain in the joints, and chills which decrease in severity throughout the attack. By the second day the temperature has usually risen to 103° or 104° F.; by the fifth day, to 105° F. Subsequently it may rise even higher and has a tendency to remit. On the fourth to the seventh day of the disease a characteristic measles-like rash appears on wrists, ankles, and back, and extends rapidly to trunk, scalp, hands, and feet. These lesions often assume a

* The various rickettsia transmitted by lice, fleas, and mites, as well as ticks, have been grouped as species of the genus *Rickettsia* (da Roche-Lima). Consequently *Dermacentronexus ricketttsi* may be more properly referred to as *Rickettsia rickettsi* (Wolbach).
petechial character and may become more or less confluent, especially on dependent parts. A certain amount of icteric coloring of the skin and sclera is also usually noted. During the third week, in the average case which recovers, the eruption begins to fade as the fever subsides, and desquamation begins.

In this disease the rickettsia bodies seem to parasitize particularly the endothelium of blood vessels. While the liver is usually only slightly enlarged, the spleen is early palpable and later greatly enlarged and tender. Constipation and kidney involvement with albuminuria are usual. If the patient survives the second week of the disease, prognosis is good and recovery generally follows. The degree of immunity produced appears to be in proportion to the severity of the infection.

Laboratory Diagnosis of Rocky Mountain Spotted Fever. In addition to presumptive symptomatic diagnosis of this disease and differentiation from other conditions presenting certain similar signs and symptoms such as measles, typhoid fever, typhus-like fevers, and cerebrospinal meningitis, there are available laboratory procedures as follows:

1. The well known Weil-Felix agglutination reaction, originally applied to diagnosis of typhus fever, which consists in testing the agglutinating power of the patient's serum (diluted to 1:300 or more) against strains of the bacillus *Proteus vulgaris*, the most commonly used ones of which are designated as OX$_2$, OX$_{19}$, OXK, and OXR. Comparative titers of serums from Rocky Mountain spotted fever and various other *Rickettsia* diseases assembled from different sources are shown below in summary:

<table>
<thead>
<tr>
<th>Disease</th>
<th>OX$_2$</th>
<th>OX$_{19}$</th>
<th>OXK</th>
<th>OXR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidemic typhus (louse-borne)</td>
<td>±</td>
<td>+</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>Endemic typhus (flea-borne)</td>
<td>±</td>
<td>+</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>Brill's disease (flea-borne)</td>
<td>±</td>
<td>+</td>
<td>-</td>
<td>±</td>
</tr>
<tr>
<td>Rocky Mountain spotted fever (tick-borne)</td>
<td>+*</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Tsutsugamushi fever (mite-borne)</td>
<td>±</td>
<td>±</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Q-fever (tick-borne)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Trench fever (louse-borne?)</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>?</td>
</tr>
</tbody>
</table>

*All agglutinations are either low or tend to be higher in the OX$_2$ group.*
diverse localities throughout the world may perhaps be epitomized in four main groups known as:

<table>
<thead>
<tr>
<th>Typhus Group</th>
<th>R. S. F. Group</th>
<th>Tsutsugamushi Group</th>
<th>Q-Fever Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidemic or European typhus</td>
<td>Rocky Mountain spotted fever</td>
<td>Tsutsugamushi fever</td>
<td>Australian Q-fever</td>
</tr>
<tr>
<td>Endemic or murine typhus</td>
<td>Brazil spotted fever</td>
<td>Rural typhus of Malaya</td>
<td>American Q-fever</td>
</tr>
<tr>
<td>Brill's disease (Tabardillo)</td>
<td>Tobia fever of Colombia</td>
<td>Mite fever of Sumatra</td>
<td></td>
</tr>
<tr>
<td>Fièvre nautique (Toulon)</td>
<td>Fièvre boutonneuse</td>
<td>Tropical or scrub typhus</td>
<td></td>
</tr>
<tr>
<td>Manchurian typhus</td>
<td>Kenya typhus</td>
<td>Queensland coastal fever</td>
<td></td>
</tr>
<tr>
<td>Urban typhus of Malaya</td>
<td>S. African tick-bite fever</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. Injection of infected blood into guinea pigs causes swelling of the testes and scrotum as in endemic typhus fever. Typical lesions consist of endothelial cells of the tunica vaginalis packed with *Rickettsia* organisms. Usually only fever without local scrotal lesions is produced by injecting blood of cases of classic epidemic typhus. This scrotal lesion is known as the Neill-Mooser reaction; it is also reported to occur to some degree in other less well known *Rickettsia* diseases summarized above.

**Prophylaxis of Rocky Mountain Spotted Fever.** Prevention of Rocky Mountain spotted fever through control of ticks over wide endemic areas has been rather discouraging, according to Parker (1933). However, encouraging results in control have been accomplished by dipping, and by rodent and grazing control. So far, attempts to parasitize ticks in endemic areas have been of doubtful value.

Prophylactic vaccination, according to Parker, confers a fairly high degree of immunity for from six to twelve months. It is thought that the use of this procedure in highly endemic areas might materially reduce the mortality.

The avoidance of tick bites by keeping away, if possible, from endemic tick-infested districts during spring and early summer is most important. Where circumstances and conditions are such that exposure to tick bite is likely, one should wear appropriate clothing, such as socks outside the trouser legs, leggings, puttees,
or, better still, high boots and like protection for arms, head, face, and neck. All ticks on the body or clothing should be promptly removed.

THE ARGASIDAE (TICKS WITHOUT SCUTUM)

The Argasidae or "soft ticks" have rough skins. The head is invisible when viewed dorsally, the stigmal plates lie between the third and fourth pairs of legs, and the anus is situated in the mid-line, nearer the middle of the ventrum than the posterior end. These ticks are chiefly parasites of birds, bats, and other animals, and only occasionally of man. They live and breed in the nest or lair of their hosts, thus having ready access to them. There are two genera of medical importance to man:

Genus Ornithodorus: ex. Ornithodorus moubata. This tick (Fig. 82, 3) is often referred to as the human relapsing fever tick of Africa. Its body is oval with rounded margins, and the mature adult is about 12 mm. long. The skin is irregularly studded with tubercles and the capitulum is even with the anterior margin of the body, the ends of the palpi projecting slightly.

O. moubata has no eyes. Both sexes bite man, often more than an hour being required for full engorgement with blood. The female lays 50 to 100 eggs in batches. The nymphs bite and feed several times before maturing, and the adults bite repeatedly.

Genus Argas: ex. Argas persicus. This common fowl tick (Fig. 82, 4) transmits spirillosis of fowls and has been suspected of transmitting a relapsing fever of man in Persia. The adult female is obovate, from 6 to 8 mm. long, and the thin marginal border is distinct even when fully engorged.

A. persicus, like O. moubata, has no eyes and the capitulum lies some distance behind the anterior margin of the body. Both nymphs and adults are very active at night, traveling considerable distances to find hosts.

Ornithodorus moubata and Relapsing Fever. O. moubata transmits the spirochaete, Borellia duttoni, causative organism of "African relapsing fever." It has been shown also that various other species of this genus in Asia, Mexico, Central and South
America, and some western states of the United States may transmit closely related spirochetes.

The organisms are found distributed generally throughout the tissues of the tick (nymph and adult) and the infection may be conveyed directly through saliva at time of biting, and indirectly through rubbing in the feces or coxal fluid.*

Experimentally, Feng and Chung (1939) reported that "shortly after spirochaetes are ingested by the ticks, they penetrate the stomach wall and reach the body cavity. From the body cavity the spirochaetes invade the salivary glands, the coxal glands, the reservoirs (blood) and the nerve ganglia."

**ABERRANT ACARINES**

Aberrant Acarines comprise the so-called "tongue worms" and related species, which are elongate, legless, more or less annulated wormlike parasites. The mouth is provided with a chitinous protective structure and four rudimentary clawlike appendages. The adults are endoparasites primarily of the air passages and body cavities of vertebrates. In a few instances, man has been reported as accidental secondary host of several species, namely, *Linguatula serrata* (Frölich) in Egypt and west Africa, and *Armillifer (Poroccephalus)* species in Africa and the Far East.

*This is an excretory product secreted by the coxal glands opening on the coxae (the broadly sessile basal segments of the legs).*
PART FOUR

POISONOUS AND VENOMOUS FORMS
CHAPTER XV

POISONOUS AND URTICARIAL ARTHROPODS

In addition to their role in the transmission of disease through the medium of their bites, insects and various other biting and stinging arthropods may give rise to both allergic and toxic manifestations. These will be discussed briefly in this chapter. Further information may be found in books dealing more extensively with this subject, some of which are listed in the Bibliography.

INSECTS (INSECTA) AS PURELY POISONOUS OR URTICARIAL AGENTS

It is a notable fact that insects such as most salt-marsh mosquitoes and the stablefly, whose bites are very painful, are seldom intermediate hosts of parasitic diseases. Herm (1939') suggests that in order to become a successful vector of disease it may be necessary for the arthropod to modify the severity of its bite. Most disease-transmitting insects pierce the skin for the purpose of sucking blood, and their salivary secretion seems to enable them to minimize, temporarily at least, the local painful effects caused by the bite per se. In some instances, however, a venom, usually of salivary origin, is also injected, which may give rise to serious systemic manifestations particularly if the bites are numerous.

The reactions of individuals to bites of the common bedbug and the fleas vary markedly. Some persons seem to suffer no ill-effects whatever, not even the usual swelling and local inflammation; others react noticeably, even violently, to one bite. Some authors believe that this manifestation is allergic in origin, and that when a tolerance is developed, immunity may have been established. There are two groups of bugs noted particularly for
the painfulness and local inflammatory effects of their bites: (1) the "cone noses" or "kissing bugs," to which belong the blood-sucking Triatomes. (2) The biting water bugs, some of which are the largest of bugs, measuring as much as 2.5 inches in length. They are popularly known as "electric light bugs," since they are most often found in the vicinity of strong light.

Certain sap-sucking insects (known as thrips) and plant-eating bugs, particularly under the urge of hunger, have been reported to resort to blood-sucking when opportunity is presented. In this connection Usinger (1939) remarks that "although the transition from sucking plant juices to sucking blood appears to be great, chemically it has been found that the same elements occur in both and often in quite similar combinations, but in different proportions."

ANTS, BEES, WASPS, AND YELLOW JACKETS
(ORDER HYMENOPTERA)

Ants, bees, wasps, and yellow jackets constitute the true stinging insects. The females are generally provided with a specialized ovipositor, known as the "sting," which may be employed both offensively and defensively.

ANTS

The sting of ants in temperate climates is soon forgotten, but in warmer countries certain species of "stinging ants" are very aggressive and may inflict wounds which are exceedingly painful. Because of their number, they may even endanger the life of their victim.* Among the more important species of stinging ants in the Western Hemisphere are: (a) Texas harvester or agricultural ant; (b) California harvester ant; (c) Mexican or California fire ant; (d) Central and South American high tropical rain-forest ants, which are said to swarm out of their nests in great numbers when disturbed, and rush for the intruder.

* The most vicious ants, however, are not stinging ants but biting ones, the best known of which are the "army ants" of the tropics. Many of them live in and among dead twigs and are especially ferocious, swarming out in a steady stream like flowing water when disturbed.
Bees

The stinging apparatus of the honeybee (representative of this group) consists of three parts: (a) the sheath, (b) the sharp, barbed, lancet-like darts with accessory parts, (c) the venom sac containing three glands, two of which secrete formic acid and the other a fluid which is alkaline in reaction. It is the combination of these two secretions at the time of stinging which causes the death of the attacked insect, or the excruciating pain and other reactions in man.

When provoked to sting, the females of the honeybee and also of certain wasps leave in the skin the posterior tip of the abdomen, together with the sting apparatus. As the muscles of this mechanism continue to function, they force the distally barbed sheath and the supporting lancet-like darts deeper into the wound. Through co-ordination of the various parts of this unique piece of apparatus, the mixed acid and alkaline gland secretions are carried to the depth of the wound produced.

Wasp

The term “wasp” generally suggests the thread-waisted wasps known also as “mud daubers.” These usually make their small nests of mud, and stock them with other arthropods such as insects and spiders to serve as food for the larvae. However, the so-called “yellow jackets” (sometimes called hornets), which make paper nests, are in reality large wasps. They will be separately described in the following section.

Among the wasps, there is one family whose members are commonly known as “velvet ants,” or “cow killers.” The stings of these insects are so annoying to man at times that they warrant brief mention. These wasps are parasites of bees and other wasps, some of the commoner forms measuring ½ to 1 inch in length. One species is reported to occur frequently on the sand beaches of Lake Erie, where they cause barefoot bathers considerable annoyance and distress.

In California there has been reported a very small wasp (barely
5 mm. long), which is said to invade houses after a warm spell during autumn. The sting, although much less intense than that of the honeybee, apparently gives rise to troublesome systemic reaction, particularly in children.

**YELLOW JACKETS (HORNETS)**

Yellow jackets build their nests of pulp made of masticated wood fiber, and often suspend them from limbs of trees; whereas most other wasps employ mud for nest building. The sting of these **HYMENOPTERA** is often exceedingly painful. Although there may be considerable local swelling and other associated objective signs, the systemic disturbances from one or two stings, if they occur, are not likely to be serious. However, a considerable number of stings at the same time, may give rise to alarming symptoms, as in the case of bee sting, notwithstanding the fact that hornets withdraw the stinging parts.

*Polistes gallicus*, a common paper-making wasp, has been shown to be an efficient distributor of ascaris, hookworm, and whipworm eggs. The genus *Polistes*, characterized by nests built widely open below, has a wide distribution. The so-called white-faced hornet is the largest species native to the United States.

**EFFECTS OF STINGS OF HYMENOPTERA**

Frequently, the reaction from the sting of **HYMENOPTERA** is only a mild inflammation, which disappears in a few hours. At other times, however, the part involved may become greatly inflamed and edematous, accompanied by appreciable or even profound systemic symptoms, due, it is thought, to a viper-like venom. Involvement of the conjunctivae is particularly dangerous. Some persons are especially sensitive to bee sting, and appear never to develop an immunity.

**TREATMENT AND PREVENTION OF STINGS OF HYMENOPTERA**

An approved general method of treating stings of **HYMENOPTERA** is the application of a warm pack. For stings of great severity, the use of a tourniquet above site of the sting is recommended when
practicable. The local application of a sodium bicarbonate paste to the sting wound may prove to be soothing.

Persons seriously affected should be encouraged to drink fluids which are alkaline in reaction, and to remain quiet. Aspirin or other drugs may be administered to relieve pain. Often the effect of a sting or bite is greatly augmented by the excitability and nervousness of the victim; in such cases sedatives are helpful.

Prevention of the effects of the stings of HYMENOPTERA is largely a matter of judicious avoidance, or of wearing appropriate clothing to prevent the insects from coming in contact with the skin.

SPIDERS (ORDER ARANEIDA)

Usually, spiders secrete a venom with which they are able to poison other arthropods and the small animals on which they feed. Only in rare instances, however, is spider venom very toxic to man. Most of the beautiful "orb-weavers" or garden spiders, as well as the large, dark, hairy, ferocious-looking tarantulas, are not particularly dangerous, although bites of the latter may at times be somewhat painful. Even large species of the "tarantula" group known as "bird spiders," although capable of killing small animals, are apparently not able to injure man seriously. The poison glands consist of a pair of sac-like structures situated in the anterior part of the cephalothorax. The venom is discharged through a duct opening at the tip of each cheliceral fang.

BLACK WIDOW SPIDER

A spider which is definitely harmful to man is Latrodectus mactans, commonly known as the "black widow," hourglass, or shoe button spider (Fig. 85). It has been reported from most parts of the United States, southern Canada, Mexico, and the West Indies, south to Peru and Chile. Collections have been made at elevations as high as 8000 feet in Colorado. Ordinarily, the black widow spider is found, together with her web and egg

* The true tarantula is a relatively small European spider. This name is, however, used for any large, tropical or subtropical spider. Most of these are harmless but some are dangerous, though not deadly.
sacs, in dark, sheltered places, such as abandoned rodent burrows, hollow stumps, long grass, brush piles, and under rocks and logs. It is often present about man-made structures, most frequently

![Figure 85](image)

**Fig. 85.** *Latrodectus mactans* (the black-widow spider). Mature female with egg sac (× 1.5 approx.); eggs, first instar spiders on right of egg sac; second and third instar spiders on left, below; fourth instar on right, below. (From Herms, Medical Entomology, Edition 3. Courtesy of the Macmillan Company.)

in cellars, outdoor privies, hen houses, pump houses, barns, and garages.

Although this spider is found throughout the year, it is relatively more abundant in late summer and early autumn. By this time, many females have matured, and some of the previous year's survivals are still in evidence. The female, as a rule, is rather
docile unless provoked to attack, very hungry, or guarding the egg sac. The mature males live only a few weeks and are likely, in the event of food shortage, to be commandeered for food by the female. After spinning a rather coarse web in a suitable location, the female spends the balance of her life in feeding on insects and other forms of life which are caught in her web, and in guarding her several egg sacs.

The adult female is shiny black to sepia brown in color, with a distinctive yellow to red hourglass marking on the ventrum (under surface) of the abdomen. Some variation in this pattern may occur, and other similarly colored markings (sometimes there are also spots on the dorsum) are still less constant. The abdomen is globose, often being likened to a shoe button, and is about 6 to 8 mm. in diameter (in gravid females it often measures 9 by 13 mm.). The over-all length (legs extended) is about 3 to 4 cm.

The whitish eggs, about 1 mm. in diameter, are deposited in a globular cocoon-like sac (about 1 to 1.5 cm. in diameter) which is spun during the summer. After about twenty days’ incubation, the small spiderlings emerge from their shells, but remain within the cocoon for a time, where they undergo one or more molts before exit into the outer world. After emerging from the cocoon, they undergo further development and moulting, preparatory to attaining sexual maturity; this period lasts for about four months under average summer conditions.

Symptomatology Following the Bite. At the site of the bite of the black widow spider, there may be little visible evidence of a lesion except slight local swelling and perhaps two small red dots where the venom-carrying pedipalps entered the skin. However, pain is felt almost immediately and increases rapidly in intensity. It reaches a maximum in one to three hours and gradually subsides in twelve to forty-eight hours.

A striking sign of venom absorption is muscular rigidity or spasm, particularly of the abdominal muscles; the abdomen presents a boardlike tightness. Commonly accompanying signs and symptoms are a slight rise in temperature, increased blood pres-
sure with profuse perspiration, and a tendency to nausea. Other symptoms which may occur are chills, urinary retention, constipation, hyperactive reflexes, burning skin sensations, and priapism.

**Treatment of Bite.** Application of heat and induction of sweating are procedures that have long been used in treating bites of *L. mactans*, as well as those of other spiders. Sweating is a remedy still highly valued by the natives of Madagascar. In Europe, during the Middle Ages, resort to dancing as treatment for spider bite may have been indirectly beneficial in bona fide cases by promoting elimination of the absorbed venom through sweating.

In recent years sedatives and hot baths have been extensively employed. The effect of morphine appears to be beneficial, but alcohol and strychnine, which were formerly given, are definitely contraindicated. Potent specific antiserum, when available, are of great value. The prompt use of a tourniquet, when practicable, may aid in minimizing the amount of poison absorbed.

Frawley and Ginsburg (1935) caution that, while prognosis is ordinarily favorable, incorrect diagnosis and improper treatment in individual cases are hazardous. In particular, they counsel against the administration of alcohol in any form. They further conclude that “the most rational form of treatment is one based on the clinical picture, which resembles hypertension in the adult and eclampsia in the child; viz., intravenous administration of magnesium sulphate.” This, it is believed, relieves the abdominal cramps and other severe symptoms following an effective bite by a black widow spider. The recommended routine treatment is as follows:

1. The patient is immediately put to bed, and iodine is applied to the site of the bite.**

2. A soapsuds enema is administered, and fluids (nonalcoholic) are given freely by mouth.

* The origin of this was undoubtedly the Tarantula dance—a pagan festival—celebrated in parts of Italy. The bite of this *Tarantula* is not serious; the venom is weak and apparently small in quantity.

** According to Curran (1937) the Aztecs did not regard the spider bite as deadly, but recommended complete rest and the drinking of a strong alkaline solution with the application of alkaline compresses. This differs little from the present-day treatment and should be recommended to the patient pending the arrival of the physician.
3. Morphine sulphate is given hypodermically to control the pain, and sodium amytal to insure rest.

4. Magnesium sulphate, 20 cc. ampule of a 10 per cent solution, is given intravenously, and the dose may be repeated as required to overcome the hypertension and spasticity of the muscles. Frawley and Ginsburg (1935) found that with one dose of magnesium sulphate, the patients were commonly free of symptoms within twenty-four hours.

Prophylaxis. Control measures and prevention must be based on knowledge of the life cycle and special habits of the offending arthropod. In this connection, it should be borne in mind that these spiders are more likely to frequent human habitations during droughts, in search of moisture. Great care must be exercised to avoid contact with them. Adults as well as children should be cautioned about their presence in outdoor privies, old lumber piles, and similar places.

TICKS

Ticks as Purely Poisonous or Urticarial Agents

Members of both families of ticks (Ixodidae and Argasidae) produce local or systemic disturbances by their bites alone, although species of the Ixodidae are more commonly responsible for such disturbances. Ordinarily ixodine ticks, such as Dermacentor occidentalis or variabilis, may remain attached to their victim for days without causing serious inconvenience. However, Ixodes ricinus frequently causes more or less systemic disturbance.

Tick Paralyses and Tick Fever

Ixodes holocyclus causes paralysis in sheep, calves, and dogs in Australia. It is believed that the bite of Dermacentor andersoni (the tick transmitter of Rocky Mountain spotted fever) may cause paralysis and fever in man in the Rocky Mountains. This, known as “Colorado tick fever,” is a disease of remittant type and of uncertain etiology. Colorado tick fever is probably also caused by the bites of other ticks.
Tick paralysis of an acute ascending type, occurring in children and in sheep in northwestern United States and western Canada, has been traced to *D. andersoni* (Fig. 82, 5, 5'). It has been shown experimentally that a similar type of paralysis may be produced in the dog. The toxic substance appears to be introduced only when the tick begins engorging after it has been attached for several days.

According to a recent publication by Mail (1940), many cases (most of whom were children) of *D. andersoni* paralysis have occurred in British Columbia and some of the adjacent provinces. He states:

"The patient, perfectly well one day, may on the next complain of a numbness in the feet and legs and have difficulty in walking; a little later it may be impossible to stand up. The hands and arms are usually affected next. Often there is a partial paralysis of the throat muscles, there being difficulty in swallowing; the tongue, too, may be affected with inability to speak properly. Other constitutional symptoms are slight; there may be some restlessness in the early stages and the patient may complain of not feeling well. There is no pain, usually no fever, but the pulse is faster than normal."

Generally it may be said that paralysis is confined to the nerves governing movement, and does not affect the special senses. The time, from the beginning of the symptoms to complete paralysis and even death, may be less than two days, but it is usually from three to five days. The chances of recovery are good if the organs of respiration or the heart are not affected.

Medical aid should be secured if possible. A search should be made immediately for other ticks, especially around the nape of the neck, in the hair, and on the back and chest. When found, the ticks should be removed as described under Prophylaxis (page 343). If the patient is likely to recover, he should show signs of this within a few hours after removal of the tick. In any event medical advice should be secured concerning further treatment.

Among the argasid ticks, certain species of the genus *Ornitho-
Poisonous and Urticarial Arthropods

Dorud are known to cause more or less serious symptoms through their bites. The bite itself of O. moubata (Fig. 82, 3) is very painful. The surrounding area swells rapidly and there is an accompanying troublesome irritation which does not subside for days. Young ticks or nymphs are more dreaded by natives than are more mature ones.

Ornithodorus coriacus, likely to be encountered in heavy underbrush on coastal hill or mountain side in northern Mexico and California, has a considerable local reputation for the severity of its bite. It is a very large tick, 8 to 10 mm. in length when not engorged. In Mexico it is called "pajarollo." A personal account of two bites by ticks of this species, given by Chandler (1939), indicates that the bite may cause considerable distress and inconvenience, but that it probably does not result in serious consequences if promptly and effectively treated.

Treatment of Tick-Bite Paralysis

In treating tick bite locally, a good practice is to bathe the wound in very hot water, then apply a strong solution of bicarbonate of soda, which should be allowed to dry on the skin. For severe itching, the lesions may be covered with vaseline ointment containing camphor or menthol. If the symptoms are not shortly relieved by this local treatment, or if complications arise, medical aid should, of course, be summoned.

Prophylaxis of Tick-Bite Paralysis

In conformity with our knowledge of the habits of D. andersoni, the most practical way to avoid infestation is not to walk through undergrowth, high grass, and the like, during the season when this tick is active (from end of March to mid-June); unfortunately this is not always practicable. A degree of protection may be afforded, however, by wearing clothing designed to exclude ticks, such as high boots with trousers tucked into them, closely fitting collar, and shirt well tucked in around the waist.

Upon retiring in the evenings, the entire body should be examined carefully for the presence of ticks. As Mail (1940) aptly points out: "It should be remembered that while the spine, the
nape of the neck and the head are the favourite sites of attachment, ticks may attach themselves anywhere on the body."

Other pertinent points in connection with both treatment and prophylaxis are as follows:

1. In attempting manual removal of ticks, it is important to avoid leaving the head embedded.

2. If the tick is found before it is too firmly attached, removal may be accomplished by gently but firmly pulling it down and away from the point of attachment. In no case should jerking be resorted to.

3. When swelling around the firmly attached tick is considerable, the skin area next to the tick should be appropriately lanced (observing usual sterile precaution), the tick and mouthparts completely removed, and the wound disinfected and dressed as any small open wound.

CENTIPEDES, MILLIPEDES, AND SCORPIONS

CENTIPEDES AND MILLIPEDES

Centipedes (Fig. 86, A), briefly described, have somewhat flattened bodies, consisting of a distinct head, with long, many-jointed antennae, and about fifteen to twenty segments to each of which is attached a single pair of jointed legs. Millipedes (Fig. 86, B), on the other hand, have a more cylindrical body and, with the exception of the most anterior segments, each segment bears two pairs of legs. Neither have nearly so many feet or legs as their names would seem to indicate. They belong to the orders CHILOPODA and DIPLOPODA, respectively.

Centipedes are ordinarily predaceous, feeding mainly on insects. In spite of the abundance of walking appendages, they are able to crawl very rapidly. Cases have been reported in which a double, reddish streak was produced on the skin where this arthropod had crawled, due to the dragging of the tips of the terminal pair of legs.

Millipedes (order DIPLOPODA) possess no organs of defense other than glands which give off an offensive odor. The head is equipped
with antennae, chelicerae, and one pair of maxillae. The chief medical interest in millipedes lies in the fact that some species have been found in the human alimentary and urinary tracts.

It is generally believed that millipedes are harmless, and it seems probable that those found in the human body entered through the external orifices.

Centipedes are provided with powerful poison claws, ventrolateral to the mouth. The venom of the centipede is only a small part of the total salivary secretion, the function of which is primarily digestive. The small centipedes found in temperate climates rarely cause more than local symptoms, but large tropical centipedes, such as *Scolopendra gigantea* (which may be 10 or more inches long) may cause death in children from venom introduced through their claw sting-punctures. General symptoms, such as vomiting, headache, fever, and even coma, may result from the bites. Local treatment of bites is the same as that recommended for tick bites and scorpion stings. All the larger centipedes are commonly regarded as venomous and are generally greatly feared. Large insects are quickly killed after the poison claws close upon them.
Scorpions are much more highly differentiated than are centipedes or millipedes. They may be recognized (Fig. 87) by their crablike appearance and long, fleshy five-segmented tail-like postabdomen, which terminates in a bulbous sac. The latter contains two poison glands separated by a muscular septum, and terminates in a curved hooklike sting with concavity dorsalward. When stinging, the postabdomen is arched over the back and the fine hypodermic needle-like point is stabbed into the prey, such as an insect or a spider, which is held firmly between the large, powerful, pincer-like pedipalps. In addition to this pair of pedipalps, scorpions have normally four pairs of segmented walking legs, by means of which they are able to move rapidly. The cephalothorax and forward segments of the abdomen function more or less as a unit, narrowing laterally to join the more flexible segmented postabdomen.

Scorpions are viviparous; after the young are born they are carried attached by their pedipalps to the back of the mother. In temperate climates, scorpions are usually rather small, but in the tropics they may reach a length of 6 or 7 inches. They are nocturnal in their habits, remaining hidden during the day beneath
stones, bark of fallen trees, boards, piles of lumber, floors, and debris of various sorts. In buildings, they find shelter in numerous ways.

**Nature and Toxic Action of Scorpion Venom.** The venom of scorpions is a transparent liquid, acid in reaction, and contains a neurotoxin, several lysins, and other related substances. Although the sting is very painful, the poison, as a rule, does not cause general symptoms in adults; but in children under five or six years of age, it may cause death, particularly in the tropics, where the larger species are common. A slow, full, and easily compressible pulse, rapid respiration, salivation, vomiting with epigastric pain and tenderness, and glycosuria, constitute the usual clinical symptoms produced by a serious scorpion sting.

**Treatment of Scorpion Sting.** Local treatment consists in applications of ammonia water or sodium bicarbonate paste, and when practicable, the use of a ligature. Analgesics may be prescribed to relieve local pain and to allay mental distress. Antiscorpion serum, when available, is indicated in severe cases. Shock, vomiting, and convulsions should be handled in approved ways by a competent physician.

**Control Measures.** Community control of scorpions is difficult, although cleanliness of streets and premises, together with improved construction of buildings, should be helpful. For prevention of the sting, individual precautions must be largely relied on. Constant thoughtfulness and intelligent vigilance in numerous ways will be helpful in avoiding the painful and sometimes toxic sting of scorpions.

**BEETLES** *(ORDER COLEOPTERA)*

Certain species of adult beetles are capable of blistering the skin or mucous membranes by means of the corrosive action of substances contained in their body fluids.

* Several species of beetles have been found to serve as intermediate hosts of helminths, the adults of which may infect man. These worms are a roundworm *(Gongylonema pulchrum)* of various vertebrates, the rat tapeworm *(Hymenolepis diminuta)*, and several acanthocephaloid (thorny-headed) worms. Temporary infestations of the digestive and urinary tracts and nares with larval or adult beetles have been reported.
There are three principal groups of these so-called "blister beetles": (1) "Spanish fly," commercially obtained from Cantharis vesicatoria; (2) "rove beetles," containing a toxic principle (other than "caantharidin" found in Spanish fly) which causes the so-called "Nairobi eye" in east Africa, when the juice of crushed beetles is rubbed into the eye; (3) "cocoanut beetles" of the mid-Pacific islands (genus Sessinia), which frequently swarm about the newly opened male flowers of the cocoanut to feed on the pollen.

BUTTERFLIES AND MOTHS (ORDER LEPIDOPTERA)

The larvae of one family of butterflies and several families of moths have special poison hairs or spines to protect them from their enemies. When these hairs or spines come in contact with tender skin, there follows more or less vesication or urtication, depending on the susceptibility of the victim, the area of skin affected, and the particular species of "caterpillar" involved. The toxic substance which causes the dermatitis is secreted by specialized hypodermal cells.

The unfortunate person coming in contact with poisonous hairs or spines of these caterpillars promptly experiences burning and stinging sensations. The affected site becomes erythematous; later, raised whitish areas with reddish macules develop. At times the part of the body involved becomes much swollen, and urticarial wheals appear at various places. This condition may be accompanied or followed by evidences of systemic involvement. When the defensive appendages of these larvae come in contact with the conjunctiva or cornea, they often cause most distressing pain which may be followed by the development of pseudotubercles at the points of contact.

Reports of dermatitis due to contact with adults of certain species of LEPIDOPTERA are apparently true in some instances. The resulting irritation is reported due to poison larval hairs clinging to the offending adult insects. It seems more probable, how-
ever, that such irritations as may be produced are due to hairs or scales occurring naturally on the bodies of the insects in question.

It is well known, for instance, that some of the "brown tail" moths bear poisonous hairs. In many instances, such hairs are probably not actually poisonous, but may nevertheless cause an urticaria when small pieces of hair penetrate the moist skin, particularly between the fingers and on other tender epidermal areas.

Unpublished reports which have come to my attention also indicate local prevalence, at times, of moths apparently bearing unusually irritating hairs or scales. Such occurrences are said to be associated with the presence in unusual numbers of certain rather inconspicuous small moths. The personnel of ships plying eastern Venezuela coastal streams have attributed a severe conjunctival and skin irritation to moths of this type, which were noticeably present at the time the irritations occurred.
CHAPTER XVI

POISONOUS, BITING, AND STINGING WATER-INHABITING GROUPS

The poisonous, biting, and stinging water-inhabiting forms (including certain disease-transmitting species) belong in one of the following categories:

1. **Coelenterates**
   - Anthozoan (actinozoan) and Hydrozoan polyps, particularly the sea anemones and certain corals
   - Jellyfish (medusae)
   - Portuguese man-of-war (genus *Physalia*)

2. **Mollusks**
   - Oysters
   - Mussels

3. **Fishes**
   - Poisonous as food per se in fresh, natural condition
   - Poisonous through bite or sting
   - Transmitters of disease in the capacity of hosts of bacteria or animal parasites

4. **Biting water bugs**

**COELENTERATES**

**Sea Anemones**

Sea anemones (Anthozoan polyps) are the most important of the stinging polyps and are well known to inhabitants and visitors at the seaside. They are often seen covering the soft posterior parts of crabs, sharing with them their food in return for protection from enemies afforded. While other crabs are very sensi-
POISONOUS WATER GROUPS

tive to the poison of the anemone (suffering paralysis and often
death), these parasitized crabs apparently acquire an immunity
against the poison of its stings. A condition which occurs among
sponge fishermen of the Mediterranean and probably elsewhere,
known as the sponge diver's disease (la maladie des plongeurs),
is caused by stings of the anemone. Although the symptoms in
man are usually limited to marked itching, burning, and erythema,
necrosis may also occur, followed by ulceration and sloughing.

JELLYFISHES

Most jellyfish * are comparatively harmless, but there are some
in the Mediterranean and in tropical waters, whose stings pro-
duce very unpleasant or even serious effects in man. The umbrella-
like, adult jellyfishes have tentacles which hang down from the
margin of the body. The particularly dangerous ones also have
long, specialized, filamentous, or ribbon-like sensory and stinging
organs attached about the mouth. In some forms these appendages
are provided with the so-called "nematocyst batteries" charac-
teristic of dangerous coelenterata.

PORTUGUESE MAN-OF-WAR

The interesting "Portuguese man-of-war" (genus Physalia),
the "thread cells" of which may inflict very painful stings, is a
colonial Hydrozoan coelenterate.

It has long locomotor tentacles which extend many feet down
into the water, and is propelled by the wind blowing against its
shiny, purple, crested bladder-like float or sail. The stings of
the "thread cells" are likely to be long remembered by those
who unwittingly come in contact with them.

NATURE AND TREATMENT OF STINGS OF COELENTERATES

Victims of the sting of dangerous Coelenterates usually experi-
ence at once a sensation of burning or stinging in the area of con-
tact, followed soon by erythema and swelling and at times a

* Many of the commonly-called jellyfishes in local waters are comparatively
harmless and incapable of stinging man severely.
vesicular dermatitis. A temporary local paralysis may occur even before the swelling is very noticeable. Manifestations of symptoms in other parts of the body may appear after ten or fifteen minutes. Not infrequently, symptoms referable to the respiratory tract arise, suggestive of allergy. Acute cardiac distress, dyspnea, muscle pain, and shock have been reported in some cases.

Treatment of stings by Coelenterates is largely symptomatic. Local applications, as for stings of Arthropods are useful. The onset of severe symptoms calls for complete rest with sedatives to relieve the pain. Respiratory embarrassment and cardiac distress require appropriate counter-measures.

MOLLUSKS

OYSTERS

Aside from the ill-digestive effects which oysters seem to have for some individuals, their real danger to man consists in a possible role as hosts of typhoid bacilli. Although oysters may be contaminated with these bacteria while growing naturally in sewage-contaminated beds, such contamination is more usually traceable to use of polluted water employed in “floating” this bivalve to improve its appearance and thus enhance its marketability. At present, floating in chlorinated sea water is considered to be a desirable sanitary procedure.

Oysters are most active during the warm summer months, ingesting large quantities of water from which the gills sift small food particles. These are caught by the mucus that coats the inner surfaces of the gills, and conveyed by progressive mass movement of the epithelial cilia into the funnel-shaped stomach. When the environmental temperature reaches 7.2° C. (about 45° F.), the oyster passes into an inactive state. The bacterial content of oysters is, therefore, more or less seasonal. Present laws take advantage of this fact and limit oyster harvesting to the months when the water is coldest. In the region of New York City, most of the beds of permissible edible oysters are located in deep, naturally cold waters.
Mussels and Related Bivalves

Mussels are widely distributed throughout the world and constitute an important article of food, particularly in Europe. The shell of the large fresh-water mussel is employed in button making. In addition to ill effects due to individual idiosyncrasy to mussels, poisonings which lead to paralysis or even death have been reported. A number of deaths traceable to eating poisonous mussels have occurred on the coast of California. The symptoms are said to have developed in from ten to twenty minutes after ingestion of the mussels. The toxin (thermostabile) is present in the mussel only during the spawning season (June through September).

Certain of the mussels and related bivalves are not considered edible at any time. In some instances certain parts only are not fit for food. William T. Davis of Staten Island (N. Y.), a naturalist, passes on an interesting story of a cat which ate discarded portions of edible scollops in the raw state. He says it was reported that "the ears gradually disappeared and there were other peculiar symptoms."

Fishes

Fish, Poisonous as Food Per Se in Fresh Natural Condition

There are certain fish whose meat is poisonous when eaten even in a perfectly fresh state. According to the scattered literature available, fish of this type generally become poisonous either by feeding upon organisms such as poisonous corals or medusae, or through poisonous metabolic products associated with spawning. Barracuda have been strongly suspected at times of being temporarily poisonous because of one or both of these reasons.

In islands of the Pacific Ocean and the Indian Ocean, certain kinds of fish are reputed by both natives and sailors to be notorious for the poisonous character of their tissues or organs. The best known of these are species of *Tetraodon* (Fig. 88), such as *T. hispidus* (the "death fish" of Hawaii), whose poison is con-
tained in the ovaries and eggs. This toxin causes both gastro-
intestinal and nervous symptoms in man, sometimes terminating
in collapse or coma.

There are also certain species of the herring family (genus
Meletta) whose flesh is poisonous. Their poison produces symp-
toms such as cramps, cold sweats, dyspnea, cyanosis, dilated pu-
pils, and sometimes even death. These fish inhabit the waters
around the West Indies and in the vicinity of islands of the Indian
Ocean.

In all these forms of fish poisoning, prompt evacuation and
washing out of the stomach is most important. Active purga-
tion should also be carried out, in order to minimize possible
absorption of poison from the intestines. Symptoms that arise
or that are anticipated require specific treatment.

**Fish, Poisonous Through Bite or Sting**

Venomous fish are found in most tropical waters, particularly
among the coral reefs of the Indian and Pacific Oceans. The
poison may be conveyed to man through (a) biting, or (b) sting-
ing. In the former group, the poison is secreted by special epithe-
lial glands situated within the mouth; in the latter, by poison
glands connected usually with stings on the dorsal fin, with spines
in the gill-covers, or with other parts of the integument, as "sting
rays" of the tails of certain Trigonidae. In Figure 88 a number
of the better known fish of this group are shown.

**Venomous Biting Fish**

Some species of the genus Muraena, which have powerful teeth,
are capable of inflicting very effective, poison-injecting bites.
The venom from adjacent poison glands is delivered through
hollow teeth into the wound at the time of biting. The venom
is neurocardiototoxic in character.

**Venomous Stinging Fish**

Venomous stinging fish are found in widely separated genera.
In some, the poison is released only when the barblike stings are
broken, often resulting in severe inflammation or even the development of tetanic symptoms. In others, the poison is delivered from special glands into most painful sting wounds, which are produced by strong, sharp, needle-like stings situated on dorsal fins, gill-covers, or caudal extremity.

A troutlike marine fish (*Trachinus draco*, Fig. 88) has poison apparatus connected with both fin stings and gill stings. French fishermen have named one of these most painful fin-stinging fish, which is red in color, "le diable," probably from its diabolic appearance as well as from its sting. Some of the sting rays (*Trygonidae*), with barb-armed tails, are particularly dangerous because of the deep character of the wound and the possibility of introducing tetanus bacilli. Persons wading in the water are in much more danger of being stung than are fishermen who handle the fish.
FISH, AS TRANSMITTERS OF DISEASE, IN THE ROLE OF HOST TO BACTERIA OR ANIMAL PARASITES

BACTERIA

Besides bacterial poisoning from stale or canned fish, there are certain instances of fish poisoning due to bacteria which are primarily the etiologic factors in epidemics among fish themselves. Most of the organisms isolated from such fish belong to the colon or proteus groups of intestinal bacteria and the poisoning is attributed to products elaborated by them. Cases of botulism-like poisoning from eating improperly salted fish have been reported. Stitt (1938) believes that such cases of poisoning may be due to the soluble toxin of *Clostridium botulinus*, which is destroyed by boiling, whereas that of Gartner's bacillus, the most common food-poisoning organism, usually withstands the temperature of cooking.

ANIMAL PARASITES

The two most important animal parasitic infections contracted by man through eating parasitized fish are (a) the broad or fish tapeworm (*Diphyllobothrium latum*), and (b) the small liver fluke (*Clonorchis sinensis*) of the Far East. The former is a common parasite of the Baltic countries of Europe and the upper Great Lakes region of the United States. The latter is particularly prevalent in China, Japan, and Korea. In both instances, the disease in man is contracted through eating raw, improperly cured, or insufficiently cooked, infested fresh-water fish, which are the second intermediate hosts. The primary hosts are a crustacean (cyclops) in the first instance, and a mollusk (snail) in the second.

The oriental lung fluke (*Paragonimus westermani*) is another important example in which the first intermediate host is also a snail, the second being a fresh-water crustacean (crayfish or crab). The definitive host (man) is infected by eating the infested soft parts of the second fresh-water intermediate crustacean host. In the case of a less important, small intestinal heterophid fluke
POISONOUS WATER GROUPS

(Metagonimus yokagawai), common in the Orient and reported also from Siberia, the Balkan States, and even Spain, infection is acquired through eating infested fresh or salted fish from fresh or salt water, the first intermediate host being a snail as in P. westermani.

BITING WATER BUGS

Some of the biting water bugs are among the largest of bugs, and are commonly referred to as the giant water bugs. They feed on other water insects, fish, young frogs, and the like. Light readily attracts them. Those known as "electric light bugs" include one, Lethocerus americanus (Leidy), which, it is said, may inflict a very severe bite, the symptoms sometimes lasting for several days.

Members of the family Notonectidae, commonly swim on their backs, hence the popular name "backswimmers." In some persons, according to Herms (1939), the bite of some species belonging to this family are often followed by symptoms approaching in severity those of bee sting.
All poisonous snakes belong to one or the other of the two families, COLUBRIDAe or VIPERIDAe.

COLUBRIDAe

The COLUBRIDAe (poisonous and non-poisonous) may be distinguished from the VIPERIDAe (all poisonous) by their relatively longer maxillary bones which lie in a more or less horizontal and fixed position. The grooved venom-teeth or fangs of poisonous colubrids are usually securely attached to these bones. In the more dangerous species, they are anteriorly situated (in front of the smaller maxillary teeth, if present); in most instances those with fangs posterior to the maxillary teeth are less to be feared by man. The gum ridge behind the “fangs,” on either side, is generally devoid of teeth except for one or more grooved accessory fang-teeth. The usual palatine and pterygoid teeth form a single row on either side in the roof of the mouth, and mandibular or lower jaw teeth are regularly present.

Classification of Poisonous Colubrids

The COLUBRIDAe fall conveniently into two general subfamily groups—the land forms (ELAPINAE) and the sea forms (HYDROPHINAE).

LAND FORMS (ELAPINAE)

The ELAPINAE have somewhat short, stout, grooved fangs, anteriorly situated in the more dangerous species, behind which there may be several grooved teeth. The poison ducts discharge
their venom into mucous folds which surround the bases of the fangs. When a fang is broken or pulled out, a potential fang to the rear takes its place. The loss of a fang does not usually injure the poison duct and thereby prevent the outflow of venom. Fangs are periodically replaced regardless of injury to the functional ones. A succession fang grows in next to the functional one before it is lost, and for a short time both fangs may function.

Many colubrids of the United States, such as the garter and black snakes, are harmless, but two venomous species of the genus *Micrurus* (*Elaps*), coral snakes (Fig. 89), are found in the southern United States. (About thirty-five other species of this poisonous genus are found in Mexico, Central, and South America.) In general, Elapines of Asia, Africa, and Australia are much larger and more numerous than those of the Americas. They include the cobras and kraits of southern Asia, numerous African venomous snakes, and all of the dangerously poisonous land snakes of Australia, where vipers do not occur.

**SEA FORMS (HYDROPHINAE)**

In contrast to many venomous colubrid land snakes, the poisonous sea snakes (Fig. 89) have relatively small heads, laterally flattened tails, and the bodies also of certain species are somewhat laterally compressed. Generally, they live in salt water, particularly in the continental and island coastal waters of the Indian Ocean. Some species are also found in islands of the western Pacific and the Pacific Coast of tropical America. According to Taylor (1922), a species of *Disteira* occurs in a fresh-water Philippine lake. However, this lake is only a few kilometers from the sea, and is connected with the sea by a short river. Sea snakes are frequently found many miles up tidal streams.

The venom of sea snakes is very toxic. The fangs are anteriorly situated with the poison grooves almost completely enclosed. Consequently, HYDROPHINAE must be considered extremely dangerous, notwithstanding assertions that their bites are frequently slow and ineffective. Such statements probably are based on mistaken identity, that is, eels or harmless snakes which are similar
in appearance and which have been confused with sea snakes. In sorting their catch, fishermen may be bitten. Ordinarily, sea snakes are not likely to bite man unless they are handled.

VIPERIDAE

Vipers are found throughout the temperate and tropical parts of the world except Australia, New Zealand, and Madagascar; there are no poisonous snakes of any kind in New Zealand. They are frequently characterized by a broad head, relatively short jaws, a narrow neck, a rather thick body, and a short abruptly tapering tail, or a short tail terminating in a rattle or series of loosely articulated horny links. In the newborn, the rattle is represented by a rigid horny bulge or button incapable of producing sound.

In contrast to elapines, the maxillary bones of vipers are shorter, relatively more massive, and inclined in relation to the long axis of the head. The jaws are blunt anteriorly, and in some vipers have a turned-up or snoutlike appearance. The articulations of the maxillae with other bones of the skull are somewhat flexible so that when the mouth is opened, they can be rocked forward carrying the fangs outward and somewhat upward.

Another notable characteristic of the vipers is the elliptical shape of the pupils, the iris often appearing slitlike vertically (Fig. 89). This, however, is not found in all species; some have round pupils, as in most colubrids. A third feature, formerly thought to be distinctive of vipers, is the presence of a single row of broad subventral scales closely behind the anal shield. While this feature does not occur in all species, it is found in the crotalines and lachesines of the United States. On the other hand, most colubrids, including the many poisonous ones, are more likely to have a double row of ventral scales. Some of these more obvious

* Some vipers, however, cannot be distinguished from colubrids by external appearances, notably the genera *Causus* and *Atractiaspis*, of Africa; some also have small rounded heads, with little or no demarcation between head and neck. They resemble coral snakes in this respect.

** This type of pupil is often found in reptiles which are predominantly nocturnal in foraging for food, and is seen in all the generally recognized poisonous snakes of the United States, except the coral snakes.
Fig. 89. Differential features of poisonous and non-poisonous snakes of the United States (except sea-snake in lower right corner). (Adapted from Stitt, Clough and Clough. Practical Bacteriology, Haematology and Parasitology. Edition 9. Copyright, the Blakiston Company, Publishers.)
distinguishing features of poisonous snakes of the United States are depicted in Figure 89.

**Classification of Vipers**

American vipers may be divided into two subfamily groups: CROTALINAE and LACHESEINAE.

(1) CROTALINAE (pit vipers with rattles—rattlesnakes) possess so-called “loreal pits,” delicate sensory organs which it is believed enable snakes possessing them to better detect the presence and location of their prey. Recently it has been determined experimentally that these organs are sensitive to heat. Pit vipers with rattles (genera *Crotalus* and *Sistrurus*) are confined to the Americas, while those without rattles are found in the Americas, in Asia, and in regions of eastern Europe.

(2) LACHESEINAE (pit vipers without rattles—“rattleless rattlesnakes” and moccasins), which comprise the genus *Agkistrodon* with one species in Mexico and two species in the United States, the large tropical genus *Bothrops* (includes *B. atrox*, the fer-de-lance), and the single species *Lachesis muta* (the bushmaster) of tropical South America and Panama.

With the exception of the two poisonous species of American coral snakes already referred to, the venomous snakes of the United States are true “pit vipers.” Non-pit vipers (VIPERINAE) which lack loreal pits are confined to the Old World and are most abundant in Africa. (However, it is believed that they have inconspicuous nasal pits which function similarly.) The daboia or Russell’s viper (*Vipera russelli*) of India is one of the most important poisonous snakes of this group.

A classified list of the generally recognized poisonous genera of the Western Hemisphere is given in Appendix IX A, while species found in the United States and Canada are given in Appendix IX B, together with their geographical ranges.

**Poison Apparatus**

All viper fangs have completely enclosed poison conduits with slanted exit openings, which suggest in appearance a curved hypo-
dermic needle (the fangs of the colubrids are grooved only). The fangs are closely approximated to papillae in which the openings of the ducts of the venom glands terminate. The venom glands are salivary * in origin, situated laterally posterior to the fangs, and are encased in bands of pterygoid, temporal, and maxillary muscles.

The fangs of vipers (Fig. 89, center), anterolateral in position, are backwardly curved and normally are folded against the palate well out of the way, being concealed from view by a hood of loose tissue called the vagina dentis. Rocking of the loosely articulated maxillary bones forward permits a 45 degree or more forward extension of the fangs, but the mouth must be opened wide and the neck flexed dorsalward in order to execute a strike of maximum effectiveness. The angle at which the lacrimal bones are hinged to the maxillae makes forward thrust of the attached fangs mechanically and synchronously effective when the parietopterygoid and sphenopterygoid muscles attached to the pterygo-transverse bar or quadrate bone are contracted.

During the progress of the combined strike and stab, while the fangs are being elevated, the vagina dentis of each fang falls backward and is gathered into loose folds at the base of the fang. As the unsheathed fangs penetrate the skin, a series of almost simultaneous muscular movements occurs, the body of the snake being rigidly fixed by its coiled posterior portion. In Figure 90 is shown a Texas diamond-back rattler coiled just preparatory to striking (left) and the same snake in the course of striking from the unfolding coil (middle and right). Although a snake may turn and strike from any posture, the “striking coil” is the attitude almost always assumed under normal circumstances and conditions.

According to evidence deduced from slow moving pictures, the bite is in reality a stab, which is perfectly timed to coincide with

* The saliva of most snakes when injected into prey has toxic effects of greater or lesser degree. In the case of the rear-fanged Colubrids, such effects must be considerable, as indicated by reaction of small animals to it. So far as man is concerned, however, the amount likely to be injected by rear-fanged snakes is generally small, often causing no symptoms or only mild ones.
the injection of venom. Apparently vipers make no attempt to force the fangs to a greater depth than that reached with the impetus of the strike. The rapidity with which both strike and recoil usually take place almost precludes many of the accessory muscular contractures described by earlier writers as part of this mechanism. If the snake misses in striking, the venom is often ejaculated notwithstanding. However, the components of the striking mechanism, although closely co-ordinated, are largely under the control of the snake. One fang only may be erected.

**Character and Potency of Venoms**

Venom is a thick, clear (or slightly milky), usually lemon yellow or colorless fluid, composed principally of modified proteins, ferments, lysins, and other ingredients. In most of the crotalines of the United States, according to do Amaral (1928), the solid portion represents about 25 to 35 per cent of the total weight of the venom. In general, there are two types of venom, correlated with families to which the venomous snakes belong.

*Venom of colubrid snakes*, such as the coral snakes of the United States and many of the poisonous land and sea forms of the Eastern Hemisphere, acts mainly upon the nervous system and ultimately brings about respiratory paralysis. Typical colubrid venom contains neurotoxin, hemolysin, and anticoagulin; the neurotoxin acting principally on the bulbar and spinal ganglion cells.
VENOMOUS SNAKES AND LIZARDS

Venom of viperid snakes, such as the pit vipers of the Americas and the non-pit vipers of the Eastern Hemisphere, acts primarily on the vascular system. Typical viper venom is characterized by the presence of hemorrhagin, thrombase, and cytolysins. Hemorrhagin destroys the endothelial lining of blood vessels, thrombase causes intravascular thrombosis, and cytolysins act deleteriously on red blood corpuscles, leucocytes, and tissue cells.

An analysis of venom of the cobra and Russell's viper, typical respectively of the venom of the colubrid and viperid families, gave the results shown in Table XIV.

The venom of practically all poisonous snakes usually contains some of the various substances mentioned. The amount of each substance present in the venom, however, varies quantitatively in the different groups of snakes, usually to a lesser degree in those most closely related. The venom of a typical elapid, like the cobra, which contains very little or no hemorrhagin but relatively large amounts of neurotoxin, causes minor local effects but intense nervous symptoms. On the other hand, while the venom of practically all North American vipers (crotalines and lachesines) produces severe local manifestations, nervous reactions (notably paralytic phenomena) may be delayed and when they do occur are less prone to endanger life.

Table XIV

ANALYSIS OF TYPICAL VENOMS
(From Byam and Archibald, 1921)

<table>
<thead>
<tr>
<th>Per Cent Toxicity of Whole Venom</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Coagulable proteids, 65° to 75°</th>
<th>Copper</th>
<th>Russell's Viper</th>
</tr>
</thead>
<tbody>
<tr>
<td>38%, includes the anticoagulin and hemolysins</td>
<td>74%, includes the hemorrhagin</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Enzymes</th>
<th>Copper</th>
<th>Russell's Viper</th>
</tr>
</thead>
<tbody>
<tr>
<td>12% proteolytic ferments of nature of kinases</td>
<td>3% proteolytic ferments of nature of kinases</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Noncoagulable proteids</th>
<th>Copper</th>
<th>Russell's Viper</th>
</tr>
</thead>
<tbody>
<tr>
<td>50%, (albumoses), includes the neurotoxin and stimulin</td>
<td>23%, (proteases, primary and secondary albumoses), includes cytolysins, thrombase, and cardiac depressor</td>
<td></td>
</tr>
</tbody>
</table>
Among American crotalines, the toxic effects of the venom per se are very similar in most instances. Working experimentally with pigeons, Githens and George (1931) found that the venom of western specimens of *Crotalus horridus* (the timber rattlesnake) may result in greater toxic action than does the venom of eastern specimens. They obtained similar greater results with the venom of *Crotalus durissus* (the tropical dog-faced rattler), which produces a highly neurotoxic venom, the local effects of the strike often being insignificant.

The coagulable proteid of cobra venom is relatively less in amount than the noncoagulable portion, whereas the reverse is true in the case of Russell's viper venom. The enzymes of cobra venom are said to be less active than those of viper venom, but they are more abundant and heat resistant. These rough quantitative analyses, considered in connection with the qualitative findings of the different fractions, suggest, in a general way, the probable effects of the two venoms on tissues and organs.

The amount of venom which a snake may inject and the minimum lethal dose (M.L.D.) for man are of vital significance. In this connection, data from Byam and Archibald (1921) given in Table XV are interesting.

According to the figures given in the last column of Table XV, all of the listed snakes, except the green pit viper, are easily capable of giving a fatal dose to man at one strike.* The circumstances and conditions attending a particular encounter with a poisonous snake may be such as to thwart its efforts to inject a fatal dose. If, however, the strike is successful and venom is forced into the wound, untoward effects of greater or less intensity are certain to follow. The actual quantity of venom injected and its specific toxicity are the factors determining the amount of injury which an individual may sustain.

* Venomous snakes are somewhat prone to strike more than once, if necessary, in order to accomplish their purpose. This is particularly true of the elapines, although vipers often do not hesitate to strike a second time. The latter are noted for their efficiency in venom injection. The green pit vipers' low rating in Table XV may be due to the fact that it must of necessity, according to these figures, strike more than once to inflict a dose of venom likely to be fatal to man.
### Table XV

#### RELATIVE TOXICITY OF VENOMS

<table>
<thead>
<tr>
<th>Snake</th>
<th>Approximate Dose Given at Bite</th>
<th>Estimated Fatal Dose for Man</th>
<th>Fraction of Fatal Dose to Approximate Amount Given at Bite* **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common cobra (Naja naja)</td>
<td>211.3*</td>
<td>15.0*</td>
<td>(\frac{1}{14})</td>
</tr>
<tr>
<td>King cobra (Naja hannah)</td>
<td>100.0 10.0  (\frac{1}{2})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Common Indian krait (Bungarus candidus)</td>
<td>5.4 1.0 (\frac{1}{2})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Banded krait (B. fasciatus)</td>
<td>42.9 10.0 (\frac{1}{4})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Russell's viper (Vipera russelli)</td>
<td>72.0 42.0 (\frac{1}{2})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Green pit viper (Trimeresurus gramineus) (Max. 37)</td>
<td>14.1 100.0 (\frac{1}{7})</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Milligrams of dried venom. **Figures are approximate.

While no general conclusions should be drawn from the figures in Table XV, they may be of value in making comparisons with results obtained by other investigators. Employing sheep as experimental animals, Fairley (1929) found the potential killing power of the death adder \(Ancomnathris antarcticus\) and of the tiger snake \(Notechis scutatus\) of Australia to be, respectively, 2.7 and 3.5 times that of the common cobra. Fairley (1929) further states that these colubrids possess to a variable degree the power of elevating and rotating forward the fangs; and that the length of the fangs seems to influence the relative toxic effect produced by the venom.

In so far as the Nearctic pit vipers are concerned, do Amaral (1928) found that the average amounts of venom secreted at one time ranged from 2 mg. of the dried venom for the pigmy rattler \(Sistrurus miliaris\) to 300 mg. for the eastern diamond-back \(Crotalus adamanteus\). For the Texas diamond-back \(Crotalus atrox\) which rates 120 mg. of dried venom on do Amaral's com-
parativc scale, Jackson (1928) found the minimum lethal dose to be about 1 mg. of dried venom per pound of body weight when given intramuscularly to dogs. If this relationship were to hold true for man, who is probably relatively more responsive to venoms, the average amount secreted at one time by the Texas diamond-back would be enough to kill a human being. Fairley's (1929) experimental work on sheep afforded figures which may be compared with these. He found that the average venom yield from a Russell's viper would kill 2 sheep; whereas the venom of a cobra would kill 31, and the venom of the most poisonous Australian elapines, 118 sheep.

EFFECTS OF SNAKE BITE

If, as Fairley (1929) remarks, snakes could inject with certainty the entire contents of their venom glands, recovery of victims would indeed be rare. Fortunately under natural conditions many factors operate to prevent this, so that often sublethal doses are injected. The following factors, according to Crimmins (1927), tend to reduce the effects following rattlesnake bite:

1. The snake missing his goal—the rattler does not see well during the day or before shedding, and may miscalculate the distance and strike a glancing blow without injecting venom.

2. Diminution in amount of venom, due to (a) hibernation, (b) aestivation, (c) previous exhaustion of the venom in feeding, (d) captivity.

3. Bite being inflicted through clothing that retains some of the poison.

4. Movement at time of strike making imperfect bite.

5. Region bitten being very lean, i.e., having little connective tissue, as the finger, toe, or shin.

6. Region bitten being very fat, so that its circulation is poor.

7. Ejection of part of the venom before the rattler strikes.

8. Age of the snake—very young or very old snakes produce little venom.
VENOMOUS SNAKES AND LIZARDS

PATHOLOGIC ACTIVITY OF CROTALINE AND LACHESININE VIPER VENOM

The untoward effects of the venom of vipers is due chiefly to a lysin * which destroys the endothelial lining of blood vessels. It also contains proteolytic ferments, which probably accounts for the softening and destructive effects on the tissues. Hemolysis from Nearctic crotalines is not nearly so marked as that produced by the venom of crotalines of Central and South America and the West Indies. There, because of the more destructive effects of the venom (particularly upon blood cells), as indicated by the rapid appearance of reflex vomiting, hemorrhages of conjunctivae, stomach, and other organs and tissues, the unfortunate individual may die within a few hours if the specific antivenin is not given promptly.

SYMPTOMATOLOGY OF SNAKE BITE

The local and systemic reactions of the body to snake bite vary quite widely. The clinical pictures presented by persons bitten by the same species of snake may exhibit considerable variation. The course, therefore, in individual cases cannot be predicted. The amount of venom injected, together with its character and toxicity and the absence or presence of certain complications, constitute the principal factors involved. It may be said in general that with a few exceptions the local symptoms caused by the bite of poisonous colubrids are much less pronounced than those arising from the bites of viperids. It must be remembered, however, that ligatures tend to distort and intensify local signs and symptoms, particularly if improperly applied.

The time of onset of systemic manifestations following bites by poisonous colubrids depends largely upon the amount of venom

* The venom of Crotalus durissus (dog-faced rattlesnake of tropical America) is an important exception among the Crotalines, in that it is strongly neurotoxic, affecting vision and respiration with slight local activity. It should be pointed out, however, that the venom of Nearctic crotalines is not by any means devoid of neurotoxic activity. Respiratory difficulty, in addition to the more common neurotoxic manifestations such as nausea and vomiting, occurs and is said to be rather more pronounced in poisoning by the Texas diamond-back, the wood's rattler of northern and central states of the United States, and the water moccasin than in others.
injected and the rapidity of its absorption. Numerous factors contribute to the ultimate result. The first general symptoms may appear within a few minutes to two hours or more after the bite. They consist of nausea, faintness, vomiting, headache, drowsiness, and lethargy. Among early neurotoxic symptoms which frequently follow quickly after successful poisonous colubrid bites are muscular weakness, paresis of the limbs, and ataxia. Various other common symptoms, such as desire to urinate and defecate, and difficulty in swallowing, are frequent. Bulbar paralysis is almost always associated with neurotoxic activity; signs of respiratory paralysis invariably appear but usually improve under artificial respiration.

Ordinarily, with the bites of most crotalines as well as vipers, there is sharp burning pain at the site of the wound, with profuse bleeding, rapid swelling and hemorrhagic infiltration, and a tendency to petechial mottling as the swelling spreads. Other symptoms which are likely to follow are nausea, cold sweats, signs of cardiac depression, and collapse, depending on the amount and composition of the specific venom injected. A marked exception to this is the neurotoxic venom of *Crotalus durissus* referred to in the footnote on page 369. The habitat of this pit viper ranges from Mexico to Brazil and the Argentine.

Cardiovascular symptoms due to vasomotor paralysis and cardiac failure are the more immediate and characteristic systemic effects produced by viperid venom. The earlier manifestations are subnormal temperature, feeble, rapid, and threadlike pulse with low blood pressure, blanched skin, cold extremities, nausea, vomiting, and sweating. Bleeding of gums, epistaxis, hematemesis, and hemoptosis may occur. When increased coagulability of the blood results, as from the venom of Russell's viper (also the common krait, an elapine snake), intravascular thromboses may occur.

Death resulting from a viperid (crotaline, lachesine, or viperine) bite may occur in one of several ways: (a) Almost immediately, from intravascular thrombosis, if the venom is injected into a vein, or when large experimental doses are given intravenously; (b) in a few hours, from cardiac failure due to vasomotor paralysis; (c) in a few days, from secondary hemorrhages
resulting from lytic action on endothelial cells of the smaller blood vessels; (d) from secondary septic infection of the necrotic area at the site of the bite.

**Treatment of Snake Bite**

The rational treatment of snake bite has been the gradual outgrowth of clinical experience and experimentation during the past fifty to sixty years. Therapeutic efforts should be directed toward (1) preventing absorption of the venom and removing or destroying it in situ, (2) neutralizing the toxic effects of the venom both locally and in the general circulation, and (3) sustaining the morale and general physical condition of the patient.

**Local Treatment**

A tourniquet properly applied and a local incision well made are an essential part of the treatment of snake bites. As an accessory measure, the application of a local chemical antidote appears to have been suggested and used first in the form of potassium permanganate by Sir Joseph Fayrer (1909) about 1870. Later, Calmette (1908) introduced the use of gold chloride solution. A very dilute solution of bichloride of platinum has also been recommended for this purpose.

Both in animals and in vitro experiments these and other chemical antidotes have been found to destroy or inactivate venom when they are brought in contact with it under favorable conditions. Investigators in India and elsewhere have found the therapeutic application of these chemicals to be efficacious only if (a) the injection in the case of neurotoxic venom is given before a lethal dose of venom has entered the circulation, which is considered to be about ten minutes or less without a ligature or one-half hour or less with a ligature; (b) the site of the bite is suitable for injecting a sufficient quantity of the solution (preferably 10 to 15 cc. of a 1 per cent solution of gold chloride; (c) the permeation of the affected area is sufficiently complete.

When a fatal dose of the venom has entered the general circulation, local remedies are of little value. In poisonous colubrid
bites, the time factor is more vital, since absorption of venom is so likely to be more rapid than it is in case of viperid bites.

The local treatment of rattlesnake bite suggested by Crimmins (1927) and used in Texas extensively, that is, the application of a ligature, incision, and suction applied over the involved area, has been very successful. It was shown by Jackson (1927), who studied this method experimentally, that a large fraction of a superfatal dose of rattlesnake venom inoculated into a dog could be successfully removed, with prompt recovery of the animal. This method is being generally substituted for the previously widely used permanganate method, which was popularized through the efforts of Fayrer, Brunton, and Rogers (1909). Regarding the use of permanganate crystals, Ditmars (quoting do Amaral) states in Cecil’s (1940) Textbook of Medicine: “Nothing could be more foreign to the treatment of snake-bite than such practice.” He therefore recommends prompt deep incision and forced suction for local treatment. Cauterization should under no circumstances be employed. A suitable pocket-sized assembly, containing the essential articles for incision and cupping, is now obtainable for about a dollar and a half. Specific directions for its use accompany each of these treatment outfits.

**IMPORTANT POINTS IN GENERAL TREATMENT OF SNAKE BITE**

There are a number of points that should be kept in mind in the local and systemic treatment of snake bites:

1. The tourniquet should be firmly but gently applied above the site of the bite, but it should not be allowed to remain tight for more than ten to twenty minutes without being temporarily released to permit the blood vessels and tissues to recover from the compression.

2. During the period of ligation, active cupping should be faithfully executed, supplemented by fairly deep subcutaneous hypodermic injection of sterile physiologic salt solution just peripherally to the area of advancing eccymosis. Better still, if available, is the use of specific antisera in place of, or in connection with, the salt solution.
3. Alcohol, strychnine, and the like are not to be considered as antidotes, and when used should be employed only in mild stimulant doses.

4. The patient should be kept quiet and warm, preferably in a prone position.

5. Administration of adrenalin is of value, if the venom is of the type that exerts a marked paralytic action on the vasomotor center.

6. To counteract the effects of respiratory paralysis or heart failure, persistent artificial respiration is helpful.

**SPECIFIC SERUM THERAPY**

Specific serum therapy should be made available as soon as possible. The time of its use and the amount to be administered should be left to the discretion of the physician. In the meantime, local and supportive treatment must not be relaxed. As a matter of fact, if local treatment is promptly, properly, and vigorously applied, resort to the use of antivenin will frequently not be necessary, particularly in bites by crotaline snakes of the United States. However, symptoms pointing to toxic absorption of venom into the general circulation call for prompt, active, specific serum therapy.

In bites by colubrid snakes, such as our coral snakes, and the cobras and kraits of Asia, prompt use of specific antivenins is an urgent measure from the beginning. In either case, when the specific antivenin is given, an adequate fresh supply should be available and it should be administered without stint or hesitation. The possibility of inducing anaphylactic serum shock, however, must be kept constantly in mind and guarded against in so far as possible. Children, owing to their small size and more rapid absorption in vital areas, often require more serum than adults in proportion to age.

In India, Burma, and the Malay States, the venom of many of the poisonous colubrid snakes, as well as that of vipers such as *Vipera russellii*, is often rapidly absorbed, with fatal effects. For this reason, it is the practice to give the specific antivenins, when
they are available, as soon as possible. This is based on the demonstration by Acton and Knowles (1914) that the specific serum must be present in the circulation before a minimum lethal dose of the venom has been absorbed, which may occur in considerably less than ten minutes if a tourniquet is not used promptly and effectively. The astute clinician, therefore, when confronted with an emergency of this type, will need to weigh most carefully all of the available evidence, knowledge, and personal experience, and, after having done so, to act promptly and vigorously.

POISONOUS LIZARDS

Lizards are nonvenomous, with the exception of members of the genus *Heloderma*. The two poisonous species are *H. suspectum* (Mexican beaded lizard) found in the states of Arizona, New Mexico, and into Mexico; and *H. horridum* (Gila monster), now found only in Mexico and named from the Gila River valley in Arizona whence the genus was first described.

These lizards are stout, yellow or salmon colored, and are partially covered with black, beadlike scales or tubercles. The length of very large adults is about 18 inches. They are desert dwellers, and deposit their eggs (which have a parchment-like shell) in the sand. In their swollen tails they store fat to tide them over periods of famine.

The poison apparatus is situated in the lower jaw, in contrast to the upper jaw in snakes. Another point of difference is lack of expansibility of the jaws, so marked in snakes. The secretion of the maxillary glands (parotid glands in snakes) contains the venom, which is carried through ducts which open at the bases of grooved teeth. When aroused, the “Gila monster” is likely to be very vicious in demeanor, and it is authoritatively reported that its closed jaws are difficult to open.

The onset of poisoning is indicated by symptoms of developing paralysis, and if the dose of venom injected is large, dyspnoea and convulsions are likely to appear. Autopsies on animals that have received a toxic dose of the venom have been reported to show a dilated heart with venous congestion of the internal organs.
APPENDICES
APPENDIX I

A WARM STAGE FOR USE WITH THE MICROSCOPE

A substitute for the somewhat expensive electrically heated warm stage illustrated on page 4 may be constructed in the laboratory.

Fig. 91. Working sketch for making simple warm stage for microscope.

A sheet of brass or copper about 1/16 inch thick is cut according to the shape and dimensions given in Figure 91. The broad end of this metal plate is shaped to cover the microscope stage and should have a circular opening corresponding to that of the microscope stage; the narrow end should project forward and outward from the microscope stage at an angle of about 45 degrees. Small clamps, preferably secured permanently to the plate, are necessary to hold it firmly in position. For the purpose of in-
sulation, and for conservation of the heat that will be applied to the projecting end of this warm stage, the under surface of the metal should be covered with thin sheet asbestos, as should also the portion of the upper surface that extends beyond the microscope stage.

In Figure 92 is shown a device of this type, clamped in position on the stage of a microscope. Any handyman or tinsmith should be able to duplicate this satisfactorily by following the directions given in Figures 91 and 92. A convenient addition to this warm stage is a thin-walled metal tube for holding a thermometer,
which may be secured to the under side of the metal plate, in contact with it and along either the lateral or the front side of the microscope stage. A careful examination of Figure 92 will make apparent details of the use of this warm stage.
APPENDIX II

CARE AND USE OF THE MICROSCOPE AND THE MEASURING ACCESSORIES

It is assumed that the student has acquired a practical working knowledge of the ordinary compound laboratory microscope, preferably of one equipped with a triple nosepiece fitted with 2/6 inch (16 mm.) and 1/6 inch (4 mm.) dry objectives, and 1/12 inch (2 mm.) oil immersion objective. However, a few pertinent words regarding the care of the microscope and its use in parasitologic study may not be amiss. Too often, protection of eyepieces, lenses, and condenser from dust and soiling, and occasional oiling of its moving parts are neglected. Particles of dirt on the glass surfaces of the microscope are likely to annoy or confuse the observer. If these become troublesome they may be located and removed by the following procedure adapted from Blacklock and Southwell (1932):

1. Move the slide; if the dirt also moves, it is on the object or the slide.

2. If the dirt is not on the slide, rotate the eyepiece while looking through the microscope steadily; if the dirt moves, it is either on or in the eyepiece. To remove it, exhale on the respective lens surfaces and clean them carefully with grit-free lens paper.

3. When neither of these procedures removes the dirt, it is either on or within the objective or condenser. Clean the exposed glass surfaces of these with grit-free lens paper moistened with water (preferably distilled), or, if necessary, with benzol. Alcohol should not be used; xylol may be employed sparingly in lieu of benzol.
4. If thorough external cleaning of the eyepiece, objective, and condenser does not give satisfactory results, the dirt may be on the internal glass surfaces, or between the respective component elements of these parts. In such instances, the mountings should be screwed apart and all surfaces thoroughly cleaned by a person competent to do so.

In order to prevent dust and similar substances falling into the microscope tube and possibly lodging upon the rear lens of the objective, the eyepiece should be kept in the draw tube or the opening should be covered with a suitable cup when the instrument is not in use.

The illumination of the microscope should be adjusted to the type of preparation under examination. In studying fresh unstained material, for instance, beginners are likely to employ too strong a light, which not only dazzles the observer, but so distorts or obscures the normally visible details as to render inspection almost useless. To begin with use of lower power objective and to exhaust its possibilities before substituting a higher power is a valuable rule to follow, and if this is done routinely, a great deal of unfruitful effort will be avoided and much time saved. Another practical consideration in this connection is racking the condenser out of focus, removing it, or wherever practicable swinging it away from the optical axis or line of vision. By so doing objects are made to stand out in bolder relief with much improvement in the visibility of colorless details.

Estimation, and occasionally actual measurements, of the dimensions of an object encountered in material under scrutiny should be made as a means of tentative classification, and also to ensure accurate detailed descriptions. Comparison of an object with red blood cells or even with leucocytes, if present, is a useful means of estimating size, but only as a preliminary measure. An eyepiece micrometer must be used to obtain accurate measurements. A micrometer of this type which can be supplied by any microscope dealer at moderate cost, is shown in Figure 99 A (magnified tenfold). This is inserted into an ordinary microscopic eyepiece which should have a diaphragm at the focal plane.
of the real image of the object. If the micrometer scale placed with ruled surface down cannot be plainly seen by the observer simultaneously with the object under scrutiny, then the eyepiece diaphragm is not on the same plane as the real image of the object and should be adjusted so as to get this result.

Before employing the eyepiece micrometer, each eyepiece-objective combination and tube-length should be calibrated against a known standard, such as a stage micrometer, or in lieu of this, a hemacytometer. The stage micrometer is marked with a scale of definite value (usually 1 mm. in length) divided into tenths and hundredths. This is etched upon a standard 1 by 3 inch glass slide and protected by a small circular coverglass. It is usually stamped with the exact value of the graduations and the name of the maker. Use of a single tube-length will simplify the calibrations of the eyepiece micrometer to some extent. If a stage micrometer is not available, the counting chamber of a hemacytometer will serve as a substitute. The lines which form the sides of the small squares used in counting red blood corpuscles are 50 μ apart.

Figure 93 B shows the ocular micrometer scale as seen through the microscope, superimposed in proper focus on the stage micrometer scale. If the former rests obliquely upon the latter, which is likely to be the case as shown here, the two scales may be brought into alignment by rotating the eyepiece of the microscope. When the two scales are parallel and the base lines of both coincide, as shown in Figure 93 B, other points at which the lines coincide may be determined. Since the values of the spaces of the stage micrometer are known, a ratio between unit spaces of the two scales may then be established. Having determined such ratios for each eyepiece, objective-combination and tube-length to be employed, it becomes a simple matter of arithmetic to work out the measuring unit values of the eyepiece micrometer for every such combination.

In each instance, the value of the included spaces on the stage micrometer is divided by the number of divisions on the ocular micrometer required to measure them. The quotient so obtained
represents the value of a unit division of the eyepiece micrometer in terms of a unit division of the stage micrometer. For example, if with a given optical combination and tube-length, it requires

![Diagram A: Ocular micrometer scale as seen in field of 10x eyepiece.]

100 spaces of the ocular micrometer to cover 17 spaces of the stage micrometer, one space of the ocular micrometer includes only 0.17 of a division of the stage micrometer, the value of which is 0.01 mm. The unit value of the eyepiece micrometer then is 0.17 of 0.01 mm., or 0.0017 mm., which in microns is 1.7. Higher optical combinations give corresponding smaller values of the
eyepiece micrometer units. The size of any object in a microscopic field is then determined by multiplying the corresponding value of one unit or space of the eyepiece micrometer by the number of spaces required to measure the length or breadth, and so on, of the object in question. This procedure is illustrated with a hookworm egg in Figure 33 D.

In Figure 93 C is shown the magnified eyepiece micrometer scale superimposed upon the ruled surface of a hemacytometer for the purpose of calibration. The principles involved are identical with those applied when the stage micrometer is employed. A comparison of A, B, and C of Figure 93 should make the procedure clear.

If an eyepiece micrometer is not available, it is possible to measure microscopic objects with a stage micrometer alone, or with the counting chamber of a hemacytometer. This kind of micrometry though somewhat less convenient, is nevertheless fairly satisfactory for comparatively coarse objects such as worm eggs. It is carried out in the following manner: A ruled scale, corresponding to the magnified image of the stage micrometer or hemacytometer rulings, is drawn upon a suitable card or sheet of paper placed on the table immediately to one side of the microscope. This card with various projected scales marked on it, constitutes the measuring device, and in each instance it should be inscribed with the value of its graduations in terms of each ocular, objective and tube-length combination employed.

Projection onto the improvised card scale, by means of the unoccupied eye, of an object seen through the eyepiece of a monocular microscope is the principle underlying this method. With the scale of the stage micrometer or hemacytometer in focus, the observer, after a little practice, should be able to see the point of a pencil or pen in writing position on the card. If both eyes are kept open, the ruled lines of the stage micrometer or image of object may be projected upon the card, the idle eye serving as a substitute for the external mirror of the familiar Abbé camera lucida designed to facilitate the drawing by projection of microscopic objects. The various divisions of the scale
appropriately subdivided, may then be compared with the dimensions of a microscopic object similarly projected.

To measure an object with this device, the card scale is placed in the position which it occupied when made (upon the table to the right or left of the microscope, as the case may be). The object on the slide is then projected upon the proper scale on the cardboard, and the space covered by it indicates linear dimension. If the counting chamber of a hemacytometer is employed in projecting this scale, the scale will be too approximate for very accurate measuring of objects of the dimensions of many protozoa, and should therefore be subdivided. If five subdivisions are made, each of these would have a value of 10 μ.

Tuttle (1935) has suggested that when blood cells are absent, lycopodium powder be sprinkled on the slide as a rough means of comparison. The particles of powder are of fairly uniform size, 30 μ in diameter. One must be cautious, however, not to employ too much powder. The lycopodium may be conveniently kept in a gelatine capsule and a faint cloud of it should be applied to a clean breath-moistened slide by gently scraping the moistened surface of the slide across the inverted cap or cover of the capsule. To do this, first shake the capsule, then remove the cap, invert it and apply the slide as described. Finally a suitable suspension in physiologic salt solution of the material to be examined should be placed upon this prepared surface and comparison of its size made with that of the lycopodium grains.
APPENDIX III

CONSTITUENTS OTHER THAN ANIMAL PARASITES OR THEIR DERIVATIVES COMMONLY FOUND IN STOOLS

In the microscopic examination of stools for evidences of the presence of animal parasites, confusion may arise from mistaking various objects present for protozoa. These irrelevant bodies fall into three groups: (1) cells derived from the tissues of the host, such as epithelial cells, mucous cells and histiocytes; (2) objects of animal and vegetable origin derived from food; (3) bacteria, yeasts, and various kinds of fungi and molds, particularly their spores.

CELLS DERIVED FROM THE TISSUES OF THE HOST

These cells come from two main sources, namely, the epithelial lining of the gut, and the blood and lymph. The latter, notwithstanding differences in the intestines from those of the plasma, in respect to medium, osmotic pressure, and so on, present appearances which may be strikingly similar to protozoa. The larger cells, such as those of the reticulo-endothelial system, with their power of ameboid movement and their phagocytic activity, including their ability to ingest red cells, may be mistaken for amebae even by an observer of some experience. Leucocyte cells often remain unchanged in stools for a considerable time. Red blood corpuscles, also, when crenated and distorted may occasionally prove to be a source of confusion.

Diarrheic and dysenteric stools present the greatest difficulties to the beginner in fecal microscopy. This is because of the numerous cellular elements they may contain, which come largely from
PSEUDOMORPHS IN STOOLS

the walls of the digestive apparatus. These cellular elements frequently have little resemblance to normal tissue cells, since they are usually in an advanced stage of degeneration when passed.

Common cells from the gut wall, which may be mistaken for dead protozoa and possibly, at times, for cysts, are the following:

1. Detached and degenerating columnar epithelial and goblet cells, which are often present in mucus from the gut wall or merely isolated in the stool itself.

2. Endothelial cells from the blood vessels in inflamed areas, sometimes containing erythrocytes and other inclusions which causes them to be mistaken for dead trophic forms of E. histolytica.

3. Squamous cells seen in stools often puzzle the beginner owing to their large size and their resemblance to some textbook pictures of amebae. Their outlines may be irregular (ameba-like or ameboid) and they may possess a clearly visible ringlike nucleus. Such cells are usually to be found on the surface of solid stools, but may also occur mixed with the feces in soft or fluid specimens. They can be distinguished readily by their centrally placed nucleus, the small bright granules in the cytoplasm, their lack of motility and their flattened scalelike form, which is un-

![Microscopical constituents of feces, × 250 app. (v. Jaksch.)](image-url)
like the more or less rounded globule of protoplasm of the ameba. The shape can often be determined by gently tapping the cover glass which frequently causes the cells to turn edgewise.

These different kinds of cells occur in varying degrees of colitis. Cells from typical amebic and bacillary dysenteric stools are shown in Figures 7 and 8.

**Objects of Animal and Vegetable Origin Derived from Food**

Derivatives of animal and vegetable origin are naturally common in stools. Fat globules in the form of colorless, highly refractile droplets of various sizes, at first sight, often suggest cystic bodies. Spicules (crystals) of fatty acids may sometimes be observed in such globules, or they may occur separately or in sheaf-like formations. Paraffin oil, commonly used as a laxative, appears in stools in the form of droplets of various shapes and sizes, but in most instances, the amount is too large to be more than a source of annoyance. Soaps formed from fats ingested with food may appear in acicular crystals, or in somewhat rounded masses, more or less amorphous in appearance, sometimes with a thick border, which may be suggestive of a cyst to an inexperienced observer.

Muscle fibers are commonly observed in microscopic stool examinations. Their appearance depends largely on the amount of digestion which they have undergone. Shreds of muscle tissue are usually clear yellow or brown in color, long and more or less rectangular in outline, and their characteristic cross striations can be observed provided that digestion has not gone too far. Connective tissue fragments are also commonly found, but are not likely to be confused with other structures or substances, with the possible exception of mucous shreds.

Animal hairs and fibers of various kinds are not infrequently seen in feces. Hairs of mice, rats, rabbits, et cetera, and, more commonly, fibers of wool may be encountered. Animal hairs have a definite central zone or canal which appears darker because of the air-containing medullary cells. Wool fibers have no central medullary cavity, and may be recognized by the irregularity of
the surface scales. In contrast, fibers of cotton appear as twisted, flattened filaments. Scales of fish, and so on, may be seen occasionally but can scarcely be confused with parasites or their derivatives.

Objects of a vegetable nature derived from food often make up a considerable part of human excrement. To the fecal microscopist, the most important among these are probably the fungal spores which are discussed briefly in connection with bacteria and fungi (page 390). Grains of pollen (round or oval in outline) can be readily recognized, generally by their external roughness and frequent irregularity. One very common pollen of distinctive nature is that of ordinary broccoli (broccoli), which is frequently three-lobed, nodulated, and dense brown in color. At times its appearance may suggest an egg of *Ascaris lumbricoides* although it is considerably smaller in size than an ascaris egg.

Another spore which may be encountered in feces is that of *lycopodium* which is also characterized by its frequently trilobed form, brown color, and prickled surface. The spores of truffles may be mistaken for ascaris eggs owing to their size—66 by 42 μ.

The common brown rust of wheat (*Tilletia tritica*) is spherical, brownish in color, and with pits or depressions on its surface remotely suggestive of a golf ball.

Vegetable hairs, coming most commonly from wheat, are elongated objects of a somewhat faint greenish hue, pointed at one extremity and swollen into bulbous form at the other. The walls are thick and the central channel clearly discernible. These will often be found associated with other vegetable hairs, with fragments of the pericarp of the cereal grains, composed of thick walled cells, hexagonal in outline. Similar debris from rice, beans, peas and potatoes, is frequently seen.

The remains of banana pulp, with the resin canals standing out prominently, are commonly encountered in feces. Cellular remnants of orange pulp and fragments of the epidermis of various fruits are sometimes to be seen. Finally, the thick cellulose walls of vegetable cells, and vegetable spirals of varying thicknesses and widths are quite common (a fragment of the latter, lying
flat on the slide may simulate a cyst except that the thickness of the wall is usually distinctly greater than that of a protozoan cyst wall. As a general rule, it may be said that the walls of vegetable cells are much thicker than those of cells of animal origin.

In Figure 94 are shown a few examples of various types of cells of animal and vegetable origin. These illustrations will give the reader a clearer idea of the appearance of such cells than verbal descriptions alone can convey. Once such cells are recognized, their presence in stools may thereafter be ignored.

**Bacterial and Fungal Floras of Stools**

Bacterial and fungal floras found in stools form an extensive subject which can only be entered into very briefly. The most important of these floras are the enterococci and the colon group of bacteria, and the spirochetes (*Treponema eurygyrata* and *stenogyrata*, particularly). Some members of the enterococcus and colon groups, as well as the two spirochetes mentioned, occur commonly in the stools of normal persons. These spirochetes frequently appear in large numbers in the mucus expelled from cases of chronic colitis. Some observers have credited them with some degree of pathogenicity; but most investigators believe that their presence is dependent on the fact that the mucus provides excellent nutriment for their growth and rapid multiplication, and therefore, is not significant.

Among the yeasts and various kinds of fungal spores,* Blastocystis hominis* (Fig. 4) (probably classifiable under the blastomyces or saccharomyces) is the only organism which may prove a formidable source of confusion. This yeastlike fungus, is not known to occur naturally outside of the animal intestine. It is sometimes difficult to distinguish its cysts from those of degenerating intestinal protozoa which often show a large central vacuole. When rapidly multiplying, as is sometimes the case in diarrheic

*Fungal spores are usually oval or round and may be confused with the smaller cysts. In properly stained preparations, however, they usually appear very dark in color and show little or no structure suggestive of protozoa. Starch granules may also be encountered frequently, either free or in their thin cellulose capsules. A blue reaction to iodine reveals their character.
and dysenteric stools, this organism of which there are probably several species, may show a great variety in form and size. The typical cell has a large central zone filled with a somewhat clear refractile substance which often takes up iodine readily, and for this reason some authors think that it may be glycogenic in nature. Extending around the periphery in a semilunar-like fashion is a narrow cytoplasmic zone, sharply separated from the central portion, which contains one or more fairly large refractile granules.

As a rule, in fresh saline preparations, the whole cell of *B. hominis* has a characteristically pale greenish appearance, similar to cysts of the significant *Endamoeba histolytica*, although on the whole it appears to be less refractile. The granules stand out by virtue of their relatively greater density. According to Lynch (1930) the peripheral zone, the groundwork of which is cytoplasmic in nature, may be transparent and colorless, while the central portion appears to be greenish and refractile or vice versa. When undergoing rapid growth, as in culture, there may be observed frequently an enveloping clear, gelatinous, slightly refractile substance which at times is as thick as the diameter of the cell itself. The refractile granules generally so distinctive in the cytoplasmic rim of *B. hominis*, are of two different kinds, one of which is evidently nuclear in nature, as revealed by staining, while the other is of the nature of "volutin" and is usually more distinct both in fresh and stained preparations. Volutin, as commonly found in both bacteria and yeasts, is generally thought to be a reserve substance, probably nucleic acid or a nucleic acid compound different from that of the nucleus. In culture these granules, at first, appear to be scanty but in older cultures they frequently appear in larger numbers, and finally seem to be used up or disappear in the course of multiplication.

*B. hominis* may be no more than 5 or as much as 20 or more microns in diameter. It can usually be found in varying numbers in the majority of stools examined microscopically. It reproduces both by budding and by fission and grows rapidly in diarrheic stools or suitable culture media, such as that employed in growing
E. histolytica. When rapid growth is taking place, very large and most bizarre forms frequently appear. At times, according to Alexeieff (1911), forms with numerous nuclei develop and the cytoplasm apparently concentrates around these nuclei, eventually producing a number of daughter forms within the original cyst membrane. The complete life cycle is not at present clearly understood.

This organism, as already mentioned, is a widespread inhabitant of the intestines of many animals, including that of man. It is not known in a free-living state and has rarely, if ever, been grown in pure culture. Its growth is undoubtedly increased by inflammatory states of the gut, and from time to time it has been accused of pathogenic tendencies. I have observed its close association with rectal ulcers in particular instances in which it appeared to be continually present in a rapidly growing state.

When one is familiar with the appearances and structure of this vegetable organism, and is constantly on the look-out for it, little confusion as to its identity should arise. However, it must be remembered that it may simulate various cellular and other components of stools, particularly those of a cystlike nature. Unlike amebae, it may be fairly well stained in thin smear preparations by ordinary blood film methods, the peripheral zone, nuclei, volutin granules and central body usually being well differentiated. In iron-alum hematoxylin preparations, the central body is usually dark, the peripheral zone lighter, and the nuclei and volutin granules more or less deeply stained according to the amount of differentiation employed.
APPENDIX IV

STAINING METHODS FOR PROTOZOA

Heidenhain’s Stain

The method of staining with hematoxylin introduced by Heidenhain in 1892 is still a standard cytological technique. It is applicable for study of both nuclear and cytoplasmic cell components. Smears should be prepared in the manner described in Chapter I (pages 7, 9), and placed immediately into a warm fixative, avoiding drying of the preparations which renders them unsuitable for staining. The various steps in the standard Heidenhain iron-hematoxylin procedure are as follows:

1. Fix the smear in body-warm Schaudinn’s solution* for from ten to twenty minutes.
2. Rinse the slide thoroughly in 50 per cent alcohol.
3. Immerse the slide in 70 per cent alcohol for several minutes.
4. Immerse in 70 per cent alcohol containing iodine (enough to give a port-wine color to the alcoholic solution) for not less than five minutes. (This procedure is for the purpose of removing the unused mercury salts of the fixative.)
5. Pass the slide successively through 70 per cent alcohol, 50 per cent alcohol (allowing at least two minutes for each immersion), and water (preferably distilled) to remove all traces of alcohol.
6. Mordant in from 2 to 4 per cent fresh aqueous solution of iron-alum (ferric-ammonia sulphate) for from six to twelve hours.

* Saturated aqueous solution of mercuric chloride, 2 parts; 95 per cent ethyl alcohol, 1 part; glacial acetic, 2 to 5 per cent, added just before using.
7. Rinse in distilled water for a few seconds.
8. Stain in 1/2 per cent aqueous solution of well ripened hema­
toxylin for from six to twelve hours.
9. Rinse in distilled water for a few seconds.
10. Differentiate by placing the slide in a 1 or 2 per cent fresh
aqueous solution of iron-alum (same as employed in mordanting)
and arrest action of the alum by rinsing freely in tap water when
sufficient decoloration has been achieved as judged by direct
microscopic inspection of the stained organisms. (To prepare a
slide for such inspection, it should be rinsed in distilled water and
a suitable cover slip applied after removal of excess water, not
allowing the film to dry at any time.)
11. Dehydrate by passing successively through 50 per cent, 70
per cent, 85 per cent, and absolute alcohol allowing from two to
five minutes for each alcohol.
12. Clear by placing first in equal parts of absolute alcohol
and xylol and then in pure xylol allowing about five minutes for
each.
13. Mount the slide in xylol-balsam or gum Damar, and place
it (coverglass uppermost) in an incubator until the mounting
medium hardens. This may require a number of days.
In the finished slide, the chromatin of the nuclei should be
black, the cytoplasm in shades of gray and the chromatoid bodies
black.
If successful results are to be obtained with the Heidenhain
staining technique, several precautions in particular should be
observed:
1. Smears should be made fairly thin and as uniform in thick­
ness as practicable. The material should be daubed rather than
rubbed on the slide, and emulsified in a small droplet of physio­
logic salt solution if necessary to produce the desired results.
2. Before mordanting and staining, alcohol should be com­
pletely removed from smears by washing them with water.
3. The smears should not be permitted to dry at any time
throughout the whole staining procedure. Special caution is ne­
cessary after the slides are withdrawn from the xylol.
STAINING METHODS FOR PROTOZOA

Staining vessels of various makes may be employed. Coplin jars, or rectangular dishes with sides grooved to accommodate twenty slides, are satisfactory.

Coverglasses may be used instead of slides for making smears. If this is done flat vessels such as petri dishes should be substituted for the coplin jars, and the coverglasses placed in them, film-side up except when fixing; while being fixed, the coverglasses should be floated on the surface, film-side down. The special small staining vessels similar to coplin jars that are now obtainable commercially, simplify this procedure.

An alternative method for differentiation may be done on a time basis, as described in Part I, for the rapid method of iron-hematoxylin staining. After the slide has been removed from the stain and rinsed in water, dehydration is accomplished by passing the slide through ascending strengths of alcohol up to 95 per cent, and then differentiating in a saturated alcoholic solution of picric acid for from four to seven minutes, depending upon the thickness of the smears. With a little experience, the probable time required for proper differentiation may be judged without resort to use of the microscope. When the organisms are few, as is not infrequently the case in chronic amebic cyst carriers, elimination of the microscope as a means of control of differentiation offers obvious advantages.

After the slides have been washed in several changes of 95 per cent alcohol to remove the excess picric acid, they should be placed in 95 per cent alcohol containing a crystal or two of potassium acetate to each 40 or 50 cc. of alcohol, in order to restore the original bluish color. From this point on, the procedure may follow the outline given for the rapid method of iron-hematoxylin staining (page 12) or for the regular Heidenhain method described above.

**DOBELL'S MODIFICATION OF MANN'S STAIN**

Mann's staining technique, as modified by Dobell (1919), has the advantages of being comparatively easy to carry out and of giving excellent pictures of both trophozoites and cysts of intesti-
nal protozoa. It furthermore stains red the erythrocytes ingested by *E. histolytica* in acute amebiasis. The usual procedure is as follows:

1. Fix the smear in body-warm Schaudinn's solution for from ten to twenty minutes.
2. Rinse the slide thoroughly in 50 per cent alcohol.
3. Immerse in 70 per cent alcohol for several minutes.
4. Immerse in 70 per cent alcohol containing iodine (enough to give a port-wine color to the alcoholic solution) for not less than five minutes.
5. After rinsing in 70 per cent alcohol to remove the excess of iodine, immerse the slide successively in 80 per cent and 90 per cent alcohol, allowing it to remain in each solution for about ten minutes.
6. Immerse the slide in distilled water for ten minutes.
7. Place the slide for from four to twelve hours in stain made according to the following formula:

\[
\begin{align*}
\text{Aqueous solution of methyl blue (1\%)} & \quad 35 \text{ cc.} \\
\text{Aqueous solution of eosin (1\%)} & \quad 45 \text{ cc.} \\
\text{Distilled water} & \quad 100 \text{ cc.}
\end{align*}
\]

8. Rinse thoroughly in distilled water.
9. Differentiate, controlling the degree of decoloration desired with the microscope, in 70 per cent alcohol containing about 10 drops of a saturated aqueous solution of orange G to 100 cc. of the alcohol.
10. Rinse in distilled water.
11. Dehydrate rapidly in ascending strengths of alcohol, i.e., 30 per cent, 50 per cent, 70 per cent, 85 per cent, 95 per cent and absolute alcohol; clear in xylol and mount in xylol-Canada balsam or gum Damar.

**Polychrome Methylene Blue Staining Methods**

Various modifications of the original Romanowsky polychrome method of staining have been introduced from time to time under the names of the proponents. Best known among these stains
are those of Jenner, Leishman, Giemsa, Hasting, Wilson, and Wright. Except in Giemsa's method, a simple solution of the staining substance in pure methyl alcohol is employed. No previous fixation of the smear is required since the methyl alcohol of the staining solution (applied directly to the slide) will accomplish this if allowed a minute or two before the subsequent dilution with distilled water free of CO₂ (pH of 7.0 or slightly more). The exact amount of dilution and time required for staining varies and is best judged by successive trials. However, a fourfold or fivefold dilution and from four to six minutes' staining will serve as a basis.

Giemsa's stain consists of a mixture of azur II eosin and azur II powders dissolved in equal parts of pure methyl alcohol and glycerin under prescribed conditions. Prior to staining, fixation of the film or smear in absolute methyl alcohol for from one to five minutes and rapid drying in air is necessary. Immediately before use one part of the stain should be diluted with from ten to fifteen parts of CO₂-free distilled water (pH 7.0 or slightly more). Staining time is from thirty to forty minutes or even more, as judged by microscopic inspection. Alkalinity of the diluent increases the intensity of the stain. It also tends to reveal better the characteristic dots of infected red cells in both benign and malignant tertian malaria, and to stain spirochetes more satisfactorily. Demonstration of Treponema pallidum usually requires from two to twelve hours' staining.

The "panoptic" or combined Giemsa stain is believed by some investigators to give even better results than the Giemsa stain alone. The air-dried film is first stained on the slide with one of the other Romanosky stains such as that of Jenner, Leishman, or Wright. After rapid washing with CO₂-free distilled water and without further treatment, the film is again submitted to staining by the Giemsa method.
APPENDIX V

CULTURE METHODS FOR PROTOZOA

INTESTINAL PROTOZOA

Various types of media have been devised for the culture of protozoa; the most generally used (for E. histolytica, particularly) have been those of Boeck and Drbohlav, and Cleveland. These media consist of a solid portion of coagulated egg or special nutrient agar in the form of a slant, preferably without butt; a growth fluid filling the culture tube to about the top of the slant; and sterile "whole" rice flour, which settles down into the angular space at the bottom of the culture tube between the slant and the wall of the tube. The growth fluid contains diluted horse or human serum (Cleveland) and the rice flour is finely ground from whole rice (Slade). Detailed instructions for preparing these and other special protozoal culture media follow.

BOECK-DRBOHLAV MEDIUM (L.E.S. MEDIUM, LOCK-EGG-SERUM)

Boeck (1921) originally introduced the use of the coagulated egg slant supplemented by the addition of dilute Lock serum (human). Drbohlav (1925) later substituted a 1 per cent solution of crystallized egg albumin (L.E.A. medium) for the diluted serum of Boeck, and later still the addition of sterile whole rice or wheat flour were introduced. Horse serum is now quite generally employed instead of human serum, as in the original Boeck (1921) medium.

The standard method of preparing the Lock-egg-serum-media is as follows:

1. Wash four clean fresh eggs thoroughly with a sterile nail brush and 70 per cent alcohol and allow them to remain in the
alcohol for one-half hour. Then dry them rapidly in the air, observing sterile precautions, and blow out the contents into a sterile beaker. To do this, make a hole in both ends of each egg; one hole should be small, just large enough to accommodate the conical end of a small bore sterile pipette, and the other larger, to facilitate exit of the contents. These holes may be satisfactorily made with sterile, stout, fine pointed tweezers. After inserting the end of the pipette into the smaller opening, it is possible, with a little practice, to blow the contents through the larger opening into a sterile beaker.

2. Maintaining sterile conditions, emulsify, in the beaker containing them, the egg yolks and albumen with 50 cc. of sterile Ringer, Lock, or similar solution, preferably employing a suitably sized, sterile egg-beater.

3. Under sterile conditions, fill sterile test tubes with enough of the sterile whole egg emulsion to produce slants without butts, which measure about 1-1/2 inches in length after coagulation.

4. To solidify the egg emulsion place it in an inspissator which is kept at a temperature of 70° C. (158° F.) until coagulation is completed.

5. The emulsion is then sterilized in an autoclave at 15 pounds pressure for twenty minutes.

If an inspissator is not available, satisfactory solidification and sterilization of the egg emulsion may be accomplished in an Arnold sterilizer or autoclave as follows:

Arnold Sterilizer Procedure. Coagulate the emulsion at 80° C. and allow it to remain at this temperature for one hour; then store it overnight at laboratory temperature. The following day, place the emulsion in an Arnold sterilizer (kept at 85° C.) for one hour, and allow it to stand at laboratory temperature overnight. Repeat this procedure at a temperature of 75° to 80° C. on the third day. Cool in an incubator at 37.5° C. and allow it to remain there for two days to make certain that it is sterile.

Autoclave Procedure. Turn the steam into the outer chamber of the autoclave until the jacket is hot, place the tubes in a suitable slanting position in the sterilizing chamber, close the door
and see that the vacuum exhaust valve is also closed. Then turn
the steam into both chambers of the autoclave and open the out­
side exhaust valve. At the first appearance of steam from the lat­
ter valve, close it and allow the pressure to climb to 15 pounds;
at this time shut off the steam and allow the pressure to decline
slowly to 0 on the dial, or that of the atmosphere. Repeat this
procedure on three successive days, storing the media at room
temperature between sterilizations.

Prior to use, fill the coagulated egg slant tubes to the top of the
slant with the solution employed in emulsifying the eggs (Ringer,
Locke, etc.) to which from 6 to 10 per cent sterile horse or human
blood serum has been added. Then add a small amount of sterile
whole rice flour (as much as can be transferred on a warm 5 mm.
wire loop or in the end of a 1 cc. wide bore bacteriologic pipette),
gently shaking the tube so that the flour will settle to the bottom
and not remain sticking to the wall above the liquid.

CLEVELAND’S LIVER INFUSION AGAR

After experimenting with about a dozen different kinds of slant
and growth liquids in various combinations, Cleveland and his
associates (1930) came to the conclusion that for slants Loeffler’s
dehydrated serum and liver infusion agar gave the most satis­
factory results; and for growth fluids, horse serum saline (1:6)
and 3 per cent solution of hydrolyzed hemoglobin. Liver infusion
agar* with horse serum saline and whole rice flour was the com­
bination which he preferred.

* The Difco Laboratories (Detroit, Michigan) now supply a special brand of
this liver infusion agar made according to Cleveland’s formula and known as “Bacto
Entamoeba Medium” (dehydrated). The ingredients of this and the method of
preparation are as follows:

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beef liver, infusion from</td>
<td>272.0 gm.</td>
</tr>
<tr>
<td>Bacto-peptone</td>
<td>5.5 gm.</td>
</tr>
<tr>
<td>Disodium phosphate</td>
<td>3.0 gm.</td>
</tr>
<tr>
<td>Sodium chloride</td>
<td>3.7 gm.</td>
</tr>
<tr>
<td>Bacto agar</td>
<td>11.0 gm.</td>
</tr>
</tbody>
</table>

Thirty-three grams of Bacto Entamoeba Medium are dissolved in 1000 cc. of
distilled water by boiling for a few minutes. The solution is then distributed in
test tubes as directed for egg emulsion, plugged and sterilized in autoclave for
twenty minutes at 15 pounds pressure (250°F). The sterile tubes are then re­
moved and cooled in an appropriate slanting position at room temperature.
Before use the tubes are filled to the top of the slant with fresh horse serum saline (1:10), and the amount of sterile finely ground whole rice flour that can be held on a 5 mm. wire loop is added to each tube. The rice flour may be sterilized in small quantities in test tubes at a dry heat of 150°C for one hour. Autoclaving at 15 to 20 pounds pressure for thirty minutes on two successive days appears also to sterilize the rice flour satisfactorily allowing the tubes to remain in the autoclave for awhile after opening in order to insure drying.

Inoculation of culture tubes may be done with a fairly large platinum loop (4 to 5 mm.). In case of formed feces a portion about the size of a split pea should be selected after the specimen has been well mixed or sampled. To secure sediment from culture tubes for examination or subculture, a suitable pipette of fairly large bore is to be preferred.

To expedite removal of material from culture tubes for examination and subculture, a special type of pipette may be made from ordinary moderately thick-walled 3/16 inch glass tubing. Anyone accustomed to drawing and bending glass tubing in a bunsen flame should be able, with a little practice, to make such pipettes without difficulty. The process is carried out as follows:

1. Cut the tubing into 15 inch lengths, and heat each piece in the middle in a blue flame, rotating all the time. When the tubing is sufficiently soft, remove it from the flame and quickly draw out the heated portion by pulling the two ends in opposite directions until a suitable diameter is obtained (not less than 1/16 inch internal diameter).

2. Alternately heat and draw out the thicker slanting parts (on either side of the drawn portion of the tube just completed) until they assume the shape of long bulbs. (This operation is not very difficult to perform, but to get uniform results, it requires a little practice.)

3. Before cooling and separating the two almost completed pipettes, and while the glass is still pliable, bring the bulbs and stems of the two nearly symmetrical halves into perfect alignment by rolling the whole tube on a smooth surface, such as a flat
board or, preferably, a flat sheet of asbestos paper or board.

4. To complete the two pipettes, separate them at the middle and anneal both ends of each.

Material should never be drawn into pipettes by sucking with the mouth. Proper use of rubber teats or bulbs insures not only greater safety but also better control in filling and emptying the pipette.

To secure material from culture for microscopic examination or for transplanting, the pipette should be manipulated as follows: After expressing a small quantity of air by pressure on the rubber teat, the pipette is inserted through the fluid portion of the culture into the growing zone or near the upper surface of the sediment. Then the pressure on the teat or bulb is relaxed sufficiently to allow the desired amount of sediment to enter the tip of the pipette without disturbing the remainder of the sediment in the culture tube. In the absence of a clear zone of growth above the rice sediment, a representative sample of the deposit should be removed. Finally the pipette containing the desired material is withdrawn and its contents expressed on to a slide, or beneath the surface of the fluid portion of a fresh tube of medium for subculture, as the case may be.

Blood and Tissue Protozoa

Flagellates

Of the various media which have been devised for cultivation of the leishmanias and trypanosomes, that known as the N.N.N. medium (Nicolle, Novy, MacNeal) is probably the most satisfactory. The agar base of this medium is made according to the following formula:

Agar ........................................ 14 gm.
Sodium chloride (common sea salt) ...... 6 gm.
Distilled water ............................. 900 cc.

These ingredients are mixed and heated to the boiling point until the agar is completely dissolved, then made up to 1000 cc.
and while still fluid 4 or 5 cc. portions are placed in test tubes. These are then cooled to 48° C. and to the fluid in each tube, one third of its volume of defibrinated rabbits' blood is added. The two liquids are promptly mixed by rotating the tube, which is then slanted and the contents allowed to solidify and set. Cooling on ice gives more waters of condensation at the bottom of the tube, in which the organisms introduced may grow.

If the tubes are stoppered with cotton plugs, these should be well saturated with paraffin to minimize evaporation. Soft rubber caps or stoppers serve this purpose better. Before tubes are used, they should be tested for sterility, and those that prove to be contaminated, discarded. Hemoglobin contained in the defibrinated blood added to the melted agar appears to be essential in the cultivation of the various blood protozoa.

A 5 mm. loop or more or its equivalent of the suspected material should be introduced under sterile conditions into the waters of condensation at the bottom of the tube, and the tube incubated for several days or longer at 22° to 25° C. Fractions for microscopic examination may be withdrawn by means of a small bore pipette such as is employed in blood typing.

Rogers (1904) has been successful in growing the flagellate form of *Leishmania donovani* by employing the citrated blood itself as culture medium at an incubator temperature of 22° C. Incubation at this low temperature seems to be necessary for development of the flagellate form, higher temperatures shortening the period of growth and tending to favor degeneration of the organisms. A special method of concentration and segregation of the infective material for its cultivation on the N.N.N. medium has also been devised by Young and Van Sant (1923).*

Although *Trypanosoma gambiense* and *rhodesiense* may be kept alive on the N.N.N. medium for a short time and they may even multiply to some degree, their cultivation on this or other media has been unsatisfactory. On the other hand, Torres (1915) is reported to have been quite successful in growing *T. cruzi* at a

* A brief description may be found in Craig and Faust's Clinical Parasitology, 2nd ed., p. 665.
temperature of from 22° to 25° C. on a medium of beef broth containing 5 per cent peptone and 7 per cent sodium chloride. The pH of the medium varied from about 6.6 to 7.2.

MALARIA PARASITES

Bass and John (1912) have been able to cultivate malaria organisms in the blood plasma of patients with malaria. Their procedure is as follows:

Under sterile conditions from 10 to 20 cc. of the blood of a patient with malaria is withdrawn and placed immediately into a sterile centrifuge tube containing 0.1 cc. of a 50 per cent sterile glucose solution, and this mixture is defibrinated mechanically by the use of a glass rod or tube extending to the bottom of the centrifuge tube. The blood is then centrifuged until at least one inch of serum is left above the cell sediment. The tube containing this plasma and centrifuged sediment is placed in an incubator kept at a temperature of 40° to 41° C. and small portions of the very uppermost fraction of cell sediment are drawn up sterilely with a small bulb capillary pipette from time to time and examined microscopically. Parasites develop in this upper cell layer; but those in the deeper lying red cells die.

If it is desired to cultivate more than one generation of parasites, the upper layer of centrifuged sediment, which contains most of the leucocytes, must be carefully pipetted off since these cells promptly phagocytize merozoites. Otherwise only parasites within the red cells may survive. Sexual forms (gametocytes) are more resistant to phagocytosis. Estivo-autumnal (malignant tertian) parasites are more resistant to phagocytosis than are P. vivax (benign tertian) parasites.
In a comparative study of the various types of anal scrapers and swabs suggested for the procurement of eggs in the diagnosis of oxyuriasis, Hall (1937) proposed a new and more satisfactory one, which he calls the N.I.H. (National Institute of Health) swab. This swab consists of a glass rod passed through a perforated cork; the lower end of the rod, which should be well rounded, is covered with a square of absorbent cellophane preferably a little smaller than the coverglass to be used in mounting it for microscopic examination. This cellophane is known commercially as “plain, transparent” cellophane. The perforated cork (a rubber stopper may be substituted to advantage) should be of a size to fit snugly into a small or medium-sized test tube and the cellophane tip should be held firmly in place on the end of the rod by means of a small rubber band or a short section of suitable rubber tubing. These parts assembled are depicted in Figure 95.

The N.I.H. swab should be employed both as scraper and swab. Pinworm eggs readily adhere to the cellophane tip or are caught in its folds. Preparatory to examination for eggs, the cellophane tip should be released from the rod, placed on a slide and mounted in a drop or two of decinormal sodium hydroxide solution. A suitable pair of tweezers will permit this operation to be accomplished without contaminating hands, or objects other than the slide on which the cellophane square is placed. A drop or two of the sodium hydroxide solution should supply sufficient fluid to make a good mount when the coverglass is applied. Special precautions must be exercised to exclude air bubbles. Stains
in low dilution may be added to the mounting medium to improve visibility of eggs present.

After the cellophane is removed, the cork and rod assembly should be returned to the tube, and the forceps sterilized by passage through a flame. The whole unit must, of course, be thoroughly sterilized before it is again used in any way. These swabs may be used repeatedly, the cellophane tip being renewed on each occasion.

The degree of personal cleanliness and periodicity of appearance of the female worms about the external anal orifice must be considered when securing material for examination. Furthermore the number of eggs on a swab is not indicative of the number of worms present since one female worm may deposit or have released from her uterus many eggs, or practically all the eggs adhering to the region of the anus and perineum may be removed through bathing. The extent of infection may, however, be ascertained by direct count of the female worms after appropriate medication.
APPENDIX VII

METHODS OF CONCENTRATION AND QUANTITATIVE ESTIMATION OF CYSTS AND EGGS IN FECES

Concentration of Cysts

A number of techniques have been devised to concentrate and more or less segregate cysts present in feces. Three of these will be described.

DIRECT CENTRIFUGATION

1. Mix from 2 to 5 gm. of the fecal specimen with about ten parts by volume of clean water and strain through a single layer of cheesecloth into a suitable centrifuge tube or, preferably, a 10 cc. pyrex Wassermann serologic tube.

2. Centrifuge at a moderate speed for from thirty to sixty seconds, decant off the supernatant liquid, and again mix the sediment with clean water and centrifuge.

3. Decant off the supernatant wash water, mix with water again and centrifuge as before. Repeat this process until the wash water is fairly clear.

4. Mix the packed sediment with a small amount of physiologic salt solution or with water, and transfer one or two drops to a microscopic slide (gently placing an ordinary coverglass over it), and examine microscopically.

A similar preparation should be made in the same way, employing Lugol's solution as a diluent, and examined in like manner.
Sedimentation Methods of York and Adams *

1. Place a portion of feces about the size of a walnut in a mortar or other similar vessel and triturate with tap water until a fine emulsion has been obtained.

2. Mix this fecal suspension with about 500 cc. of tap water in a tall glass cylinder in which it is allowed to stand for fifteen minutes.

3. At the end of this time three distinct layers will be noticeable. On the top a sort of scum usually forms, at the bottom a considerable amount of sediment accumulates, while the larger middle zone consists of a fairly even suspension of fecal particles, which also contains many of the cysts. Remove the scum from the surface and disregard the sediment.

4. Syphon off the remainder of the fluid into a suitable smaller cylinder of 150 to 200 cc. capacity. Leave the debris at the bottom of the original cylinder and a little fluid above it untouched. Discard the scum and the sediment at the bottom of the cylinder.

5. Allow the fluid that has been syphoned into the second cylinder to stand overnight, in order that the cysts may settle to the bottom of the cylinder.

6. Syphon off the supernatant fluid, in step 5 and discard it, retaining the sediment at the bottom of the cylinder.

7. Wash the sediment obtained in 6 several times by agitating with water, centrifuging and decanting the wash water each time.

Alternative Method

1-4. Proceed as in steps 1 to 4 of preceding method.

5. Centrifuge the fluid from the middle zone.

6. Wash the sediment obtained by agitating with tap water and centrifuging as directed under 7 in the preceding method.

Second Alternative Method

1-4. Proceed as in steps 1 to 4 of preceding method.

5. Centrifuge the fluid syphoned off, from the middle zone.

CONCENTRATION-SEGREGATION METHODS

6. Shake up the deposit obtained with a solution of cane sugar of a specific gravity of 1.080 (app. 20 per cent).
7. Centrifuge at a high speed.
8. Remove the supernatant fluid, which contains the cysts, and dilute with four times the amount of water.
9. Centrifuge once more at a high speed.
10. Wash the deposit obtained several times by agitating with water and centrifuging.

ZINC SULPHATE CENTRIFUGAL FLOTATION

TECHNIQUE OF FAUST (1940)

1. Emulsify portion of formed feces about the size of a small pecan in from 5 to 10 cc. of clean lukewarm water.
2. Strain this suspension through one layer of cheesecloth (placed in a small funnel), and collect 10 cc. in a 10 cc. Wassermann tube.
3. Centrifuge this 10 cc. of strained liquid feces for from forty-five to sixty seconds at about 2500 R.P.M., pour off the supernatant water, break up the sediment by shaking or tapping with 2 or 3 cc. of clean water, and add water to fill the tube to within about 1 cm. of the top.
4. Repeat step 3 until the supernatant fluid is clear; three or four washings are usually required.
5. Pour off the supernatant fluid from the last washing, add 3 or 4 cc. of zinc sulphate solution of sp. gr. 1.18 (about 28.5 per cent granular U.S.P. ZnSO₄ + 7H₂O), break up the sediment as in steps 3 and 4 and add sufficient of this solution to fill the tube to within about 1 cm. of the top.
6. Centrifuge again for from forty-five to sixty seconds at top speed of about 2500 or more R.P.M., remove several loopfuls of the surface film, place on a microscopic slide, and examine for cysts.

This method of flotation may also be employed in concentration of eggs in feces excepting those of the Trematodes.
Concentration of Eggs

To concentrate, segregate, or estimate eggs in feces, the following techniques are useful.

WILLIS-MALLOY (1920) METHOD (ESPECIALLY FOR HOOKWORM EGGS)

1. Mix about 1 to 2 gm. of solid feces with ten times its volume of a sodium chloride solution, sp. gr. 1.150 (app. 20 per cent of table salt).

2. Fill brimful with this suspension a suitable (preferably flat) container such as a tin pillbox about 1-½ inches in diameter and 1 inch in depth. Then place on this brimful container a clean, grease-free glass slide of such size as to slightly more than cover it.

3. Allow the covered container to stand for ½ hour. Then carefully remove the slide keeping it in a horizontal position and turn it quickly to bring the under surface uppermost. In doing this none of the adhering brine should be lost. Most of the hookworm eggs present will be found in this fluid adhering to the glass surface.

While this method is reasonably effective in detecting both nematode and cestode eggs discharged in feces, it fails to float operculated eggs such as those of flukes and fish tapeworms. These eggs burst in the salt solution and do not rise to the surface film.

LANE METHOD

In the Lane Method (1932), known as direct centrifugal flotation (D.C.F.), a strong solution (sp. gr. 1.150) of common salt (NaCl) is employed for flotation, as in the Willis method for hookworm eggs already described. If 1 cc. of feces is used, this method lends itself to quantitative determinations for which it was originally intended. To obtain the best results, however, the apparatus required to carry out the procedure should be of the design recommended by Lane. The technique involved may, however, be carried out approximately by the use of the ordinary
CONCENTRATION-SEGREGATION METHODS

laboratory centrifuge (employing 10 cc. Wassermann serologic or ordinary conical urine centrifuge tubes) as follows:

1. Measure out 1 cc. of the fecal specimen and comminute or emulsify it thoroughly in water. The total volume should not exceed 10 cc.

2. Centrifuge for one minute at 1000 to 1500 R.P.M. and decant off the supernatant fluid.

3. Fill the tube containing the fecal sediment with the strong salt solution (sp. gr. 1.150–20 per cent sodium chloride) to within 1 cm. of the top and centrifuge again for one-half minute at the same speed as before.

4. Remove a drop from the surface by means of a 5 mm. wire loop in the manner described for the zinc sulphate centrifugal flotation technique for protozoal cysts (page 409) and examine microscopically under a low power objective.

This method of flotation on strong brine solution is suitable only for ancylostome and trichostrongulus eggs. Like the Willis-Malloy method, that of Lane fails to float operculated eggs, which burst in strong brine and fail to rise to the surface.

ZINC SULPHATE CENTRIFUGAL FLOTATION TECHNIQUE

This technique is the same as that described for protozoal cysts (page 409). It is questioned, however, whether this method of flotation is as satisfactory and as reliable for the concentration of eggs as Faust and his associates have shown it to be for protozoal cysts. At present it is the only practical method for concentrating both eggs and cysts in feces at the same time.

STOLL-HAUSHEER TECHNIQUE (1926) FOR QUANTITATIVE ESTIMATION OF HOOKWORM EGGS

1. Measure 4 cc. of feces by displacement in a suitably graduated glass cylinder or Erlenmeyer flask of about 100 cc. capacity marked at 56 and 60 cc. To do this fill the vessel to be used to the 56 cc. mark with an N/10 sodium hydroxide solution and then add feces until the fluid level reaches the 60 cc. mark.

2. Add ten small (3 mm.) glass beads, close the cylinder with
a rubber stopper and shake it vigorously until a homogeneous suspension is obtained. If the fecal specimen is hard, it may be necessary to allow the feces to remain in contact with the solution for several hours or more in order to secure satisfactory disintegration.

3. Transfer 0.15 cc. of the fecal suspension to the center of a 2 by 3 inch glass slide and cover with a 22 by 40 mm. No. 2 rectangular coverglass, or use two different portions of 0.075 cc. each of the suspension and employ two 25 mm. No. 2 square coverglasses.

4. Count all the eggs and multiply the result by 100 to get the number of eggs in 1 cc.-gram of feces. (Where two 0.075 cc. portions of fecal suspension of feces are employed, the average of the two counts may be chosen and the result multiplied by 200 to get the number of eggs in 1 cc.-gram of the original feces.)

This method is considered to be from 80 to 90 per cent accurate in the majority of specimens tested. However, consistency and other characteristics of the feces may lower this efficiency considerably. (Over a period of time, the variable daily output per female worm also must be considered in the comparative evaluation of consecutive egg counts.) Such a quantitative method is of particular value in estimating the so-called “worm burden” of an individual and in determining the efficacy of treatment.

Recently Otto et al. (1941) substituted zinc sulphate solution with a sp. gr. 1.18 for the strong sodium chloride solution (sp. gr. 1.15) employed in the method of Willis-Malloy. They found that their modification gave as good a concentration of hookworm eggs as the original Willis technique did, and was nearly as efficient as the regular zinc sulphate flotation method of Faust.

These workers believe straining the material through cheesecloth has little merit, and may actually reduce the efficiency of the zinc sulphate method. This opinion is in agreement with Lane's objection to screening. They also suggested that exposure of cysts to the hypertonic zinc sulphate solution for from twenty to thirty minutes instead of for one hour as stipulated by Willis, would probably increase the recovery of protozoan cysts. They stated, furthermore, that the greatest efficiency with the zin
sulphate solution is to be obtained by employing an apparatus similar to that described by Lane (1932) in which a coverglass is held firmly against the mouth of the tube while it is being centrifuged.
Fecal material constitutes a most fruitful hunting ground for protozoa, such as ameba, flagellates and occasionally a ciliate, Balantidium coli; and parasitic worms such as roundworm, hookworm, pinworm, whipworm and tapeworms. The blood offers a somewhat less extensive “range” in variety of fauna than the feces does, but from the standpoints of morbidity and mortality the parasites present in blood are of the greatest importance. Disease conditions in point are the malaria infections, the leishmaniases, and trypanosomiases.

Localization of parasites in other parts and special organs of the body are common. Some for instance as Trichomonas buccalis and Trichomonas vaginalis inhabit only the mouth and vagina, respectively, while others are to be found principally in certain tissues and organs. Examples of these are leishmania parasites in skin, mucous membrane, spleen and liver; filaria in lymphatic and connective tissues; larval forms of worms such as Trichinella spiralis in the musculature, Echinococcus scolices in the liver, and rarely the cysticercus of Taenia solium in the brain or other organs of the body.

Methods of collection, preparation, and preservation of parasitologic material vary rather widely. The procedures commonly used will be outlined in the following pages.

Protozoa in Stools

In collecting feces for the diagnosis of protozoa, great care must be exercised to secure the material in a condition or state most favorable for preserving the essential diagnostic characteristics.
PRESERVATION OF MATERIAL

of the parasites. This may at times appear to be too troublesome or nonessential. Nevertheless it is most important, if authoritative identification is to be obtained in the least time and at minimum expense. In this connection it should be remembered that the motile trophozoites of the various parasitic amebae, and trophic forms of the flagellates and ciliates, occur principally in fluid or semifluid stools. On the other hand, the protozoal cysts are usually found only in semiformald or formed stools.

When feasible, it is advantageous to have the stools passed in or near the laboratory shortly before inspection, and sampling. If this is impracticable, particularly when the patient is suffering from diarrhea or dysentery, the stool may be put into a suitable warm container. The latter in turn may be placed in a second vessel containing water at body temperature, both vessels covered to conserve heat, and transported immediately to the laboratory. If carriage to a considerable distance is necessary, a suitable wide-mouthed thermos bottle may be employed. However, warm water must be placed in the thermos bottle in contact with the specimen box, preferably in a suitably sized container similar to a hot water-bag. An improvised device of this sort, shaped to fit into the lower two-thirds of an ordinary wide-mouth thermos bottle, can be made readily from a section of bicycle inner-tubing.

Formed stools to be examined for cysts require no such special handling and may even be mailed in suitable cardboard containers, provided they reach the laboratory within two or three days of the time of collection. However, even here the fresher the stool the better. If it is desired to obtain motile trophozoites from persons passing formed stools, a mild saline cathartic such as sodium phosphate should be given, and mucous portions of subsequent stools (particularly the second one) should be examined immediately in a fresh warm condition. Likewise, stools once positive for cysts and subsequently apparently negative may again prove to be positive after a suitable laxative or a high tepid enema or a glycerine suppository.

The vessels or containers in which stools are collected must be scrupulously clean and should not contain antiseptic or fluid of
any kind. If possible, the whole stool should be available for inspection and discriminative sampling. Urine should not be voided into the same container as the feces. The presence of oil, barium or bismuth makes examination for motile trophic forms most difficult and unsatisfactory.

Methods of preparation for microscopic examination of fecal material and other body excretions or secretions suspected of containing protozoa may be summed up as follows:

1. Direct saline and iodine suspension. For details of this method see pages 3 to 7.

2. Permanent staining (a) for rapid modified Heidenhain method (pages 7 to 13); (b) for the regular Heidenhain and Mann methods, see Appendix IV.

3. Cultivation. For details of methods, see Appendix V.

4. Cyst concentration. For details of methods, see Appendix VII.

Preservation of protozoologic material in bulk is confined largely to that containing cyst forms, although Stone's (1935) staining in bulk process is satisfactory when applied to trophozoites of *E. histolytica*. Cysts of the amebae and other intestinal protozoa can be more or less satisfactorily preserved in feces thoroughly mixed with an equal volume of 10 per cent formalin. Coccidial cysts, likewise, may be kept for future demonstration or reference. The most satisfactory method of preservation, however, is that of hematoxylin staining on slides (pages 7-13 and Appendix IV).

**Protozoa in Blood, Organs, and Tissues**

Direct microscopic examination of fresh peripheral blood frequently reveals most significant information relative to the diagnosis of the malarias and trypanosomiases, and at times of visceral leishmaniasis. To obtain positive diagnostic evidence in the trypanosomiases, it is necessary also to examine the gland juice of enlarged lymphatic glands early, and the cerebrospinal fluid later in the disease. In visceral leishmaniasis, material secured by puncture from enlarged lymphatic glands, spleen, and liver is
PRESERVATION OF MATERIAL

often necessary before a microscopic diagnosis can be made.

Culture, and animal inoculation, in both leishmaniases and the
trypanosomiases, of suitable material from persons suspected of
having these diseases, constitute important accessory diagnostic
adjuncts.

Preparation of material for critical and differential microscopic
study, as well as for permanent preservation of the organisms, re­
quires the use of special staining methods. Although the finer
details of the structures of these organisms are to be seen best in
preparations fixed in Schaudinn's fluid and stained with iron­
hematoxylin (pages 7-13) this procedure is ordinarily not neces­
sary in diagnostic examinations of blood and tissues. One of the
well known Romanowsky stains such as Hasting's, Leishman's,
Wright's or Giemsa's fulfills all but the most exacting require­
ments. Details of the preparation of materials and staining by
these methods are given in Appendix IV.

HELMINTHIC EGGS AND LARVAE IN STOOLS

Feces offer the most important source of evidence of worm in­
fecations. It should be remembered, however, that eggs (Fig. 41)
of the pinworm or seatworm (Enterobius vermicularis) are not
commonly found in feces of infected persons; perianal and peri­
neal swabbings constitute more certain and satisfactory means of
securing the eggs for identification and preservation. Taenia eggs,
likewise, may not be found in stools of persons passing the mature
proglottids. Therefore, the number of Taenia eggs found in stools
may have little relation to the number of parent worms infecting
the host. This is also true of Ascaris lumbricoides. In hookworm,
strongyloid, and Trichura (Trichocephalus) infections, however,
there is some degree of co-ordination between the egg count and
the probable number of adult worms present in the intestines.

Preparation of the feces for microscopic search for the eggs of
worns consists in emulsifying on a glass slide one or more small
representative portions of the fecal sample in one or two drops
of physiologic salt solution. The emulsified material should be
spread so that the ready passage of light is permitted. Some inves-
tigators prefer not to use a coverglass when employing the low power objective; but use of a higher objective ordinarily requires a coverglass over the preparation. The oil-immersion objective is rarely necessary for diagnosis of eggs or larvae in feces.

Eggs and larvae in feces are probably best preserved in formalin solution, in which the feces have been thoroughly comminuted with an equal volume of 10 per cent formalin (4 per cent formaldehyde) solution. Slide mounts of washed, centrifuged sediment may be made in glycerin jelly or in carbol-xylol ringed with melted parafin or gold-size.

The various methods for concentrating eggs and larvae and segregating them from fecal material are briefly discussed in Appendix VII. When an accurate count is desired, the Stoll technique is recommended. Helminth eggs, particularly those of the roundworms, may be isolated from a representative sample of fecal polluted soil by treatment of a 5 to 10 gm. portion with 10 cc. of 90 per cent "antiform," for one hour in a 50 cc. centrifuge tube, stirring frequently.* The tube is then filled with sodium dichromate solution (sp. gr. 1.35), the whole thoroughly mixed by shaking and then centrifuged at 1000 R.P.M. for two minutes. After allowing the tube to stand for from ten to fifteen minutes, the surface film is transferred by loop or pipette to an ordinary centrifuge tube with conical bottom, the tube filled up with distilled water, shaken, centrifuged, and the sediment examined for eggs.

The eggs of *Schistosoma mansoni* and *japonicum* may be concentrated, according to Faust, (1939) by diluting the feces freely in water, pouring the material into conical glasses similar to those used in urine analysis, allowing it to settle, and then examining the sediment for eggs. Several such decantations and washings may be required; the total time allowed for sedimentations should not exceed from four to six hours to avoid hatching of the miracidia.

Alternative methods, in which hatching of the schistosome miracidia is taken advantage of, are carried out as follows:

1. The washed sediment is suspended in clear water in a glass

*This is an adaptation of the Caldwell and Caldwell egg-count technique (1926) in which antiform and sugar solution (sp. gr. 1.230) are employed as substitute for the decinormal NaOH used by Stoll and Hauscheer (see pages 411, 412).*
PRESERVATION OF MATERIAL

cylinder or flask, and allowed to stand for from twelve to twenty-four hours to permit hatching of miracidia. According to Faust and Meleney (1924), the free-swimming miracidia of *S. mansoni* (Fig. 60, b) and *S. japonicum* collect near the surface of the water while those of *S. haematobium* are equally distributed and all may be recognized under low power magnification, or even with a good hand lens. However, possible confusion with free-swimming infusoria must not be overlooked.

2. In the procedure devised by Fülleborn a small quantity of feces, the size of a hazel nut, is placed in a conical glass. This is emulsified in a 2.5 per cent salt solution and put aside in the dark to settle for five minutes. At the end of this time the solution is poured off from the sediment. After two or three repetitions of this procedure, the final sediment thought to contain schistosome eggs should be flooded with distilled water, heated to 120° F. (48.9° C.), and exposed to bright light. The embryonic free-swimming miracidia will soon escape from viable eggs, and may be seen readily with a good hand lens.

Roundworm larvae, such as those of *strongyloides* and the hookworms, may be isolated readily from feces by the following culture method: Mix the feces thoroughly with an equal amount of powdered animal charcoal diluted with enough water to facilitate forming the mass into a small round wafer. This should be of such size that it can be supported on several thicknesses of circular filter paper in a deep petri dish, leaving a clear space of about ⅛ inch between the periphery of the circular filter paper base and the wall of the dish. The wafer should be kept moist, and at the time of collection enough water provided in the free space around the filter paper so that fluid can be drawn up in a small pipette for inspection or harvesting (the larvae particularly in the filariform stage readily migrate from the culture into the clear water at the periphery).

Brief mention also should be made of the *Baerman* (1917) technique for isolation of nematode larvae from soil. The principle underlying this method is based on the fact that nematode larvae will migrate from soil into water kept at a somewhat higher
temperature when this water is brought in contact with the lower surface of the soil. The apparatus required consists of an ordinary glass funnel (preferably ribbed), with a short rubber tube and pinchcock attached to the stem. A basket or sieve somewhat smaller than the diameter of the funnel (with bottom of 1 mm. mesh bronze screening lined with cheesecloth to hold the soil sample) is placed within the funnel resting in a horizontal position.

The soil is spread over the screen-bottom of the basket or sieve supported on the sloping sides of the funnel. The funnel is then filled with lukewarm water so that the lower layers of the earth are bathed in it to a slight degree. In from ten to fifteen minutes, larvae derived from feces contaminating the soil, as well as free-living soil larvae, may be observed settling down into the stem of the funnel. After an hour the majority of these larvae will have migrated downward into the funnel's stem; the water may then be allowed to escape into a suitable centrifuge tube by releasing the pinchcock, and centrifuged; or allowed to stand to sediment; the supernatant water in either case is pipetted off and discarded. The sediment contains the larvae which were in suspension. Obviously differentiation must be made between the parasitic and free-living larvae of the soil.

**Adult Worm Parasites in Stools**

Intestinal parasitic worms or portions of them may be found at times in stools naturally passed. More often, however, they are seen after treatment, following a purge or at autopsy. In this connection there are several points worthy of note:

1. The gravid females of the pinworm or seatworm (*E. vermicularis*) migrate through the anus (mostly at night) to deposit their eggs upon adjacent parts of the skin. They are often passed in feces, particularly after a purge.

2. Mature and immature ascaris worms are frequently passed spontaneously and may at times even escape through the mouth or nares.

3. Tapeworm segments, singly or in chains, are discharged at intervals with the feces.
4. In nematode, tapeworm, and similar infections, all the stools should be saved for at least twenty-four hours, following anthel­mintic medication, and in tapeworm infections, the head or scolex should be looked for particularly.

5. Follow-up examinations of feces for eggs are most important in all intestinal worm infections, including liver flukes infecting man as well as the schistosome infections.

Most helminths (after thorough washing to free them of debris and mucus) may be fixed satisfactorily in 70 per cent alcohol containing 10 per cent formalin (4 per cent formaldehyde), and they may be stored in the same solution.

Small roundworms and the delicate flukes should be washed in physiologic salt solution in a test tube and fixed by dropping into 5 per cent formalin or 70 per cent alcohol containing 5 per cent glycerin heated to 60° or 70° C.

If they are fixed in formalin, the specimens may be placed in lactophenol diluted with an equal volume of water for several hours and then transferred to pure lactophenol* for mounting. The coverglass of the completed preparation may be ringed with paraffin (m.p. 60°) or gold-size.

If fixed in 70 per cent alcohol containing glycerin, the alcohol should be allowed to evaporate after transfer to an open dish in a 37° C. incubator. The specimens, remaining in pure glycerin, may then be mounted in glycerin jelly, preferably in a concave slide and ringed with paraffin or gold-size.

When large tapeworms are desired for future demonstration, they should be allowed to die and then placed in the desired position before fixation in 5 per cent formalin. Where retention of the natural color is desired, the well known Kaiserling technique may be employed.

Arthropods

The collection of arthropods varies according to the particular group. Many may be transported in the natural state, packed in

* The lactophenol consists of two parts of glycerin and one part each of distilled water, crystallized carbolic acid and lactic acid.
tissue paper and placed in suitable containers, without any further preparation.

Adult fleas on animals such as the cat, dog, or rat may be killed or stupefied by contact with a camel’s hair brush moistened with xylol; they may then be removed with fine pointed tweezers to a vial. Rats caught in traps may be placed in a tight chamber and chloroformed to obtain mites, lice, and fleas. Larvae of fleas may be readily collected by sieving the material swept from floors of buildings occupied by infested animals. Bedbugs can be obtained by careful search of infested buildings.

Blood-sucking winged insects can often be trapped while feeding by carefully placing a wide-mouthed bottle or jar over them, and when their meal is interrupted or terminated, sliding a piece of cardboard over the mouth of the bottle. The container may then be capped or corked, and the insect killed, or removed alive if desired. Sometimes this same method may be employed successfully when such diptera are perched on furniture, walls, and so on. However, a special funnel-mouthed device made of celluloid or glass similar in principle to a funnel-mouthed fishnet or trap (from which the air may be partially exhausted through use of a large rubber bulb) constitutes a much more effective piece of apparatus. Suitable types of insect nets are also obviously useful in catching small as well as large winged insects.

Eggs, larvae, and pupae of insects may be brought indoors to mature under suitable conditions, and the adults identified and employed experimentally if desired. This method may be applied effectively in the investigation of mosquito breeding places as basis for planning and conducting logical control measures.

Except for very small winged insects, freshly killed arthropods are often pinned and mounted in closed celluloid or glass vials or in special insect boxes. Wingless forms, such as mites, ticks, centipedes, millipedes, spiders and scorpions may be satisfactorily preserved in 70 per cent alcohol. Ticks and spiders, particularly, shrink badly if allowed to dry out. Large arthropods with hard resistant exoskeletons may be killed with chloroform or by placing them in a “cyanide bottle” or jar; and preserved in 70 per cent
alcohol, or dried, thinly shellacked, and mounted in a suitable
manner.

Arthropods should not be stored in formalin since this pre-
servative tends to cause disintegration of the exoskeleton. Insects,
particularly scaly and spotted ones such as mosquitoes when not
pinned, should be placed (as obtained) in a small, stout box be-
tween layers of lens, or other similar, paper, and well padded all
round. They should never be placed in alcohol or other preserva-
tive since this renders them unsuitable for dry mounting, whereas
if dried in the natural state, the appendages may be relaxed by
suitable methods.
APPENDIX IX

A. NEOTROPICAL ELAPID AND VIPERID VENOMOUS SNAKES FOUND IN THE WESTERN HEMISPHERE *

Family: COUBRIDAE†

Subfamily: ELAPINAE

Genus: Micrurus

Cobras, coral snakes.

Thirty-five species and 15 subspecies. Of these M. coralinus, M. frontalis, and M. nigrocinctus are the most common. The first two are South American, the last Central American.

Family: VIPERIDAE

Subfamily: CROTALINAE

Genus: Crotalus

Includes subfamily: VIPERINAE (without loreal pits), confined to the Eastern Hemisphere; subfamilies: CROTALINAE and LACHESISINAE (the former with loreal pits and rattles, the latter with loreal pits only).

Two species in Central and South America: Crotalus unicolor (confined to Aruba Island, Dutch West Indies off Venezuela), and C. durissus terrificus (South American rattle-snake), and C. d. durissus, Central America into Mexico. There are nine strictly Mexican species, and seven subspecies of species occurring in the United States.

Sistrurus

(Ground rattlers)

S. raus (Mexican ground rattlesnake), known only from central Mexico.

* The summaries of distribution and numbers of species, together with the nomenclature, are based on the most recent literature, the principal authorities being Schmidt (1922) on the coral snakes; do Amaral (1925), Klauber (1934-1938), and Gloyd (1949) on the crotalines and lachesines.

† One widely distributed poisonous colubrid species of the Asiatic sea snakes, subfamily HYDROPHINAE reaches the Pacific coast of Central America and northeastern South America north into the Gulf of California: Pelamis platurus (bicolored sea snake).
Subfamily: LACHESINAE

Genus: Agkistrodon

One species in Mexico, A. bilineatus (Mexican moccasin).

Bothrops

Thirty-seven species, 12 subspecies in South America, Central America, and Mexico. Best known of these is B. atrox (known as "fer-de-lance," distributed over practically the entire range of the genus; B. jararaca ("Jararaca"), very common in southern Brazil and B. schlegelii (Schlegel's eye-lash or palm viper), an arboreal species frequently carried to this country in bunches of bananas from northeastern South America and southern Central America.

Lachesis

One species only, Lachesis muta, the bushmaster, found in tropical South America north into Panama.

B. NEARCTIC ELAPID AND VIPERID VENOMOUS SNAKES FOUND IN THE UNITED STATES AND CANADA *

Family: COLUBRIDAE

Subfamily: ELAPINAE

Genus: Micrurus

One species, M. fulvius (coral snake, harlequin snake) with two subspecies; all are found in southern United States, North Carolina south through Florida, west into Texas and north in the Mississippi Valley to Missouri and Illinois.

Genus: Micruroides

One species, M. euryxanthus (Sonoran coral snake). Southern New Mexico and Arizona, northern Mexico and Tiburon Island.

* The list of species for the United States and Canada is based on the most recent edition (1939) of Stejneger and Barbour, "Check List of North American Amphibians and Reptiles," and Gloyd's monograph, "The Rattlesnakes, Genera Sistrurus and Crotalus" (1940).
Family: **Viperidae**
Subfamily: **Lachesisinae**
Genus: *Agkistrodon*

“Rattleless rattlesnakes” and moccasins (with loreal pits, without rattles).

Two species, *Agkistrodon mokasen mokasen* (Southern lowland copperhead). Atlantic coastal plain and lower Piedmont areas of Georgia and the Carolinas, exclusive of peninsular Florida, west to eastern Texas. *Agkistrodon mokasen cupreus* (Northern or Highland copperhead). Eastern Oklahoma and eastern Kansas; higher areas of west central and northwestern Arkansas, Missouri (except southeastern portion), north to central Illinois, central Indiana, southern and eastern Ohio, Pennsylvania, and southeastern New York; Appalachian highlands from the Tennessee River and northeastern Alabama to eastern Massachusetts.

*Agkistrodon mokasen laticinctus* (Wide-banded or Texas copperhead). Western and central Texas, western and central Oklahoma, north to Cowley County, Kansas.

*Agkistrodon piscivorus* (Cotton-mouth moccasin). Dismal Swamp, Virginia, through Florida and the Gulf States, north in the Mississippi Valley to Missouri and Illinois.

Family: **Viperidae**
Subfamily: **Crotalinae**
Genus: *Sistrurus*

“Rattlesnakes” (with loreal pits and rattles).

*S. catenatus catenatus* (massasauga; swamp rattlesnake). Eastern Kansas, western Missouri northeastward to eastern Minnesota and south central Wisconsin, the Georgian Bay region in Ontario, western Pennsylvania and west central New York.

*S. catenatus tergeminus* (western massasauga). From southeastern Arizona, the valley of the Rio Grande and the gulf coast of Texas, north through southeastern Colorado and central Oklahoma to eastern Kansas and southeastern Nebraska.
VENOMOUS SNAKES

Family: Viperidae—(Continued)
Subfamily: Crotalinae—(Continued)
Genus: Sistrurus—(Continued)

*S. miliarius miliarius* (Carolina pigmy rattlesnake). From central Alabama and central Georgia northward east of the Allegheny Mountains to east central North Carolina.

*S. miliarius barbouri* (dusky pigmy rattlesnake; Barbour's pigmy rattlesnake). The southeastern Coastal Plain from southern South Carolina and southern Georgia, throughout Florida and westward to the valley of the Pearl River in Mississippi.

*S. miliarius streckeri* (western pigmy rattlesnake). Valley of the Pearl River in Mississippi north into southwestern Tennessee, southern Missouri and central Oklahoma and west to central and southeastern Texas.

Genus: Crotalus

*Crotalus triseriatus pricei* (Price's rattlesnake; Arizona spotted rattlesnake). High elevations in the western Sierra Madres and adjacent mountains from southeastern Arizona to southern Durango, Mexico.

*C. lepidus lepidus* (Texan rock rattlesnake). Western Texas, with the exception of the El Paso region, eastern Coahuila, Nuevo Leon and San Luis Potosí, Mexico.

*C. lepidus klauberi* (green rock rattlesnake). The mountains of southeastern Arizona, west central New Mexico, and El Paso County, Texas, and higher elevations on the Mexican Plateau in Chihuahua, Durango, Zacatecas, and Jalisco.

*C. molossus molossus* (northern black-tailed rattlesnake; dog-faced rattlesnake). From the Grand Canyon, Arizona, and central New Mexico south through Sonora, Chihuahua, and western Texas to southern Coahuila and south central Nuevo Leon; and on San Esteban Island in the Gulf of California.
Family: **Viperidae**—(Continued)
   Subfamily: **Crotalinae**—(Continued)
   Genus: **Crotalus**—(Continued)

* C. *horridus* *horridus* (timber rattlesnake; northern banded rattlesnake). Upland areas from east central Oklahoma, eastern Kansas, southeastern Nebraska, the Ozark Highlands of western Arkansas and Missouri, north in the Mississippi Valley about to the forty-fifth parallel in Wisconsin; eastward through south central Illinois, southern Indiana, Kentucky, central and eastern Tennessee, northeastern Alabama and throughout the Appalachian Highlands to northern New York and southern Maine; along the Atlantic Coastal Plain from New Jersey to northeastern Massachusetts.

* C. *horridus* *atrcaudatus* (cane-brake rattlesnake; southern banded rattlesnake). Lower Mississippi Valley from southeastern Texas north as far as southeastern Missouri and Jackson County, Illinois, and the southeastern Coastal Plain, except peninsular Florida, north at least to southern Virginia.

* C. *scutulatus* *scutulatus* (Mojave rattlesnake). The Mojave Desert of California, southern Nevada, extreme southwestern Utah, the Colorado Desert and adjacent areas of the southwestern half of Arizona, southwestern New Mexico, Trans-Pecos, Texas, and south on the Mexican Plateau to San Luis Potosi.

* C. *atrox* (western diamond rattlesnake). Desert, plains, and foothill areas from southeastern California to west central Arkansas, south to central Mexico.

* C. *ruber* (red diamond rattlesnake). From southeastern Los Angeles County, California, south approximately to latitude 26° in Baja California.
C. adamantaeus (eastern diamond rattlesnake). The coastal plain of the southeastern United States, including all of Florida from southern Mississippi to east central North Carolina.

C. viridis viridis (prairie rattlesnake). The Great Plains region of North America from southeastern Alberta and southwestern Saskatchewan to northeastern Sonora, northern Chihuahua, and west central Texas; from the Rocky Mountains eastward through the central portions of North and South Dakota and north central Nebraska to extreme western Iowa, central Kansas, and western Oklahoma. Toward the west it has spread through gaps in the Rocky Mountains to eastern Idaho (Lemhi Valley), southeastern Utah, and northeastern Arizona.

C. viridis oreganus (Pacific rattlesnake). The Pacific slope from British Columbia to Central Baja California. An isolated population occupies the central plateau of Arizona and adjacent areas.


C. viridis abyssus (Grand Canyon rattlesnake). The Grand Canyon of the Colorado River, Coconino County, Arizona.
Genus: *Crotalus*—(Continued)


*C. viridis numtius* (Arizona prairie rattlesnake). The basin of the Little Colorado River and adjacent territory in north central and northeastern Arizona.

*C. mitchelli pyrrhus* (southwestern speckled rattlesnake). Southern California, extreme southern Nevada, western Arizona, and Baja California.

*C. mitchelli stephensi* (panamint rattlesnake). Mountain and rocky desert areas of east central California and southwestern Nevada.

*C. tigris* (tiger rattlesnake). Rocky foothills of mountains and their adjacent desert slopes in south central Arizona and north-eastern and central Sonora, from the vicinity of Phoenix, Arizona, to Guaymas on the gulf coastal plain of Sonora.

*C. willardi* (Willard's rattlesnake; ridgenosed rattlesnake). High elevations from the Santa Rita and Huachuca Mountains, Arizona, through the Sierra Tarahumara and Sierra Madre of eastern Sonora, western Chihuahua, Durango, and western Zacatecas.

*C. cerastes* (horned rattlesnake; side-winder). Desert areas of the southwest including: California east of Sierra Nevada and southern coastal ranges from southern Mono County southward; southern Nevada; extreme southwestern Utah; Arizona south and west of the line Kingman-Miami-Nogales; extreme northwestern Sonora and northeastern Baja California.
1. CLASSIFICATION OF ANIMAL PARASITES AND ANTHROPODS

A useful, simple classification of animal parasites in general is one arranged by Stiles as follows:

1. Unicellular animals (without tissues), as the parasites of malaria .......................................................... PROTOZOA
   Pluricellular animals (with tissues); metazoa .......................... 2

2. Body more or less flattened dorsoventrally .......................... 4
   Body ordinarily round in transverse section .......................... 3

3. Body never annulated; never provided with legs; jaws absent ... 5
   Body annulated, or at least provided with mouth-parts; usually breathe through a tracheal system; adults with jointed legs or other appendages ........................................... 7

4. Intestine, but no anus present; one or two suckers present; body not segmented; parasitic in liver, lungs, blood, intestine, occasionally elsewhere; flukes ....................... TREMATODA
   Intestine absent; two or four suckers on head; body of adults segmented; tissue usually contains calcareous corpuscles; adults (tapeworms) parasitic in intestine; larvae (bladder worms) parasitic elsewhere .............................................. CESTODA
   Intestine and anus present; sucker on posterior end; body annulated like an earthworm; parasitic in upper air passages, or externally; leeches, blood suckers ....................... HIRUDINEA

5. Intestine absent; armed rostellum present; very rare in man, in intestine; thorn-headed worms .................. ACANTHOCEPHALA
   Intestine present; no armed rostellum .................. NEMATODA 6

6. Intestine rudimentary in adults; lateral chords absent; rare, accidental parasites in intestine of man; hair snakes or horse-hair worms .................................................. GORDIACEA
   Intestine present; lateral chords present; parasitic in intestine, muscles, lymphatics, etc., very common and important; roundworms .......................................................... EUNEMATODA

7. Six legs present in adult; wings present in most species; larva annulated much like an earthworm; breathe through trachea;
adults ectoparasites; occasionally larva is parasitic under skin, or in wounds, or an accidental parasite in the intestine or bladder; insects ........................................... INSECTA
Eight legs present in adult, six legs in larva; head and abdomen coalesced; ectoparasites; some burrow under the skin or live in the hair follicles; acarines ............................ ACARINA
Four claws around the mouth; larva encysted in various organs; adult occasionally parasitic in nasal passages; tongue worms

LINGUATULIDAE
Numerous legs present; occasionally accidental parasites in nasal passages or intestine; thousand leggers ................. MYRIAPODA

A practical classification for use of the student of medical parasitology and entomology, arranged from Stitt et al., Chitwood and Chitwood, is as follows:

THE PROTOZOA

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<td>SARCOSPORIDIA</td>
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The nematoda or true roundworms have recently been divided by Chitwood and Chitwood into two main subgroups as follows:

1. Phasmids (PHASMIDIA) which have "phasmids" or caudal chemoreceptors but no caudal glands; simple porelike amphids, and somewhat modified excretory ducts.

2. Aphasms (APHASMIDIA) which do not have "phasmids" or caudal chemoreceptors; amphids highly modified externally; caudal glands typically present; excretory system greatly reduced.

The most important roundworms of the PHASMIDIA (Chitwood and Chitwood, 1933) arranged under superfamilies are as follows:
Superfamily: **Rhabditoida**, Travassos (1920)

*Strongyloides stercoralis.* (Occurs generally in warm climates, sporadically in temperate regions)

*Rhabditis spp.* (Temporary parasite of damaged tissues and lumens of organs)

*Turbatrix aceti.* (Vinegar eel. Accidental parasite of urine and vaginal exudate)

**Anuillulinoidea**, Pereira (1931–1932)

*Anuillulina putrefaciens.* (Parasite of onion)

*Heterodera radicicola.* (Common parasite of roots and stem of garden plants)

**Strongyloidea** (Weinland, 1858), Hall (1913)

*Ancylostoma duodenale*

*Ancylostoma braziliense*

*Necator americanus*

*Terridens deminutus.* (Man and various primates in Africa)

*Oesophagostomum apiostomum.* (Primates in W. Africa, Philippines, and China, and man in N. Nigeria)

*Oesophagostomum stephanostomum var. thomasi.* (Brazil — rare)

*Syngamus laryngeus.* (Cattle gape-worm)

**Trichostrongyloidea**, Cram (1927)

*Trichostrongylus spp.*

*Haemonchus contortus.* (Sheep wireworm)

*Mecistocirrus digitatus.* (Stomach and small intestine of domestic animals in Orient)

**Metastrongyloidea**, Cram (1927)

*Metastrongylus elongatus.* (Porcine lung worm)

**Oxyuroidea**, Railliet (1916)

*Enterobius vermicularis*

*Syphacia obvelata.* (A cosmopolitan parasite of rats and mice and accidental parasite of man)

**Ascaroidea**, Railliet and Henry (1915)

*Ascaris lumbricoides*

*Toxocara canis.* (Cosmopolitan intestinal parasite of dogs)

*Toxocara cati.* (Cosmopolitan intestinal parasite of cats)

*Lagochilascaris minor.* (Parasitic in intestine of cloudy leopard, *Felis nebulosa*)

**Spiruroidea**, Railliet and Henry (1915)

*Gongylonema pulchrum.* (Comparatively rare in mouth, esophagus, stomach, and intestine of man; commonly
occurs in various ruminants, monkeys, pigs, bears, and hedgehogs)

*Gnathostoma spinigerum.* (Reported in gastric tumors of tiger, wild and domestic cat and dog in Orient; rare in man)

*Gnathostoma hispidium.* (Relatively common in stomach wall of wild and domestic pigs in Europe and Asia)

*Physaloptera caucasica.* (Relatively common in the alimentary canal of natives of tropical Africa)

*Thelazia callipaeda.* ("Oriental eye worm" of rabbit, dog, and man in Orient)

*Thelazia californiensis.* (Reported a few times in dogs and once in the cat, and once also in man in California)

**FILARIOIDEA** (Weinland, 1858), Stiles (1907)

*Wuchereria bancrofti.* (Bancroft's filarial worm)

*Onchocerca volvulus.* (Convoluted filaria)

*Acanthocheilonema perstans.* (Persistent filaria)

*Mansonella ozzardi.* (Ozzard's filaria)

*Dirafilaria immitis.* (Heart filaria of dogs. Somewhat similar filaria have been reported in man a few times.)

*Loa loa.* (Eye worm)

**DRACUNCULOIDEA,** Cameron (1934)

*Dracunculus medinensis.* (Medina, guinea, serpent, or dragon worm)

The most important roundworms of the subgroup **APHASMIDIA** (Chitwood and Chitwood, 1933) have been arranged under superfamilies as follows:

Superfamily: **TRICHINELLOIDEA,** Hall (1916)

*Trichinella spiralis.* (Trichina worm)

*Trichurus trichiura.* (Whipworm)

*Capillaria hepatica.* (Relatively common in rat and related species)

**MERMITHOIDEA,** Wülker (1934). Accidental human parasites from contaminated food and water

**DICTOPHYMOIDEA,** Railliet (1916)

*Dictophyme renale.* (Rarely found in kidney and abdominal cavity of man; more commonly in certain fish-eating mammals including the dog in certain localities)
### THE FLATWORMS (PLATYHELMINTHES)

<table>
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<th>Class</th>
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<th>Genus</th>
<th>Species</th>
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<td>Paragonimus</td>
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<td>Troglopus</td>
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<td>Echinostomatidae</td>
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<td>Diplogonoporus</td>
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<td>Taenia</td>
<td><em>T. solium</em></td>
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<td><em>T. saginata</em></td>
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Two larval Taeniidae are found in man (*Cysticercus cellulosae* and *Echinococcus granulosus*); also two larval Diphyllobothridae (*Sparganum mansoni* and *Sparganum proliferum*).

### THE ARTHROPODS

Arthropods, which comprise the arachnids, insects and their allies are subdivided into several classes, as follows:

1. **Crustacea**, comprising on the one hand small forms known as "cyclops," and on the other hand the common crayfish, lobsters, and crabs. These animals are largely aquatic, breathing through gills.

2. **Chilopoda**, which usually have but one pair of legs and one pair of spiracles to each postcephalic segment. The first pair of legs is modified to form the "poison-claws." The name commonly given to these is "centipede."

3. **Diplopoda**, which have two pairs of legs and two pairs of spiracles or respiratory openings on most of the body segments. Representative examples are the millipedes or "thousand-leggers."

4. **Arachnida**, which comprise forms in which the head and thorax are fused. They lack antennae, and the adults possess four pairs of legs.
Arthropods of medical importance classed here are the mites, ticks, spiders, and scorpions.

5. **INSECTA**, which have a distinct head, thorax, and abdomen, and possess one pair of antennae, one or two pairs of wings (on the thorax). Forms of considerable medical importance are mosquitoes, flies, lice, bugs, and fleas; of minor importance are beetles, wasps, butterflies, and moths.

The medical significance of arthropods is based on the ability of a comparatively few of them, owing to their wide geographical range, to transmit diseases with high morbidity rates and in some instances with high mortality. As examples may be cited plague, a bacterial disease; *typhus fever*, a rickettsia disease; malaria, a protozoal disease; and *yellow fever*, a virus disease. In the past these infections have been the cause of enormous economic losses as well as many untimely deaths. At times some have reached pandemic proportions.

### THE ARACHNIDS

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GLOSSARY

II. DEFINITIONS

Aberrant (L. ab, from, + erro, wander). In botany and zoology, differing in some of its characters from the group in which it is placed.

Acaricide (G. akari, a mite, + L. caedere, to kill). A medicinal agent used to destroy acari or mites.

Accolé or Appliqué Forms (F. accolé, from L. accolo, dwell by; F. appliqué, applied). In subtertian-infected red corpuscles, these frequently appear, when stained by the Romanowsky method, as short streaks on the cell periphery. They have red nuclear dots and often a bacilliform appearance.

Acetabulum (L. acetabulum, a shallow cup). A cup-shaped depression on the external surface of the innominate bone, into which the head of the femur fits; applied to the ventral sucker of the Trematoda and related worms.

Aedes (G. aedes, unpleasant, unfriendly). A genus of mosquito of small size found in tropical and subtropical regions. In this genus is included the vector for yellow fever and dengue.

Agamous (G. agamos, unmarried). In zoology, having no distinguishable sexual organs.

Aggregatidae (L. ad, to, + gregare, to flock). A family, belonging to the coccidia, in which there is both an alteration of generation and of hosts.

Albumin (L. albus, white). A protein widely distributed throughout the tissues and fluids of plants and animals. It is rich in sulphur and like all proteins is very complex.

Ameba, amoeba (G. amoibe, change, alteration). A unicellular, protozoan organism of globular shape when at rest, but endowed in the trophic stage with the power of extruding pseudopodia and assuming various shapes. Many amebas are parasitic; some pathogenic. Caudamoeba (L. cauda, a tail). A genus of amebae having a tail-like prolongation. Endamoeba or Entamoeba (G. entos, within). A genus of ameba within the body.

Amebiasis, amoebiasis (G. amoibe, change, + iasis, denoting a condition). The state or condition of being infected with amebae; more specifically infection with Endamoeba histolytica.
Amebic dysentery (G. amoibe, change; dysentere, bowel). A bloody, mucoid diarrhea caused by *Endamoeba histolytica* and occurring as one of the manifestations of amebiasis.

Amebicide (G. amoibe, change, + caedere, to kill). A substance capable of killing amebae.

Ametabolic (G. a, without, + metabolos, changeable). Not due to, causing, or undergoing metabolism.

Amorphous (G. a, without, + morphe, form). Without definite form; shapeless.

Amphids (G. amphi, around). Integumentary receptors; sensory structures located laterally on the cephalic end of roundworms. They are commonly found in free-living species and are believed to be present in simple form in all or most parasitic species.

Anaerobic (G. an, without, + aer, air, + bios, way of living). A microorganism which thrives best or only when deprived of oxygen.

Anal gill (L. anus, from annus, a ring; Dan. giselle, gill). One of the respiratory organs of such animals as breathe the air that is mixed with water. Example: mosquito larvae have four anal gills (membranous projections on the ninth or last abdominal segment).

Ancylostoma (G. ankylos, hooked, + stoma, mouth). The genus of "old world hookworm." It is a human hookworm of the warmer parts of the eastern hemisphere. The method of infection is percutaneous and once through the skin, the larvae follow an indirect route to the intestine via the venous system to the lungs, thence out into the air passages and over the epiglottis into the intestinal tract.

Androphilous (G. andro, man, + philos, love). Preferring the blood of man to that of lower animals as applied to feeding habits of insects, particularly mosquitoes.

Anisogamous reproduction (G. anisos, unequal, + gamos, marriage). Fusion of two gametes of unequal form or size.

Anopheles (G. anopheles, harmful). A genus of dipterous insects (mosquitoes), belonging to the family Culicidae. The sporogenous cycle of the malarial parasite is passed in the stomach and body cavity of the female of the genus *Anopheles*.

Anorexia (G. an, without, + orexis, appetite). Loss or absence of appetite.

Antenna (G. ana, up, + teinein, to stretch). A sensory process (usually segmented) on the head of insects, myriapods, and crustaceans. Antennata, tracheates with one pair of antennae. A subphylum or class of Arthropoda consisting of the true insects (Hexapoda) and the Myriapoda.

Anthelmintic (G. anti, against, + helmins, worms). A medicinal agent used to destroy or expel worms from the intestinal tract.

Anthomyidae (G. anthos, flower, + myia, a fly). A family of non-biting flies frequently found about plants. The eggs are commonly deposited
upon decayed vegetable matter and excrement in which the larvae develop. The larvae of some species may be parasitic in insects and other animals; those of *Fannia (Homalomyia) canicularis* may find their way into the intestine or even the urinary tract of man.

**Antibodies** (G. *anti*, against, + A.S. *bodig*, body). Specific constituents of the blood and fluids of an immune animal.

**Antivenin** (G. *anti*, against, + *venenum*, poison). The active ingredient in an antiserum against the venom of an animal such as a snake or an insect.

**Apodal** (G. *a*, without, + *pod*, foot). Without feet.

**Apterous** (G. *a*, without, + *pteron*, wing). Without wings.

**Aquatic** (L. *aqua*, water). Growing in or frequenting water.

**Arachnida** (G. *arachne*, spider). A class of arthropods to which belong ticks, mites, spiders, and scorpions.

**Arboreal** (L. *arbor*, tree). Attached to or frequenting trees.

**Arista** (L. *arista*, the awn* of an ear of corn). A feathered, tactile bristle borne on the enlarged terminal (third) segment of the antenna.

**Arthropoda** (G. *arthron*, a joint, + *pod*, a foot). A phylum consisting of animals with segmented bodies, some or all of whose segments bear jointed appendages. The body has a chitinous exoskeleton. It embraces crustaceans, insects, arachnids, myriapods, and protostomates.

**Ascariasis** (G. *askaris*, slender worm). The symptoms produced by the presence of ascarid worms or larvae in the body.

**Ascaris** (G. *askaris*, slender worm). A parasitic nematode worm of cosmopolitan distribution.

**Autochthonous** (G. *auto*, self, + *chthon*, land). Originating in the place where found; said of a disease originating in the part of the body where found, or of a disease acquired in the place where the patient is.

**Autoclave** (G. *auto*, self, + L. *clavis*, a key; in the sense of self-locking). An apparatus for sterilizing objects by steam heat under pressure.


**Balantidium** (G. *balantidion*, a little bag). A genus of ciliates, parasitic in the large intestine of vertebrates.

**Basal granule** (L. *basis*, base; *granulum*, dim. of grain). A small body in the blepharoplast from which a flagellum originates.

**Basis capituli** (L. *basis*, base; *capituli*, dim. of *caput*, head). Basal portion of the capitulum or head of a tick.

**Basophil** (G. *basis*, base, *phileo*, I love). A cell, especially a leukocyte, which stains readily with basic dyes.

* A barbed appendage which terminates the flowering glumes or scales of a spikelet.
Bifid (L. bifidus, forked). Cleft or division into two parts or branches.

Bilharzia (Bilharz — German helminthologist, 1824–1862). See Schistosoma.

Binary fission (L. binus, a couple; fissus, to cleave). Reproduction by splitting into two equal parts.

Bivalve (L. bi, two, + valva, a valve). Having two valves or shells, as an oyster.

Bladderworm (A.S. blaedre, bladder, + wyrn, worm). The larval stage of cestode worms. See Cysticercus.

Blastocystis (G. blastos, a germ, + kystis, a cyst). A genus of yeastlike organisms found in the intestines.

Blepharoplast (G. blephar, an eyelash, + plassein, to mould). The locus of origin of the axoneme of a flagellum. Many believe the blepharoplast is of nuclear origin since in certain stages a flagellate may lose its flagellum and become rounded, and when the flagellum reforms, a granule separates from the karyosome and passes through the nuclear membrane to become the blepharoplast.

Blood platelets (A.S. blod, blood; G. platys, flat). Colorless or slightly bluish bodies. They appear to be constricted-off portions of the pseudopodia of certain giant cells of the bone marrow. Their function is not fully known but is in some way connected with coagulation.

Bothria (G. bothrion, dim. of bothros, a pit). A boat-shaped, sucking groove on either side of the scolex of diphyllobothrid parasitic flatworms.

Bowman’s capsule (Sir William Bowman, English anatomist, physiologist, and ophthalmologist, 1816–1892). The expanded beginning of a uriniferous tubule. See Glomerulus.

Breathing trumpets (A.S. braeth, breath, odor; F. trompette, dim. of trompe, trumpet). The breathing apparatus of Culicidae pupae (tumblers). See Tumbler.

Brood capsules (A.S. brod, offspring; L. capsula, dim. of capsa, chest). Small vesicles formed in the hydatid cyst (echinococcus) by invagination of portions of the inner membrane (germinal layer). A brood capsule may contain many (sometimes 40) scolices invaginated up to the neck and attached to the capsule internally by a stalk. Both stalks and scolices are surrounded by a cuticular layer.

Bubo (G. boubon, the groin). Inflammation and swelling of a lymphatic gland, particularly of the groin.

Buccal (L. bucca, cheek). Pertaining to the cheeks or mouth cavity.

Buccal capsule (L. bucca, cheek; capsa, chest). In nematodes, a large mouth cavity of chitinous nature, portions of which may be developed into cutting organs.

Budding (M.E. budde, bud). A method of reproduction by which a protuberance from the parent organism develops into a new organism.
Capitulum (L. capitulum, dim. of caput, head). The head of a tick.
Caryogamy (karyogamy) (G. karyon, kernel [nucleus], + gamos, marriage). Fusion of the nuclei of two cells, as occurs in zygosis or true conjugation.
Cast (M.E. casten, to throw). A mould of a tubular structure, such as a bronchial tube or a renal tubule, formed by a plastic exudate.
Caudal (L. cauda, a tail). Pertaining to the tail.
Caudal glands (L. cauda, tail; glan[-d]s, acorn). These are usually three in number arranged in tandem in the forward part of the roundworm’s tail. They empty through a small spinneret at or near the tip of the tail. They serve to cement the tail to objects and are common in free-living and non-bursate parasitic species (often absent or highly modified in bursate parasitic species).
Cell (L. cellare, to cover). Originally the envelope or covering of a living unit; later by association, the living unit as a whole. A granular mass of protoplasm containing a nucleus. The typical adult cell consists of cytoplasm, a nucleus, and within the latter, one or more nucleoli. The cell may or may not have a cell wall.
Centriole (G. kentron, a point, center). In higher forms, a deeply staining granule in the middle of the centrosome. It is assumed that an intranuclear centrosome, the centriole, is present in the nuclei of Protozoa. This is supposed to be embedded in the karyosome; and only to become recognizable during nuclear division. The centriole and blepharoplast are considered synonymous by Minchin and Cleveland.
Centrosome (G. kentron, center, + soma, body). In higher animals, a small body situated outside the nucleus. It is concerned in mitosis with the formation of the spindle and chromosomes and the subsequent separation of the daughter chromosomes.
Cephalic (G. kephale, head). Pertaining to the head.
Cephalothorax (G. kephale, head, + thorax, breastplate). The fused head and thorax of arachnids.
Ceratophyllus (G. keros, keratos, horn, + phyllos, from phyllon, having leaves). A genus of Siphonaptera (fleas).
Cercaria (G. kerkos, a tail). A free-swimming, terminal, larval stage of trematode worms having a tail-like appendage.
Cercomonas (G. kerkos, a tail, + monas, a unit). A genus of flagellates.
Cestode (G. cestode, a girdle). Shaped like a girdle or ribbon; applied to worms of which Taenia is a type.
Chagas’ disease (Carlos Chagas, Brazilian physician). A disease caused by the protozoan flagellate Trypanosoma cruzi; also referred to as “Brazilian trypanosomiasis.”
Chelae (G. chele, a claw). Pincer-like terminations of certain of the limbs of arachnids and crustaceans.
Cheliceræ (G. chele, a claw, + keras, horn). The first pair of appendages of the Arachnida. These lie in front of the mouth.

Chilomastix (G. cheilos, a lip, + mastis, a whip). A genus of flagellates.

Chitin (G. chiton, a coat of mail). A horny substance forming the harder parts of the integument of insects and other arthropods.

Chlamidozoa (G. chlamys, a cloak, + zoon, a living animal). A group of organisms supposed to occur in body cells. They are surrounded by a cloak or zone caused by intercellular reaction.

Chondriosomes (G. chondros, cartilage, + soma, body). A protoplasmic inclusion. Chondriosomes occur as small spherical or oval granules or rodlike bodies and show a tendency to adhere to protoplasmic surface. Chemically they are similar to the Golgi body — protein compounded with a lipoidal substance. Their function has not been definitely determined.

Chromatin (G. chroma, color). The portion of the nucleus of a cell which is readily stained by dyes; distinguished from the nonstainable portion or achromatin. Achromatinic (G. an, not). Not giving the same staining reactions as chromatin. Chromatolysis (G. luein, to dissolve). Breaking up and disappearance of the nuclear chromatin, one of the earliest stages of nuclear degeneration.

Chromatoid bodies (G. chroma, color, + eidos, appearance). Deeply staining rod-shaped bodies in the cytoplasm characteristic of certain amebae.

Chromosome (G. chroma, color, + soma, a body). One of the small, usually rod-shaped or threadlike bodies into which the nuclear chromatin is segregated during the process of mitotic division. As the cell divides, these bodies split longitudinally — one-half going to the nucleus of each of the daughter cells.

Chrysops (G. krysos, gold, + ops, opas, face, eye). A genus of Tabanid fly with spotted wings, commonly called deer fly, mango fly.


Ciliates (L. cilia, eyelashes). Protozoa which move by means of short vibratile filament called cilia. See Cilia.

Cimex (L. cimex, a bug). A genus of true bug to which the bedbugs belong (family Cimicidae).

Cirrus (L. cirrus, a curl). A protrusible portion of the vesicula seminalis (widened portion of the vas deferens) found in the tapeworms and flukes.

Cloaca (L. cloaca, a sewer). A terminal sac into which the intestine and the excretory and reproductive ducts of many lower animals open.

Clonorchis (G. clon, a branch, + orkhis, testicle). A genus of flukes (Trematoda) with branched testes. The Chinese liver fluke is Clonorchis sinensis.
Clypeus (L. clypeus, a round shield). A plate or shield of the anterior median part of an insect's head. It usually bears the labrum.

Coarctate (L. coarctatus, confined, i.e. confined in its larval skin). In the coarctate form, the pupating larva shrinks in its skin so that the resulting pupa lies in a case or puparium formed by the hardened skin of the larva. Example: the pupa of the housefly.

Coccidia (G. kokkos, a berry). A group of organisms belonging to the class Sporozoa.

Coccidiosis (G. kokkos, berry, + osis, increase in the organism). The group of symptoms produced by the presence of coccidia in the body.

Cocoon (Fr. cocon, dim. of coque, shell). The envelope in which larval worms are enclosed in the chrysalis or pupal state.

Coelenterata (G. koilas, hollow, + enteron, intestine). A diploblastic group of animals with an embryonic body cavity.

Coelom (G. koiloma, a hollow). A cavity in the mesoderm lined by epithelium; into it the excretory organs open and from its walls the reproductive cells originate.

Coelozoic (G. koiloma, a hollow, + zoon, animal). Pertaining to parasites which live in the coelomic cavities of the body.

Coenurus (G. koinos, common + oulos, type). The larva of the tapeworm Multiceps multiceps, with multiple scolecies, known as Coenurus cerebralis, which causes a condition in sheep called "gid" or staggers.

Coleoptera (G. coleos, sheath, + pteron, wing). Insects with four wings, the sheathlike fore-wings covering the membranous hind-wings; biting mouth parts; complete metamorphosis.

Coli (G. kolon, the large intestine). A species name denoting a parasitic inhabitant of the large intestine.

Colubridae (L. coluber, serpent). A family of snakes containing about 90 per cent of all living snakes. This family includes both harmless and poisonous snakes. They have fixed facial bones, and teeth on both jaws.

Commensalism (L. con, with, + mensa, table). The living together of organisms neither bearing a parasitic relation to the other, without harm or prejudice to either. SYN. Symbiosis, mutualism.

Complete metamorphosis (L. compleo, fill intensely; G. meta, beyond, after, morphe, form). A term used to describe the changes from a larva which is utterly unlike its parents, to an adult. The larva eats voraciously until it reaches full size, then it becomes a dormant pupa. This stage ends suddenly and the insect emerges a sexually mature imago. SYN. Holometabolous.

Conjugation (L. conjugatus, joined together). The sexual union or fusion of two cells, with partition of the chromatin and subsequent division into two new cells.

Contractile vacuole (L. contrahere, to contract; vacuolum, dim. of vacuum,
an empty space). A cavity formed by the accumulation of fluid in the extoplasm of a protozoan; after increasing for a time it empties itself externally by a sudden contraction.

Convolution (L. convolvere, to roll together). A fold, twist, or coil of any organ or organism.

Coprozoa (G. kopros, dung, + zoom, an animal). A name used for the free-living organisms found in stale feces and fecal culture.

Copulation (L. copula, joining). Sexual union of male and female.

Copulatory bursa (L. copula, joining, + bursa, a purse). A membranous expansion at the posterior end of Strongyloid males; somewhat bivalvular and supported by ribs of stouter tissue.

Corium (L. corium, leather). The deep layer of the skin. SYN. Dermis.

Costa (L. costa, rib). A strong vein running along the front edge of the wing of insects. It may extend around the wing margin or may end abruptly at or near the tip.

Coxa (L. coxa, hip). The hip or hip joint. In insects and other arthropods the first segment of the leg from the body, articulating with the second segment or trochanter.

Crithidia (G. krithe, a barleycorn). Flagellates of the genus Crithidia show, in the course of their development, leishmania, leptomonas, and crithidia forms. They are limited to invertebrate hosts and are transmitted by means of cysts.

Crotalinae (G. krotalon, rattle). A subfamily of the Viperidae which possess two "loreal pits" situated on a line below and between the eyes and nostrils on either side of the head. The Crotalinae with rattles at the end of the tails are American, while those without are found in Asia and the Americas.

Crustacea (L. crusta, a crust). A class of the Arthropoda, including Cyclops, crabs, lobsters, shrimps, etc.

Ctenidium (G. ctenidios, dim. of cteis, ctenos, comb). A flat, chitinous row of spines situated on the head or pronotum of cicadas.

Culex (L. culex, mosquito). A genus of mosquito belonging to the family Culicidae. A species of this genus (C. fatigans) is a transmitter of Filaria bancrofti.

Culicidae (L. culex, mosquito). A family of insects, belonging to the order Diptera, which includes all the true mosquitoes.

Cuticular (L. cutis, skin). Resembling or pertaining to the epidermis or outer skin.

Cyclops (G. cyclop, a mythical one-eyed animal). A small, fresh-water crustacean, a species of which serves as the intermediate host of Dracunculus medinensis, and as the first intermediate host of Diphyllobothrium latum (the fish tapeworm).

Cyst (G. kystis, a cyst or bladder). A stage in the life history of many lower
animals during which the organism is protected by an enclosing wall secreted by the body of the organism.

**Cysticercus** (G. *kytis*, bladder, + *kerkos*, tail). Formerly a genus of bladderworms, now known to be the encysted larvae of various tapeworms. SYN. Bladderworm.

**Cytogenous** (G. *kytos*, cell, + *genos*, generation). Producing cells—designating specifically lymphatic or adenoid tissue.

**Cytoplasm** (G. *kytos*, a cell, + *plassein*, to form or mould). The substance of a cell exclusive of the nucleus and various other inclusions.

**Cytopyge** (G. *kytos*, cell, + *pyge*, buttocks). Slitlike anus of certain Protozoa, as *Balantidium coli*.

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**Cytozoic** (G. *kytos*, a cell, + *zo*, I live). Pertaining to parasites which live within the cell cytoplasm.

**Daughter cysts** (A.S. *dauher*, daughter; G. *kystis*, bladder). Endogenous and exogenous daughter cysts are formed by the mother echinococcus cyst and are not attached to it. The endogenous type is formed within the original cyst from broken bits of germinal layer, from brood capsules, or from scolices. The exogenous type is formed by the outward budding of the germinal layer. These cysts are exactly like the original and may produce brood capsules, scolices, and daughter cysts.

**Deirids** (G. *deiro*, the neck, especially its anterior portion). See Papilla.

**Dentate** (L. *denatus*, toothed). Having a toothed margin or toothlike projections.

**Deposition** (L. *depositum*, to lay down). The act of depositing, especially of eggs.

**Dermacentor** (G. *derma*, skin, + *kentor*, a goader). A genus of tick belonging to the family *IxoRidae*. It is important as the vector of Rocky Mountain spotted fever, tularemia, and bovine anaplasmosis.

**Diaphragm** (G. *dia*, across, + *phragm*, wall). In microscopy, an apparatus placed between the mirror and the object to regulate the amount of light that is to pass through the object.


**Digenetic** (G. *di*, two, + *genesis*, generation). A term applied to those trematodes in which an asexual multiplication takes place in the larval stages. As the name implies there are two generations—namely, a sexual and an asexual.

**Dimorphism** (G. *di*, two, + *morphe*, form). The property of assuming or of existing under two distinct forms.
Dioecious (G. di, two, + oikos, a house). In biology, having the two sexes in different individuals or in two households, as staminate and pistillate flowers on separate plants.

Diphyllobothrium (G. di, two, + phyllon, leaf, + bothrion, dim. of bothros, pit). A genus of CESTOIDEA or tapeworm which has, instead of the usual four sucking disks, one sucking groove on either side of the head. SYN. Dibothriocephalus.

Diploblastic (G. diplous, double, + blastos, germ). Formed of two germ layers.

Diplopoda (G. diplos, double, + pod, foot). A class of Arthropoda having two pairs of legs for each of the body segments except the first two and the last one. To this class belong the millipedes.

Diptera (G. di, two, + pteron, wing). Insects with one pair of wings attached to the mesothorax; the metathorax bears the “halteres” or balancers.

Dipterous (G. di, two, + pteron, wing). Having two wings; belonging to the insect order DIPTERA.

Dipylidium caninum (G. dipylus, with two entrances; L. caninus, dog). A species of dog tapeworm, the larvae of which are harbored by fleas; the adult worm may also infest man. Each proglottid has two sets of genital organs and the genital pores lie symmetrically at the lateral margins.

Distomum, distoma (G. di, two, + stoma, mouth). A genus of trematode worms or flukes, the members of which are now usually referred to other genera as Fasciola, Fasciolopsis, Paragonimus, Clonorchis, Opisthorchis, Cotylogonimus, Dicrocelium, and Schistosoma.

Dorsal (L. dorsum, back). Pertaining to the back part of an animal or organ.

Dorsum (L. dorsum, back). The back surface of an animal.

Dracunculus (L. dracunculus, dim. of draco, serpent). A genus of parasitic threadlike worms belonging to the order FILARIOIDEA. The important human species is D. medinensis ("guinea worm").

Dutton’s membrane (L. membrana, membrane). A thin membrane in CULICIDAE at the end of the labium, between the latter and the labellae.

Ecdysis (G. ek, out, + dyein, to put on, enter). The act of putting off an outer covering and appearing in a new one as in the larval forms of parasitic roundworms, insects, crustaceans, and in snakes. SYN. molting.

Echinococcus (G. echinos, hedgehog, kokkos, a berry). A genus of Cestodes which forms “hydatid (echinococcus) cysts.” The definitive hosts of E. granulosus are dogs and cats while various mammals including man may serve as intermediate host.

Echinococcus cyst (G. echinos, hedgehog, + kokkos, a berry; kystis, bladder). A fluid-filled vesicle formed from the Echinococcus embryo. It has
a fairly thick cuticle, concentrically laminated, and an inner germinal layer which may give rise to "brood capsules," or daughter cysts, or it may be sterile. syn. Hydatid cyst.

**Ectoparasites** (G. ekto, outside, + parasitos, a guest). A parasite that lives on the exterior of its host.

**Ectoplasm** (G. ekto, without, outside, + plassein, to mould). The outer layer or zone of body substance in a protozoan cell.

**Egg** (A.S. aeg, egg). The female sexual cell once a shell has been secreted around the ovum.

**Ejaculatory duct** (L. ejaculare, to throw out; ducere, to lead). A receptacle formed at the junction of the vas deferens and the duct of the seminal vesicle which carries the semen into the urethra.

**Elapidae** (G. elaps, ellaps, a sea-fish, serpent). A family group name applied to poisonous terrestrial colubrids.

**Elephantiasis** (G. elephas, elephant, + iais, denoting a condition). Hypertrophy of the skin and subcutaneous tissues due to obstructed circulation in the lymphatic vessels. A specific form is attributed to the presence of *Wuchereria bancrofti*.

**Elytra** (G. elytron, from elyein, to roll around). The thickened fore-wings of beetles, serving to cover the hind-wings.

**Embryophore** (G. embryon, embryo, + phoros, a bearer). The embryonic envelope of the egg which in some forms may be ciliated.

**Encystment** (G. en, in, + kystis, bladder). The process of becoming enclosed in a cyst or capsule.

**Endemic** (G. en, in, + demos, people). Of a disease, found in a certain place more or less constantly.

**Endogenous** (G. endon, within, + gennao, I produce). Originating or produced within the organism or one of its parts.

**Endomixis** (G. endon, within, + mixis, intercourse). At certain stages, the macronucleus of a ciliate degenerates and disappears, while the micronucleus divides into two parts one of which becomes the new macronucleus. This process of regeneration occurs most frequently during conjugation but may also occur during the course of asexual multiplication, when it is known as endomixis.

**Endoplasm** (G. endon, within, + plasma, thing formed). The inner portion of the body substance in a protozoan cell.

**Endosome** (G. endon, within, + soma, body). One or more conspicuous bodies other than chromatin granules occurring within the nuclear membrane. See Karyosome.

**Enterobius** (G. enteron, intestine, + bios, life). A genus of Nematoda or roundworms. *Enterobius vermicularis* (commonly called pinworm) is often found in the human rectum and is about one-quarter of an inch long. syn. Oxyuris.
Enterozoic (G. enteron, intestine, zoom, animal). Pertaining to parasites which live in the lumen of the digestive tract.

Eosinophile (G. eosin, dawn, + philos, fond). A cell or other element, especially a leucocyte, which stains readily with eosin.

Epidemic (G. epi, upon, + demos, the people). Common to or affecting at the same time a number of persons in a locality; applied to a disease which spreading widely attacks numerous people at the same time.

Epimerite (G. epi, upon, + meris, part). An organ by which parasitic Protozoa may attach themselves to their host. It may be a simple swollen body embedded in the cytoplasm of the parasitized cell and connected with the parasite by a neck. It may be sucker-like with filaments passing between or into the cell or it may have spines.

Epimeron (G. epi, upon, + meris, part). A lateral part or piece of the wall of the somites of arthropods situated between the notum (tergum) and the insertion of the appendages. In insects the term is applied specifically to a piece of the pleuron of the thoracic somites which is situated posterior to the epistemum.

Episternum (G. epi, upon, + sternon, breast). The piece of pleuron in the thoracic region of insects which is situated anterior to the epimeron.

Epithelium (G. epi, upon, + thele, nipple). A cell layer which covers the surfaces of the body both without and within, such as the skin and lining of the alimentary canal. It may be protective, absorptive, secretive, and excretive; and may be flat (squamose), columnar, or stratified.

Erythematous (G. erythema, flush or redness). Redness of the skin, rose-rash.

Erythrocyte (G. erythros, red, kytos, cell). A red blood corpuscle.

Espundia. A chronic, ulcerating, protozoan disease of the skin more or less resembling Oriental sore but often associated with refractory ulcerating granulomatous lesions of the nasal and buccal mucosas. It is caused by infection with Leishmania braziliensis. SYN. Uta, forest yaws, nasopharyngeal leishmaniasis.

Estivo-autumnal (L. aestivus, summer; autumnus, autumn). Relating to or occurring in summer and autumn.

Exogenous (G. exo, outside, + genna, I produce). Originating or produced outside the body.

Exudate (L. ex, out, + sudare, to sweat). The material that has passed through the walls of the vessels into the adjacent tissues.

Facet (Fr. facette, dim. of face, face). A smooth, flat, circumscribed surface.

Facultative parasite (L. facultas, faculty, ability; parasitos, a guest). An organism that is capable of living either free or as a parasite.

Fang (A.S. fangen, to seize). A long prominent tooth.
Fannia. A genus of non-biting fly. It bears a superficial resemblance to the housefly but is smaller. syn. Homalomyia.

Fasciola (L. fasciola, dim. of fascia, a band). A genus of Trematoda or flukes which has a cosmopolitan distribution throughout the sheep-raising areas of the world. It lives in the biliary passage of various mammalian hosts where it produces a disease known as "liver rot."

Fascioliasis (L. fascia, a band). A term employed for distomiasis. The presence of a genus of trematode worms, Fasciola.

Fasciolopsis (L. fasciol, dim. of fascia, a band, + G. opsis, form, appearance). A genus of large intestinal fluke of pig and man in the Orient.

Fauna (L. Fauna, a Roman goddess, the sister of Faunus). The aggregate of the animals of a given region.

Ferment (L. fermentum, leaven). A substance which in contact with another substance is capable of setting up changes (fermentation) without itself undergoing change. Proteolytic ferments convert proteins into peptones and albumoses.

Festoons (L. festum, festival). A decorative band hanging in a curve between two points. In entomology, applied to the scalloped appearance of the lateral edges of an insect's body, as exemplified in the human louse.

Filaria (L. filum, a thread). Formerly established as a genus of nematode threadworms, but now replaced by various new genera. The members of this group live parasitically in the subcutaneous, lymph and connective tissue and body fluids of animals.

Filariform larva (L. filum, thread, + formare, to form; larva, a mask). A nematode larval form produced by the moulting of the rhabditiform larva. It has an elongate esophagus and no mouth. This is the infective stage for man and other animals.

Filiform (L. filum, thread, + formare, to form). Threadlike.

Fission (L. fendere, to cleave). Reproduction by simple division of the body into two parts, each of which becomes a complete organism.

Flagellum (L. flagellum, a whip). An organelle of locomotion; a whiplike appendage or process of a cell.

Flame cell (L. flamma, flame; cella, small room). A large, hollow cell of the excretory system of Platyhelminthes. It has a long tuft of cilia extending into a central cavity which empties into the excretory tubule. The cell collects wastes from its surroundings and propels these substances (by ciliary movement) toward the bladder.

Flora (L. flor, flower). The aggregate of the plants of a given region.

Fossa (L. fossa, a trench). A depression or pit.

Free living (A.S. freo, free; lifian, to live). An organism that is not parasitic.
Frons (L. frontis, forehead). The forehead, particularly of insects.

Gamete (G. game,es, husband; game, ete, wife). One of two cells undergoing caryogamy or true conjugation. In heredity any germ cell, whether ovum, spermatozoon, or pollen cell.

Gametocyte (G. game,es, husband, game, ete, wife, + kytos, cell). A sexually differentiated cell giving rise to a gamete, as in the plasmodia of malaria.

Genal (L. gena, cheek). Pertaining to the cheek.

Genital atrium (L. genitalis, genital; atrium, antechamber). An indented portion of the body wall which harbors the opening of the vagina and the vas deferens.

Genital tubules (L. genitalis, genital; tubulus, tube). A general term applied to the female reproductive ducts leading to the uterus of parasitic worms, such as Ascaris lumbricoides.

Germ layer, germinal layer (L. germen, germ; A.S. liegan from liegan, to lie). Any of the layers of cells that are differentiated in the early stages of embryonic development. In the evolution of the gastrula, two layers are formed, the inner (endoderm) and the outer (ectoderm). Later a third layer (mesoderm) forms between these two.

Gills (Dan. giaelle, gill). The respiratory organs of such animals as breathe the air that is mixed with water.

Gingivalis (L. gingivae, gums). The specific name of an Endamoeba which inhabits the human mouth.

Glomerulus (L. glomerulus, dim. of glomus, a skein). A plexus of capillaries; in the kidney, a tuft formed of capillary loops at the expanded beginning of each uriniferous tubule. This tuft with its capsule (Bowman's capsule) constitutes the Malpighian body. SYN. Malpighian tuft.

Glossina (G. glossa, tongue). A genus of biting flies (tsetse flies) belonging to the family MUSCIDAE. Some species are vectors of trypanosomiasis.

Glycogen (G. glykys, sweet, + genes, producing). So-called animal starch, found, for example, in liver and in cysts of amebae.


Golgi body (Golgi, Italian anatomist, 1844). It is found in metazoan cells and in many protozoans. The Golgi substance is osmiophile and argentophile and possesses a strong affinity to neutral red. Golgi bodies occurring in protozoa are small osmiophile granules or larger spherules which are composed of osmiophile cortical and osmiophobe central substances. They are composed of lipoidal material in combination with a protein. Their function is not definitely known.

Granular (L. granulum, granule). Consisting of grains or granules.

Gregarious (L. gregarius, gregarious). Inclined to gather together, as to live in groups, flocks, or herds.
Grubs (M.E. grubben, grobben, grub). The larvac of insects such as beetles.

Gubernaculum (L. gubernaculum, a rudder). A cuticular thickening on the dorsal wall of the cloaca in many of the male Eunematoda (Ex. Anclyostoma duodenale) which guides the spicules during copulation.

Gynecophoric canal (G. gyne, woman, + phoreo, I carry; L. canaliss, channel). A channel formed by the infolding of the lateral margins of the body wall of a male schistosome worm, which the female occupies during copulation.

Habitat (L. habitare, to dwell). The natural abode of an animal or plant.

Haematobia (G. haemato, blood, + bios, life). A genus of biting fly, species of which resemble Stomoxys calcitrans but are somewhat smaller. They are troublesome, particularly to cattle.

Haemocoele (G. haema, blood, + koiloma, cavity). The system of blood-containing spaces pervading the body of arthropods and mollusks.

Halteres (G. halteres, weights held in the hand in leaping). The rudimentary hind-wings or balancers of Diptera.

Haploid (G. haplous, simple, + eidos, resemblance). One-half the number of chromosomes typical of the species. The fully mature gametes possess the haploid number of chromosomes as a result of meiosis.

Hausellum (L. haustus, p. p. of haurire, to draw in, breathe, swallow). A proboscis adapted to take food by suction, as in many insects.

Hematozoic (G. haima, blood, + zoon, animal). Pertaining to parasites which live in the blood.

Hemelytra (G. hemi, half, + elytron, from elysein, to roll around). The partially thickened anterior wings of Heteroptera as contrasted with those of Homoptera which are about the same thickness throughout.

Hemiptera (G. hemi, half, + pteron, wing). An order of insects commonly known as "bugs" comprising the suborders Heteroptera and Homoptera.

Hemocytometer (G. haimo, blood, + kytos, cell, + metron, measure). An instrument for estimating the number of corpuscles in the blood.

Hemoglobin (G. haimo, blood, + globus, a ball). The coloring matter of the red corpuscles.

Hemoglobinometer (G. haima, blood, + globus, globe, + G. metron, measure). Instrument for estimating the amount of hemoglobin in the blood, indicated in percentages of the normal.

Hemolysis (G. haimo, blood, + lysin, solution). Destruction or disorganization of the blood corpuscles.

Hermaphrodite (G. Hermaphroditos, a mythological character of dual personality). An organism which has both male and female sex organs. See Monoecious.
Hermaphroditism (G. Hermaphroditos, a mythological character of dual personality). The occurrence or union of the two sexes in the same individual.

Herpetology (G. herpeton, a reptile, + logos, discourse). The study of reptiles.

Herpetomonas (G. herpetos, a creeper, + monas, a unit). Flagellates of the genus Herpetomonas are purely invertebrate parasites. In their cycle are found leishmania, leptomonas, crithidia, and trypanosome forms. Infection is passed on by encysted leishmania forms.

Heterophyes (G. heteros, different, + phyte, shape). A genus of trematode worm (fluke) which is small and pyriform in shape.

Heteroptera (G. heteros, different, + pteron, wing). A suborder of hemiptera, normally characterized by having the first pair of wings thickened at the base and the distal overlapping portion membranous. These are “true bugs,” further characterized by a jointed proboscis attached anteriorly, which, when not in use, is commonly folded under the head.

Hexacanth (G. hex, six, + acantha, thorn). The six-hooked tapeworm embryo; the onchosphere.

Hexapod (G. hex, six, + pod, foot). A six-footed animal; a true insect as applied to diptera.

Histolytica (G. histes, a web, + luein, to dissolve). The specific name of the pathogenic, tissue-invading Endamoeba of man.


Holophytic (G. hолос, whole, + phyton, plant). Wholly or distinctively vegetative in nutrition, involving photosynthesis.

Homalomyia (G. homalos, even, + myia, a fly). See Fannia.

Homoptera (G. homos, same, + pteron, wing). A suborder of hemiptera in which the first pair of wings is of about the same thickness throughout.

Hookworm (A.S. hok, hook, + wyrm, worm). A common name for worms of the genera Ankylostoma or Uncinaria and Necator. This designation was originally made (vide Stiles) because the bursal rays of the male were erroneously interpreted as hooks (Goeze, 1782).

Host (L. hospes, entertainer). An organism which harbors a parasite.

Definitive host. The host which harbors the adult stage of the parasite.

Intermediate host. The host which harbors the larval stage or stages of the parasite. First intermediate host. The first host parasitized by the larval stages of the parasite. Second intermediate host. The host parasitized by stages at a later period in the life cycle.

Hyaline (G. hyalos, glass). A glassy or transparent substance or surface.

Hydatid cyst (G. hydatis, a drop of water). See Echinococcus cyst.


Hymenolepis (G. hymen, membrane, + lepis, rind). A genus of Cestoda
(tapeworm), including the dwarf tapeworm of man (H. nana) and that of rats, mice, etc. (H. diminuta).

**Hymenoptera** (G. hymen, membrane, + pteron, wing). Insects possessing two pairs of membranous wings with few veins; the first abdominal segment fused or partly fused with the thorax; mouth-parts both mandibulate and suctorial; female with an ovipositor; metamorphosis complete.

**Hypopharynx** (G. hypo, under, + pharynx, throat). In mosquitoes and flies, the hypopharynx is a constituent part of the proboscis. With the labrum-epipharynx, it forms a tube through which the blood is sucked. It contains also the salivary canal down which pass the salivary juices and malaria sporozoites when present.

**Hypopygium** (G. hypo, beneath, + pyge, buttocks). The last one or two segments of insects, modified for sexual purposes; the external gentilia (hypopygium) of the male.

**Hypostome** (G. hypo, under, + stoma, mouth). A median, probe-shaped anchoring organ of acarines arising from the basis capituli and having numerous backwardly projecting darts or spines.

**Idiosome** (G. idios, one's own, + soma, body). The attraction-sphere of a spermatid or of an oocyte.

**Imago** (L. imago, likeness). The final or adult stage of insects.

**Immunity** (L. in, not, + munis, serving). Exemption from disease; the condition of the body wherein it resists the development of morbid processes; resistance to infection.

**Incidental parasite** (L. incidere, to fall into; parasitos, a guest). A parasite that establishes itself in a host in which it does not usually live.

**Incomplete metamorphosis** (G. meta, after, beyond, + morphe, form). In this type of metamorphosis the larva is recognizably like the adult but differs chiefly in absence of wings, in having a larger head, and shorter antennae. It becomes more like the adult with each moult until sexual maturity is reached.

**Incubation period** (L. in, on, + cubare, to lie). The first phase of a disease extending from the time the parasite enters the body until symptoms appear. It may be longer than the prepatent period since evidence of the presence of parasites often precedes the onset of clinical symptoms.

**Infundibuliform** (L. infundibulum, funnel). Having the form of a funnel.

**Insect** (L. insectum, p.p. of insecare, to cut in) so called because the body appears to be cut in or almost divided. Any member of the class INSECTA (syn. HEXAPODA). In the broadest sense, a class of arthropods comprising the true insects, the MYRIAPODA and the ARACHNIDA; in a more restricted sense, a class consisting of true insects together with the MYRIAPODA and equivalent to ANTENNATA, having a clearly defined head thorax and abdomen with only three pairs of legs.
Inspissator (L. *inspissare*, to thicken). A device employed to coagulate or to thicken by evaporation.

Isogamous (G. *isos*, equal, + *gamos*, marriage). Denoting conjugation of two gametes of the same form and size.

Kala-azar (Kala-azar = black fever, a name given to visceral leishmaniasis by the Garos of Assam). A fatal disease due to a protozoan parasite, *Leishmania donovani*, often referred to as the Leishman-Donovan body.

Karyosome (G. *karyon*, a kernel, + *soma*, body). A small granule found in the nuclei of certain Protozoa said to be composed of chromatin, plastin, linin, and in some instances, to contain the centrosome. See Endosome.

Kinetonucleus (G. *kinetes*, one that sets in motion, + L. *nucleus*, dim. of *mix*, nut). The kinetoplast or the parabasal body alone is often termed kinetonucleus. It is a misleading term since it is doubtful that it is a nucleus. See Kinetoplast.

Kinetoplast (G. *kinetes*, an originator, mover, + *plastos*, formed). The parabasal body and the blepharoplast when united into one body. It is usually found in trypanosomes and allied flagellates. Some believe it is a true nucleus in view of the fact that it divides with the true nucleus prior to cell division. It is safer, however, to consider it a structure concerned with the activities of the flagellum.

Labellae (L. *labella*, dim. of *labium*, lip). Small, stiff lobes at the distal end of the labium of insects.

Labium (L. *labium*, lip). In insects, the lower lip; in mosquitoes, it is a fleshy, scaled, grooved organ. It terminates in two short-hinged processes known as labellae. In the groove of the labium and protected by it rest the actual piercing organs which consist of the labrum-epipharynx, the hypopharynx, the mandibles, and the maxillae.

Labrum (L. *labrum*, lip). In insects, the uppermost part of the lip or sheath of the proboscis.


Larva (L. *larva*, a mask). The wormlike form of an insect on issuing from the egg; a grub, maggot, or caterpillar. The young of any animal differing in form from its parent.

Larviparous (L. *larva*, a mask, + *parere*, to bear). Describing animals, such as certain flies, which produce larvae directly after they have been nourished in the uterus.

Larviposit (L. *larva*, a mask, + *pono*, to put). To deposit larvae by a pregnant female as in insects and worms.

Latent period (L. *latere*, to be concealed; *peri*, around, + *odos*, way). A quiescent state following active clinical manifestations of a disease and
characterized by apparent recovery. It may be followed by a period of relapse, usually less severe than the initial attack.

**Lateral** (L. *latus*, side). At, belonging to, or pertaining to the side; situated on either side of the median vertical plane.

**Lateral lines** (L. *latus*, side; *linea*, line). Whitish lines extending the whole length of a roundworm and symmetrically situated on the sides.

**Lecithin** (G. *lekythos*, yolk of egg). A complex, nitrogenous fatty substance occurring widely throughout the animal body.

**Leishmania** (Leishman, British Army surgeon). Flagellates of the genus *Leishmania* resemble those of the genus *Leptomonas* in having only the leishmania and leptomonas forms, but they differ in having both a vertebrate and an invertebrate host.

**Leishmaniasis** (Leishman, British Army surgeon). A disease due to infection with any species of *Leishmania*.

**Lepidoptera** (G. *lepis* (lepid), scale, + *pteron*, wing). An order of insects distinguished by feather-like scales and spirally coiled suctorial apparatus. This order includes butterflies and moths.

**Leptomonas** (G. *leptos*, thin, + *monas*, a unit). Flagellates of the genus *Leptomonas* are those which never develop beyond the leptomonas stage. In the course of their life history, they show only leishmania and leptomonas forms; they are confined to invertebrate hosts, and pass from one to another by means of cysts voided with the dejecta.

**Leukocytes** (G. *leukos*, white, + *kytos*, cell). White corpuscles, or leukocytes, are found in the blood. There are several varieties. They all contain nuclei, and most of them contain granules which vary in size and staining properties. Their function is not fully understood. It appears to be concerned chiefly with the protection of the body against harmful agencies, in part through phagocytosis, in part through production of antitoxic substances and of ferment.


**Limax** (G. *limax*, a slug). A type of movement suggested by that of a slug or snail. **Endolimax**. A genus of amebae living in the intestine.

**Linin** (L. *linum*, thread). A threadlike nonstaining substance forming the network of the cell nucleus, containing in its meshes the nucleoplasm.

**Loa loa** (loa, a term applied to a worm by the natives of Angola, W. Africa). A species of filaria which invades the connective tissue and the conjunctiva.

**Lobe** (L. *lobus*, lobe). A somewhat rounded projection or division of an organ or part.

**Loreal pit** (L. *lorum*, thong — zool., space between eye and bill in birds, and the corresponding region in reptiles and fishes). A heat sensory
organ possessed by crotaline snakes. It is found on either side of the head somewhat below and between the eye and nostril.

**Lycopodium** (G. _lykos_, wolf, + _pud_, foot). The spores of _L. clavatum_ (official in the U. S. P.) occurring in the form of a light, fine yellowish powder.

**Lymph** (L. _lympha_, clear spring water). The fluid in the lymphatic vessels, the product of the filtration of the liquid portion of the blood through the walls of the capillaries.

**Lymphocytosis** (L. _lympha_, lymph, + _kytos_, cell). An excess of lymphocytes in the blood and other body fluids.


**Macrogamete** (G. _makros_, large, + _gamete_, wife). The large female gamete or germ cell.

**Macrogametocyte** (G. _makros_, large, + _gamete_, wife, + _kytos_, cell). The mother-cell producing macrogametes, or female elements of sexual reproduction in Protozoa.

**Macronucleus** (G. _makros_, large, + L. _nucleus_, dim. of _nux_, nut). The larger nucleus of a binucleate Ciliate. It is vegetative in function and governs metabolism and activities of the cell other than reproduction. It is formed by a division of the micronucleus.

**Macrostoma** (G. _makros_, great, + _stoma_, a mouth). A genus of flagellates which was originally called _Macrostoma_ (Alexieff) and which since has been named _Chilomastix_ (Alexieff).

**Malaria** (It. _malo_ [fem. _mala_), bad, + _aria_, air, referring to the old theory of the miasmatic origin of disease). A disease associated with the presence in the blood of a protozoan parasite, the _Plasmodium vivax_, the _Plasmodium malariae_, and the _Plasmodium falciparum_; it is characterized by periodic fever, enlargement of the spleen, and the presence in the blood within the erythrocytes of the specific parasites.

**Malpighian body or corpuscle** (Malpighi, Italian anatomist, 1628–1694). This consists of a capillary plexus (glomerulus) projecting into the funnel-like commencement of a uriniferous tubule (Bowman’s capsule).

**Malpighian tuft** (Malpighi, Italian anatomist; O.F. _tufa_, tuft). See Glomerulus.

**Mandibles** (L. _mandibula_, a jaw). In arthropods, the anterior pair of mouth parts which form biting jaws or piercing organs.

**Mansonella** (Manson, Sir Patrick, pioneer in tropical medicine). A nematode genus of filarial worms with sheathless embryos believed by many to be nonpathogenic.

**Margaropus** (G. _margaros_, the pearl oyster, + _pous_, foot). A genus of tick, commonly feeding on horses and serving to transmit Texas cattle fever.

**Mastigophora** (G. _matis_, a whip, + _pherein_, to bear). A class of Protozoa.
the members of which are characterized by the possession of organelles of locomotion called flagella.

**Maurer's clefts or dots** (Maurer, English malariologist). Coarse, brick-red or pinkish dots in Romanowsky-stained subtertian malaria-infected erythrocytes. These are usually fewer in number and are larger and more irregular than Schüffner's dots appearing in similarly stained tertian malaria-infected erythrocytes.

**Maxillae** (L. maxillae, jaw bones). In arthropods, paired appendages behind or below the mandibles, usually serving as accessory biting or piercing organs.

**Maxillary** (L. maxilla, jawbone). Pertaining to the maxillae (jaws or accessory piercing organs).

**Meiosis** (G. μεισιος, a lessening). The process of reduction division; i.e., the reduction of chromosomes in the nuclei of the cells of organisms preparatory to syngamy or sexual union.

**Merocyte** (G. μερος, a part, κυτος, cell). A schizont which shows divisions into asexual sporelike bodies, the merozoites.

**Merozoites** (G. μερος, a part, ζωον, animal). Asexually formed spores of the sporozoa.

**Mesonotum** (G. μεσος, middle, notum, back). This is the part of the notum that covers the mesothorax and in dorsal view covers almost all of the thorax. It is divided into the scutum and the scutellum.

**Mesothorax** (G. μεσος, middle, thorax, breastplate). The middle segment of the thorax of an insect.

**Metabolism** (G. μεταβολη, change). The physico-chemical processes in cells concerned in the building up of protoplasm, and its alteration or degradation.

**Metacercaria** (G. μετα, beyond, κερκος, tail). A term applied to cercaria after emergence from the molluskan host and loss of tail.

**Metachromatic** (G. μετα, change, + chroma, color). Relating to any color change, whether natural or produced by staining fluids.

**Metacyclic** (G. μετα, beyond, + κυκλος, circle). A type of trypanosome which occurs at the end of the insect cycle and resembles the blood forms of vertebrates but is usually smaller.

**Metagonimus** (G. μετα, posterior, + γονιμος, genitalia). A genus of small intestinal trematode worms, usually pyriform in shape.

**Metamere** (G. μετα, after, + μερος, part). One of a series of segments composing the body, as in many worms and arthropods.

**Metamorphosis** (G. μετα, beyond, + μορφε, form). Change of form or structure, as illustrated in the larval, pupal, and imago stages of an insect's development.

**Metanotum** (G. μετα, beyond, + notum, back). That part of the notum
which covers the metathorax. It is more or less overlapped by the mesonotum.

Metaphyta (G. meta, beyond, + phytan, plant). Plants consisting of many cells; all plants above the Protophyta.

Metathorax (G. meta, after, + thorax, breastplate). The posterior segment of the thorax of an arthropod.

Metazoa (G. meta, after, + zooon, animal). All animals above the Protozoa (first animals). Metazoa include animals that begin life as a single cell, which gradually develops into an organized community of diversified cells. In the embryonic state, they possess at least two distinct germinal layers.

Miasma, miasm (G. miasma, stain, pollution). A term loosely applied to the floating germs of any form of microbic life, especially those generating in marshy localities. A noxious effluvium or emanation.

Miasmatic (G. miasma, stain, pollution). Relating to or caused by miasma.

Microfilaria (G. mikros, small, + L. filum, a thread). A term employed to designate the larval forms of filaria worms.

Microgamete (G. mikros, small, + gametes, husband). The male germ cell. It is usually smaller than the female, actively motile, and similarly has a haploid number of chromosomes.

Microgametocyte (G. mikros, small, + gametes, husband, + kytos, cell). The parent male sex-cell which gives rise to the flagellate microgametes.

Micrometer (G. mikros, small, + metron, measure). An instrument designed for measuring minutely; applicable in its various forms to both macroscopic and microscopic objects.

Micron (G. mikros, small). One thousandth of a millimeter; a unit of microscopic measurement.

Micronucleus (G. mikros, small, + L. nucleus dim. of nux, nut). The smaller nucleus of a binucleate Ciliate (EUCILIATA). It is usually assumed to be the sex nucleus. When syngamy takes place the macronucleus disintegrates and the micronucleus alone takes part in the process.

Micropyle (G. mikros, small, + pyle, a gate). A small opening in the cyst wall of a female or macrogamete of a sporozoan which permits the entry of the male or microgamete.

Microscope (G. mikros, small, + skopeo, I view). An apparatus through which minute objects are rendered visible. It consists of a lens or a group of lenses by which a magnified image of the object is produced.

Midgut (A.S. mid, middle, + gut, gut). A portion of the digestive tube found in insects. It is divided into two parts: a tubular portion, the cardia, found in the thoracic region, and a dilated portion, the stomach proper, located in the abdominal region. It is in the stomach wall of the female mosquito that the oocysts of malaria are seen.
Minimum lethal dose (L. minimum, least; letum, death; G. dosis, dose). The quantity of a poisonous substance or toxin which will just kill an animal.

Miracidium (L. miracidium, early adolescence). The ciliated embryo of trematode and diphyllobothrid worms; in an appropriate molluscan host, it develops successively into rediae and cercariae.

Mitochondria (G. mitos, thread, + chondros, cartilage). Fine threads or filaments described as occurring within the cytoplasm of a cell.

Mitosis (G. mitos, thread). The process of indirect nuclear division.

Mollusca (L. molluscus, soft). A phylum of invertebrate animals, including snails, clams, and squids. They are mostly free living.

Monad (G. monos, single, alone). A unicellular organism; specifically a flagellate infusorian.

Monoecious (G. monos, single, + oikia, house). A species with both male and female reproductive organs in or on the same individual. See Hermaphrodite.

Monomorphism (G. monos, single, + morpha, form). Uniform in shape or structure.

Mononuclear (G. monos, single, + L. nucleus, dim. of nux, nut). Having but a single nucleus.

Morula (L. morula, dim. of morus, mulberry). The solid mass of cells resulting from early segmentation of the vitellus of an ovum. It differs from the typical blastula in having no central cavity.

Moulting, molting (L. mutare, to change). The shedding of the hair, feathers, or outer layer of the skin, which is replaced by a new growth. See ecdysis.

Mucosa (L. mucosa, fem. of mucosus, mucous). A mucous membrane.

Mucus (L. mucus, mucus). A viscid fluid secreted by mucous membranes. It consists of water, mucin, and inorganic salts, together with epithelial cells, leukocytes, etc., held in suspension.

Multiple fission (L. multus, many, + plica, fold; fissio, findere, to cleave). Division of the nucleus, simultaneously or successively, into a number of daughter nuclei, followed by division of the cell body into an equal number of parts, each containing a nucleus. syn. Sporulation

Musca (L. musca, fly). A genus of diptera belonging to the Muscoidea. To this genus belongs the common housefly (M. domestica).

Mutualism (L. mutus, in return, return). The living together of two organisms of different species, for the advantage that each derives from the other. syn. Commensalism, symbiosis.

Myasis. Same as Myiasis.

Myiasis (G. myia, a fly). A disease caused by the presence of the larvae of flies in or on the body. syn. Myasis, myiosis.

Myiosis. Same as Myiasis.
Myzomyia (G. myzoein, to suck + myia, a fly). A subgenus of anopheline mosquitoes restricted primarily to the Eastern Hemisphere.

Nana (G. nanos, dwarf). The name of a species of the genus Endolimax found in the human intestine.

Necator (L. necator, a murderer). The genus of new world hookworm. It is a natural parasite of the small intestine of man. Its life cycle is similar to Ancylostoma. SYN. Uncinaria.

Nemathelminthes (G. nema, thread, + helmins, a worm). A phylum of metazoan worms which contains a considerable number of parasites. They are elongate, round, unsegmented animals, mostly dioecious.

Nematocyst (G. nema, thread, + kystis, bladder). A pear-shaped component of a specialized cell containing a coiled structure designed to project piercing accessories into the bodies of other animals when released by an appropriate mechanism.

Neoplasm (G. neos, new, + plasma, thing formed). A circumscribed new formation of tissue characterized by abnormality of function, structure or location. The term includes all true tumors as well as tumor-like growths due to micro-organisms.

Neutrophil (L. neuter, neither, + G. phileo, I love). A cell, especially a leukocyte, which does not stain readily with either acidic or basic dyes.

Notum (G. notos, back). The simplest form of arthropod skeletal segmentation is a ring consisting of a dorsal arc or notum, and a ventral arc or sternum; between the notum and the sternum laterally there may be two side pieces or pleura. The pleura are sometimes inconspicuous as in the abdomen of an insect; sometimes large and complex as in the thorax of many arthropods. SYN. Tergum.

Nucleic acid (L. nucleus, dim. of nux, nut; acidus, sour). A substance of definite chemical constitution, the combination of which with proteins forms nucleins.

Nucleus (L. nux, meis, a nut). A highly differentiated mass of cell protoplasm; the executive center of the functional activity of the cell.

Nymph (G. nympha, a nymph). In incomplete metamorphosis, a larval arthropod when the wings have made their appearance.

Nyssorhynchus (G. nyssein, to prick + rhynehos, a beak). A subgenus of tropical American Anopheline mosquitoes of which members of the Nyssorhynchus group (white-hind-footed) constitute the most important malaria vectors of the Caribbean littoral.

Obligate parasite (L. ob, to, + ligare, to bind; G. parasitos, a guest). Applies to an organism which can exist only as a parasite; normally cannot live saprophytically.

Obtected larva (L. obductus, protected, i.e., merely protected by chitinous secretion). In the obtected form, the larva as it pupates casts off its
skin and the pupa is enveloped only in its own chitinous pellicle. Example: the pupa of the mosquito.

**Ocellus** (L. *ocellus*, dim. of *oculus*, eye). A small simple eye of many invertebrates.

**Octomitus** (L. *octo*, eight, + G. *mitos*, thread). A genus of flagellates with eight flagella.

**Octopod** (L. *octo*, eight, + G. *pod*, foot). Having eight feet, as in adult arachnids.

**Oesophagus** (G. *oïsophagos*, the gullet). The gullet; the portion of the digestive canal extending from the pharynx to the stomach.

**Ommatidia** (G. *ommatidios*, a diminutive eye). A compound eye is made up of many *ommatidia*, each of which is a tiny ocellus of a peculiar kind. Each ommatidia is structurally complete and morphologically independent, though functionally they all act together as a single organ. Compound eyes are found only in the phylum Arthropoda.


**Onchospere** (G. *onkos*, tumor, + *sphaira*, sphere). The tapeworm embryo; the hexacanth embryo.

**Ontogeny** (G. *on*, being, + *genesis*, origin). The development of the individual as distinguished from phylogeny, or the evolutionary development of the species.

**Oocyst** (G. *oon*, an egg, + *kystis*, a cyst). A zygote or fertilized female after encystment.

**Ookinete** (G. *oon*, an egg, + *kinetos*, motile). A motile, vermicular body which the fertilized macrogamete or zygote assumes prior to encystment as a mature zygote to become an oocyst. syn. *Vermiculus*.

**Oospore** (G. *oon*, an egg, + *sporos*, seed). Same as Zygote.

**Operculum** (L. *operculum*, cover or lid). A lidlike process or part. As applied to the eggs of flukes and the fish tapeworm, it is the lidlike process at one end of the egg through which the embryo escapes.


**Ornithodorus** (G. *ornis* (ornith-), bird, + *doros*, bag). A genus of tick, one species of which (*O. moubata*) is the agent of transmission of African relapsing fever.
Osmium (G. *osme*, smell, because of the strong odor of the tetroxide). A metallic element of the platinum group; symbol Os.

Osmosis (G. *osmos*, a thrusting, an impulsion). A kind of diffusion which takes place between two miscible fluids separated by a semipermeable membrane.

Osmotic pressure (G. *osmos*, a thrusting; L. *pressus*, p. p. of *premere*, to press). The force with which a solution enclosed in a semipermeable membrane attracts water. The unbalanced pressure which gives rise to the phenomena of diffusion and osmosis, as in a system in which there are differences in concentration.

Ovary (L. *ovarium*, egg receptacle). A glandular organ in which ova are formed.

Oviduct (L. *ovum*, egg, + *ductus*, duct). A small tube through which the ovum passes to the uterus.

Oviparous (L. *ovum*, egg, + *parere*, to bear). Producing eggs that hatch after they have passed from the body of the parent.

Oviposition (L. *ovum*, egg, + *positus*, p. p. of *poneTe*, to place). The laying of eggs, especially applied to insects and arachnids.

Ovipositor (L. *ovum*, egg, + *positus*, p. p. of *poneTe*, to place). A specialized organ, as in certain insects and arachnids, for depositing eggs.

Ovoviviparous (L. *ovum*, egg, + *vivus*, alive, + *parere*, to bear). Producing ova with a well-developed shell or covering in which the embryo is nourished by the yolk as in oviparous animals, but which incubate within the body of the parent.

Ovulation (L. *ovulation*, dim. of *ovum*, egg). The formation of ova in the ovary; the discharge of the ova from the ovary.

Ovum (L. *ovum*, egg). The female sexual cell. It is usually larger than the male element, and when fertilized gives rise to a new individual. When a shell is secreted, the term “egg” is used.


Palatine (L. *palatum*, palate). Relating to the palate; a pair of bones situated between the superior maxillaries and in front of the pterigoids.

Palpi (L. *palpare*, to feel). Appendages, usually organs of touch or taste, attached to the mouth-parts of insects and other arthropods.

Papilla (L. *papilla*, a nipple). A small, pimple-like projection. In parasitic nematoda, the papillae are organs of special sense confined to the labial and cervical regions and (in the male) to the genitals. Cervical papillae are also known as “deirids.”

Parabasal body (G. *para*, beside, + *basis*, to go). A substance which stains deeply with many chromatin dyes. According to Kofoid and Swezy, it varies in size in relation to metabolic demand and is a “kinetic reservoir.” Duboscq and Grassé maintain it is the Golgi apparatus.
**Glossary**


**Parasite** (G. *parasitos*, a guest (*para*, beside, + *silos*, food)). An animal or vegetable organism which lives on or in another and draws its nourishment therefrom.

**Parasiticide** (G. *parasitos*, parasite, + *caedere*, to kill). A substance that destroys parasites.

**Parietopterygoid** (L. *paries*, wall, + G. *pteryx*, wing, + *eidos*, resemblance). Pertaining to the parietal bone and pterygoid process of the sphenoid bone.

**Parthenogenesis** (G. *parthenos*, a virgin, + *genesis*, production). The production of asexual forms from a sexual element or gametocyte which has not undergone any form of syngamy or sexual union. It applies particularly to reproduction by unfertilized eggs.

**Pasteurella** (Pasteur, French chemist and bacteriologist, 1822–1895). A group of nonciliated and nonspore-bearing bacteria of the family Bacteriaceae.

**Patent period** (L. *patere*, to lie open). A phase of a parasitological disease. It begins with the time when evidence of the parasite is found by recovering (usually by means of some laboratory technique) some stage of the parasite (eggs, cysts, etc.); and ends when presence of the parasite cannot be demonstrated.


**Pediculus** (L. *pediculus*, louse). A genus of lice, two varieties of which (*P. capitis* and *P. vestimenti*) are human parasites.

**Pedipalpi** (L. *ped*, foot, + *palpare*, to feel). Pincer-like appendages of arachnids, located on either side of the mouth.

**Periblast, periplast** (G. *peri*, around, + *blastos*, a germ). The cytoplasm surrounding the nucleus of a cell.

**Periplast** (G. *peri*, around, + *plassein*, to mould). As applied to a cell, see periblast.

**Peristome** (G. *peri*, around, + *stoma*, the mouth). An area surrounding the cytostome or mouth of certain organisms.

**Phagocyte** (G. *phago*, I eat, + *kytos*, cell). A cell having the property of engulfing and digesting foreign matter harmful to the body.

**Pharynx** (G. *pharynx*, the throat). Part of the alimentary canal extending from the mouth to the esophagus.

**Phasmids** (G. *phasma*, light). In nematodes the phasmids consist superficially of a pair of lateral, postanal, porelike openings connected by means of tubules with corresponding sensory pouches and glands. They
are chemoreceptive in nature and do not occur in species having caudal glands.

**Phlebotomus** (G. *phleps* [phleb], vein, + *tonos*, a cutting). A genus of blood-sucking flies known as sandflies.

**Phylogenetic** (G. *phyton*, a tribe, + *genesis*, origin). Pertaining to the ancestral history or evolution of an animal or plant.

**Physalia** (G. *physalis*, a bladder). A genus to which belongs the "Portuguese Man-of-War," a colonial Hydrozoan coelenterate.

**Phytonastigina** (G. *phyton*, a plant, + *mastis*, a whip). A group (subclass) of flagellates, some of which infect the latex of certain plants.


**Piroplasmidae** (L. *pirum*, a pear, + G. *plasma*, a thing formed). Nonpigmented parasites of mammalian erythrocytes which are transmitted by ticks. *Babesia* (Piroplasma) *bigemina*, the cause of Texas cattle fever, *B. canis*, the cause of malignant jaundice in dogs, and *Theilera parva*, the cause of East Coast cattle fever, belong to this group.

**Plasma** (G. *plasma*, anything formed). The fluid part of the blood and the lymph.


**Platyhelminthes** (G. *platys*, flat, + *helmins* [helminth-], worm). Flat-bodied, more or less elongated, worms, the individuals of which contain both sexual elements. They include tapeworms (cestodes) and fluke worms (trematodes).

**Pleomorphic** (G. *pleön*, more, + *morphē*, form). Occurring in more than one form; multiform.

**Plerocercoid** (G. *pleres*, full, + *kerkos*, tail). An elongate, larval stage of *Diphyllobothrium* (broad or fish tapeworm). It encysts in the viscera and muscles of fish and is the infective form for man.

**Pleuron** (pl. pleura) (G. *pleuron*, side). See Notum.

**Plumose** (L. *pluma*, feather). Feathery; plumelike.


**Poikilocytosis** (G. *poikilos*, manifold, + *kytos*, cell). A condition of the blood characterized by the presence of poikilocytes (erythrocytes of irregular shape).

**Pollensose** (L. *pollensose*, fine flour). Bearing a powdery or pollen-like substance.

**GLOSSARY**

**Polymorphism** (G. *polys*, many, + *morphe*, form). Occurrence in various forms.

**Polymorphonuclear** (G. *polymorphos*, multiform, + L. *nucleus*, dim. of *nux*, nut). Applied to leukocytes which have a nucleus divided into segments connected only by thin bands of nuclear material.

**Postabdomen** (L. *post*, behind, + *abdere*, to hide). Consists of body segments situated posterior to the true abdominal segments.

**Postspiracular bristles** (L. *post*, behind, + *spiro*, I breathe; A.S. *bryst*, bristle). Stiff hairs found behind the spiracle or breathing orifices of Arthropods.

**Precystic stage** (L. *praee*, before, + G. *kystis*, bladder; L. *status*, p. p. *stare*, to stand). Rounded and somewhat inactive trophozoites which are formed prior to encystment. They cease to ingest food, extrude remains of food particles, and are therefore smaller in size.

**Predacious** (L. *praeda*, booty). Living by feeding on other animals.

**Prehensile** (L. *prehendere*, to seize). Adapted for grasping.

**Prehension** (L. *prehendere*, to lay hold of). The act of grasping or seizing.

**Prepatent period** (L. *praee*, before, + *patere*, to lie open). This extends from the time the infective parasites enter the body of the host until their eggs, cysts, etc., can be recovered.

**Proboscis** (G. *pro*, before, + *bosko*, feed). The tubular process of the head, especially of insects and arachnids, made up of various mouth-parts.


**Prodromal period** (G. *pro*, before, + *dramos*, a running; *periodas*, a circle). An early or initial stage of a disease.

**Proglottid** (G. *pro*, before, + *glossa*, tongue). The segment of a tapeworm.

**Pronotum** (G. *pro*, before, + *notos*, back). The part of the notum that covers the prothorax of an insect.

**Proctolytic** (G. *protos*, first, + *lysis*, loosening). Pertaining to, characterized by, or effecting proctolysis (change produced in proteins by ferments that convert them into diffusible bodies). See Ferments.

**Prothorax** (G. *pro*, before, + *thorax*, breastplate). The anterior segment of the thorax of an insect.

**Protista** (G. *protistas*, superl. of *protos*, first). Haeckel’s name proposed for a group comprising the lowest forms of animal and plant life.

**Protophyta** (G. *protos*, first, + *phyton*, plant). A group of the lowest orders of the vegetable kingdom, including the bacteria.

**Protoplasm** (G. *protos*, first, + *plasma*, a thing formed). Living matter; the basic substance of which animal and vegetable cells and tissues are largely composed. Undifferentiated protoplasm is a clear, viscid, colloidal fluid, of a specific gravity of about 1.250, resembling the white
of egg, and composed of carbon, nitrogen, oxygen, hydrogen, and a number of other elements, in complex and unstable combination.

Protozoa (G. protos, first, + zoon, a living animal). A phylum of the animal kingdom consisting of unicellular organisms.

Proventriculus (L. pro, before, + ventriculus, dim. of venter, belly). A muscular dilation of the esophagus in front of the stomach. It is often armed with chitinous teeth for triturating food.

Pruritus (L. prurire, to itch). An intense degree of itching.

Pseudopodia (G. pseudos, false, + pod, foot). Processes of the protoplasm of a cell which may be protruded or retracted, as for locomotion or for food taking.

Psilosis (G. psilosis, a stripping). The removal of the hair from a part.

Psoroptes (G. psora, scabies). A genus of "scab mites" belonging to the family SARCOPTIDAE. They do not burrow as the "sarcoptic mites" but live at the base of the hairs of the host, piercing the skin and causing an exudate which partially hardens, forming scabs which pile up as a crust of loose humid matter. This condition is known as scabies or scab.

Pterygoid (G. pteryx [pteryg-], wing, + eidos, resemblance). Wing-shaped; a term applied to various anatomical parts in the region of the sphenoid bone.

Pterygoid bone (G. pteryx, wing, + eidos, resemblance; A.S. ban, bone). In all vertebrates except mammals, a bone which connects the palatine bones with the points of suspension of the lower jaw. In mammals it is the homologue of the inner plate of the pterygoid process of the sphenoid bone.

Pterygomaxillary (G. pteryx, wing, + L. maxilla, jawbone). Of or relating to the pterygoid bone or process and the maxillary bone.

Pterygopalatine (G. pteryx, wing, + L. palatum, palatine). Relating to or connected with the pterygoid process and the palatine bone.

Pterygoquadrate (G. pteryx, wing, + L. quadrate, square). Of, pertaining to, or connected with the quadrate and the pterygoid bones.

Ptilinum (G. ptilon, a feather, from its appearance). In coarctate pupa, a distensible bladder-like sac which protrudes like a hernia from the forehead of the emerging fly. This enables the fly to force open the end of the puparium, after which it later shrivels and is retracted into the head of the fly. A crescent-shaped scar (frontal lunule) which may embrace the roots of the antennae is the only visible evidence of the former presence of this temporary accessory process. (Sometimes also supplementary suture-like markings (frontal sutures) extend downward on either side.)

Pubescent (L. pubescere, reaching the virile age). Arrived at puberty, or the earliest age at which the reproductive function can be performed; covered with hair.
Pultaceous (G. pollos, porridge). Macerated, pulpy, paplike.

Pulvilli (L. pulvillus, a little cushion). Membranous pads on the last tarsal segment of a fly’s leg.

Pupa (L. pupa, a doll). The second stage of development of the egg, of such insects as undergo complete metamorphosis.

Puparium (L. pupa, a doll). In coarctate pupae, a case in which an insect is enclosed between its larval stage and the state of full development or imago.

Pupiparous (L. pupa, doll, + parere, to bear). Pertaining to insects in which the young are born at the commencement of or in the pupal stage.

Pygidium (G. pygidion, dim. of pyghe, rump). A pitted and setose or bristled sensory plate found on the dorsal, posterior part of the flea.

Pyiform (L. pyrus, a pear, + forma, form). Pear-shaped.

Quadrate (L. quadratus, square). Designating or pertaining to a bony cartilaginous element on each side of the skull to which the lower jaw is articulated in most vertebrates below the mammals. It is especially conspicuous in birds and reptiles and is present in one form or another in amphibians and fishes.

Quartan (L. quartanus, relating to a fourth [thing]). A form of intermittent malarial fever, the paroxysms of which occur every fourth day.

Rectum (L. rectus, straight). The lower part of the large intestine, extending to the anus.

Redia (Redi, Italian naturalist, 1626–1698). A larval stage of digenetic trematodes. The rediae are produced from germ cells in the sporocyst. This generation of redia may produce a second and a third generation of redia which develop into the cercariae. The rediae have at their anterior end a sucker-like structure; they also have a pharynx, a simple intestinal tube and a birth aperture.

Reticulation (L. reticulum, dim. of rete, a net). The presence or formation of a reticulum or network, such as is seen in erythrocytes during active blood regeneration.

Reticulocyte (L. reticulum, dim. of rete, a net, + G. kytos, cell). Erythrocytes having a coarse network of granular filaments. It is characteristic of very young red corpuscles, and the number of these in the circulating blood is probably the best available index of the activity of blood regeneration. These filaments are blue in a Romanowsky-stained preparation.

Reticulo-endothelial (L. reticulum, a little net; G. endon, within, + thele, nipple). A term applied by Bischoff to a cell group with endothelial and reticular attributes, and a certain common behavior toward dyestuffs. These cells are found in the liver, spleen, bone marrow, and hemolymph nodes.
Rhabditiform (G. rhabdos, a rod, + L. forma, shape). A type of esophagus with an anterior wider portion followed by a narrow, shorter portion and terminating in a spherical bulb.

Rhabditoid larva (G. rhabdos, a rod, + eidos, resemblance; L. larva, ghost). A type of larva found in nematode worms characterized by a rhabditiform esophagus. It is the feeding stage of larval roundworms.

Rhizoplast (G. rhiza, root, plastos, formed). The portion of the axoneme or axial filament of the flagellum between the blepharoplast and the surface of the body where the flagellum commences. This term is often applied to other structures as a fibril from the blepharoplast to the nucleus.

Rhizopoda (G. rhiza, root, + pod, a foot). A subclass of the Sarcodina but sometimes used synonymously with the class name.

Rickettsia (Ricketts, American pathologist, 1871–1910). A group of organisms, either bacterial or protozoal, found in typhus fever, trench fever, and Rocky Mountain spotted fever. Knowledge of their exact nature and pathogenicity is incomplete.

Rostellum (L. rostellum, dim. of rostrum, beak). A small mound with hook-like processes, especially the hook-bearing portion of the head of certain worms.

Rostrum (L. rostrum, a beak). A beak-like process or appendage.

Saccharomyces (G. sakcharon, sugar, + mykes, fungus). A family of unicellular vegetable organisms, of which the yeast plant is a common example.

Saprophagous (G. sapros, putrid, + phagein, to eat). Subsisting on decaying matter.

Saprophyte (G. sapros, decayed, + phyton, plant). Any vegetable organism living on dead or decaying organic matter.

Sarcodina (G. sarka, flesh, + dina, a whirling). A class of the phylum Protozoa comprising the subclasses Rhizopoda and Actinopoda.

Sarcoptes (G. sark-), flesh, + kopto, I cut). The genus of mange or itch mites. They make burrows in the skin where the female deposits her eggs. The name S. scabei is applied to all species inhabiting the skin of mammals, the differences being slight, many interchanging hosts.

Schistosoma (G. schistos, cloven, + soma, body). A genus of digenetic trematode worms, characterized by the absence of a muscular pharynx and by having nonoperculated eggs. They are dioecious and inhabit the portal blood stream, the females being frequently found in the vesical and pelvic plexuses. Syn. Bilharzia.

Schistosomiasis (G. schistos, cloven, + soma, body, + iasis, denoting a condition). Infestation with Schistosoma (a genus of digenetic trematode worms).
Schizogenesis (G. schizo, split, + genesis, production). Reproduction by fission. SYN. Schizogony.

Schizogony (G. schizo, I split, + gone, generation). The process of asexual multiplication by which a trophozoite, after completion of growth, becomes a merocyte containing a number of smaller bodies called merozoites.

Schizont (G. schizo, split, + ont, a being). A sporozoan parasite of the asexual generation, more particularly as it matures to form the merocyte or male and female gametocytes.

Schüffner’s dots (Schüffner, German malarialogist). When tertian-infected erythrocytes are stained with dye after the Romanowsky formula, the protoplasm is often speckled with chromatophilic particles — Schüffner’s dots — which are at first very fine, but soon become coarser and more prominent. They are characteristic of P. vivax and P. ovale.

Sclerites (G. skleros, hard). Protective, chitinous plates connected by slightly chitinized membranes to permit flexibility of movement of Arthropods.

Scolex (G. skolex, a worm). The head of a tapeworm, either in the larval or adult stage.

Scutellum (L. scutellum, dim. of scutum, shield). The small, posterior lobe of the meso thorax of a dipterous insect, which overhangs the mesonotum.

Scutum (L. scutum, a shield). The large anterior part of the dorsal covering of mesothorax in the family DIPTERA. It may be divided into two nearly equal parts by a transverse groove. In the IXODINAE (hard ticks), more or less of the dorsum of the body is protected by a chitinous shield also called the scutum.

Seminal vesicle (L. semen, seed; vesicula, little bladder). A saclike organ of the male genital system which is a reservoir for semen.

Serrate (L. serra, a saw). Notched or toothed on the edge.

Setae (L. seta, bristle). Chitinous bristles, commonly found on most segments of annelid worms.

Setose (L. seta, bristle). Having bristles.

Sigmoidoscope (G. sigmoeides, sigmoid, + skopeo, I view). An appliance for the inspection of the sigmoid flexure of the large intestine.

Signet rings (L. signum, mark; A.S. hring, ring). The ringlike forms malarial parasites assume when they enter the red blood cells.

Simulium (L. simulus, snub-nosed). A genus of small, hump-backed flies, also known as black flies or buffalo gnats. They are vicious biters by day and attack cattle and other animals in swarms.

Siphon (G. siphon, a tube). A breathing appendage on the eighth abdominal segment of non-anophelines or culicine mosquitoes.
Siphonaptera (G. siphon, a tube, + apterus, wingless). Degenerate insects without wings, with sucking mouth-parts and undergoing complete metamorphosis.

Somatic (G. somatikos, bodily). Pertaining to the body as a whole or to the wall of the body.

Somite (G. soma, body). Primitive segment; one of the pair of masses, formed from the mesoderm between the digestive canal and the surface of the body in the embryo, from which the segment, provertebra, or metamere is developed.

Sparganum (G. spargan, to be distended). The sparganum is a polymorphous larva, which may multiply by budding. In the cases described they are found in subcutaneous tissues, in the intermuscular fasciae as well as in the walls of the alimentary canal, mesentery, kidneys, lungs, heart and brain. The adult stage where determined has proved to be a diphyllobothrid tapeworm in most instances.

Spermatheca (G. sperma, seed, + theke, a box). Chambers where, after copulation, spermatozoa are stored for the fertilization of ova.

Spermatozoa (G. sperma (spermato-), seed, + zoon, animal). Male elements capable of fecundating the ovum. Each consists of an oval head and a long, mobile cilium or tail and constitutes the essential constituent of the semen.

Sphenopterygoid (G. sphen, wedge, + pteryx (pteryg-), wing, + eidos, resemblance). Pertaining to the body of the sphenoid bone and to the pterygoid process.

Sphincter (G. sphinkter, a band or lace). A muscle surrounding and closing an orifice.

Spicules (L. spiculum, a point). Elongated, rodlike bodies, found in the posterior part of nematode worms. They are protrusible and function as accessory, male reproductive organs.

Spiracle (L. spirare, to breathe). A breathing orifice, as in the tracheal openings of insects.

Spirillum (L. spirillum, a coil). A genus of motile organisms characterized by having spiral bodies and sometimes assisted in locomotion by a terminal flagellum.

Spirochaete (spirochete) (G. speira, a coil or twist, + chaite, hair). A general term used for certain spiral organisms which have flexible bodies. They have been variously classified with bacteria, algae, and protozoa. Descriptions of their minute structure differ widely.

Spore (G. sporos, seed). A germ or seedlet of one of the lower animals or plants. The active reproductive body of sporozoans, often called the sporozoite.
Sporoblast (G. sporos, seed, + blastos, germ). The precursor of the spore or infective agent.

Sporocyst (G. sporos, seed, + kystis, bladder). A case or cyst containing spores.

Sporogenesis (G. sporos, seed, + genesis, production). Reproduction by means of spores.

Sporogony (G. sporos, seed, + gone, generation). Same as sporogenesis; more specifically as applied to evolution of sporozoites in the body of anopheline mosquitoes, following fusion of the gametes; similarly applied to coccidia (usually outside the host).

Sporozoa (G. sporos, seed, + zoon, a living animal). A class of protozoa having no special organs of movement such as flagella or cilia, and which are disseminated by means of spores or spore-related reproductive bodies.

Sporozoite (G. sporos, seed, + zoon, animal). One of the young active spores of a sporozoan produced by division of the passive spores contained in the oöcyst (sporocyst).

Sporulation (L. sporula, dim. of spora, spore, + tio[n], a suffix denoting an act or state). Spore formation. SYN. Multiple fission.

Sprue (sprew) (D. spruw or sprouw, a name given by the Dutch in Java to aphthae tropicae). A chronic disease occurring mostly in southeastern Asia and characterized by a chronic catarrhal inflammation of the entire alimentary tract.

Sternum (G. sternum, the chest). See Notum. SYN. Sternite.

Stigma (G. stigma, a mark). An opaque spot on hymenopterous insects near the middle of the anterior margin of the front wing. (In botany, applied to the pollen-receiving portion of the pistil.)

Stigmata (G. stigmata, plural of stigma, a mark). Small spots or marks, usually applied to the openings of the tracheae in insects. In this group there are usually ten pairs of stigmata (but often less); namely, two larger pairs on the thorax, and eight smaller pairs on the anterior abdominal segments. The spiracles may be opened and closed by muscles or valves.

Stomoxys calcitrans (G. stoma, mouth, + oxyx, sharp; L. calcitrare, to kick, denoting the efforts of cattle to rid themselves of this insect). A species of biting fly, resembling in size and general appearance the common housefly.

Strobila (G. strobile, a twist of lint). An adult tapeworm.

Strongyloides (G. strongylos, round, + eidos, resemblance). A genus of nematode worms, parasitic in animals. It enters the body through the skin, goes through the blood stream to the lungs, up to the epiglottis,
where it is swallowed and develops into the parasitic generation in the intestine.


**Stylets** (L. *stiletto*, dim. of *stilus* or *stylus*, a stake, a pen). Piercing organs of various kinds of insects, particularly biting bugs and lice.

**Subpatent period** (L. *sub*, under, + *patere*, to lie open). The parasitological period of a disease occurring after the patent period when the parasites are present but cannot be directly demonstrated. Proof of their presence is that another patent period may follow when the presence of the parasites can be proved, as in amoebiasis and malaria.

**Sucker** (L. *sugere*, suck). An organ by which a worm attaches itself to other animals or objects.

**Suctorial** (L. *sugere*, to suck). Adapted for sucking.

**Suctorial disc** (L. *sugere*, to suck). An organ serving to adhere or to draw up fluid by suction.

**Symbiosis** (G. syn, with, + *bios*, life, mode of living). The intimate association of two or more organisms for their mutual advantage. syn. Mutualism, commensalism.

**Syngamy** (G. syn, together, + *gamos*, marriage). Sexual union; conjugation of male and female reproductive cells.

**Tabanus** (L. *tabanus*, a gadfly). A genus of biting flies; gadflies, horseflies.

**Taenia, Tenia** (G. *tainia*, band, tape). A genus of tapeworm which parasitizes man (principally as primary host) and other vertebrates. They live attached to the wall of the small intestine. The two important species of man are *T. saginata*, the beef tapeworm, and *T. solium*, the pork tapeworm. The infection is acquired by the consumption of raw beef or pork (respectively) containing the larvae known as cysticerci.

**Tarsus** (G. *tarsos*, a wicker-work frame). In insects, the small segments forming the distal termination of the leg and articulating with the tibia.

**Temporal** (L. *tempus* (tempor), temple). Of or pertaining to the temple or temples or the sides of the skull behind the orbits.

**Tentacle** (L. *tentaculum*, a feeler). A slender process for feeling, prehension, or locomotion in the lower vertebrates and the invertebrates.

**Tergite** (L. *tergum*, the back). See Notum. syn. Tergum, notum.


**Tertian** (L. *tertianus*, relating to a third [thing]). Recurring every third day, as tertian fever, a form of malaria.

**Thorax** (G. *thorax*, breastplate). That part of the body which is between the cervical or cephalic and abdominal segments.
Thread cells (A.S. *thraed* from *thrawan*, twist). Cells which by the discharge of their nematocyst batteries can inflict painful stings. See Nematocyst.

Tibia (L. *tibia*, pipe, flute). In insects, the fourth segment of the leg, articulating proximally with the femur and distally with the tarsus.

Tracheae (G. *tracheia*, rough, from the rings of gristle of which it is largely composed). The air-conveying tubules forming the respiratory system of insects and other arthropods.

Tracheal gills (G. *tracheia*, rough; Dan. *giaelle*, gill). A system of respiration found on abdominal segments in some aquatic forms of insect larvae. In Culicidae, it is found on the last abdominal segment. The tracheal gills obtain oxygen from the water as do the blood gills which they resemble in structure except that the capillaries are represented by fine tracheae.


Trematoda (G. *trematodes*, pierced with holes). Platyhelminthes which are true parasites during a large portion of their life. They derive their name from the fact that they usually have large suckers. Some have a relatively simple life history without alternation of generation (Mongenea), others have a complicated life history with an alternation of generation (Digenea). The adults are covered with nonciliated integument. A circulatory system is usually lacking. SYN. Flukes.

Trenchant (O.F. ppr. of *trencher*, cut). Sharp; cutting.


Trichina (G. *thrix* [trich-], hair, + *ina*, termination of feminine nouns). A genus of nematode worms, more correctly called Trichinella. SYN. Trichinella.

Trichinella (G. *thrix* [trich-], hair, + *ella*, dim.). A genus of parasitic nematode worms. When raw flesh infected with the larval cysts is eaten, the cysts are separated from the flesh in the stomach, excyst in the duodenum and invade the mucosa to mature. The viviparous larvae discharged into the lymph spaces and circulatory system are dispersed throughout the body coming to rest particularly in muscle such as the diaphragm and deltoid. SYN. Trichina.


Trichiniasis (G. *thrix* [trich-], hair, + *iassis*, denoting a condition). A painful and frequently fatal disease produced in man by eating meat, especially the flesh of pigs, raw or insufficiently cooked and infested.
with the larvae of *Trichinella spiralis*. SYN. Trichinosis, trichinellosis.

**Trichinosis** (G. *thrix* [trich-], hair, + *osis*, denoting a condition). See Trichiniasis. SYN. Trichiniasis, trichinellosis, trichinelliosis.

**Trichocephalus** (G. *thrix* [trich-], a hair, + *kephale*, head). See Trichuris. SYN. Trichuris.


**Trichuris** (G. *tlzrix* [trich-l, hair, + *aura*, tail). A genus of parasitic nematode worms, also known as “whipworms.” The adults live in the cecum. Infection results from the ingestion of the fully embryonated eggs. SYN. Trichocephalus.

**Trochanter** (G. *trocbanter*, originally the head of the femur). In insects, the second segment of the leg, articulating proximally with the coxa and distally with the femur.

**Trophonucleus** (G. *traphe*, nourishment, + L. *nucleus*, dim. of *nux*, nut). The nucleus in protozoa which is concerned with vegetative and metabolic functions. It is distinct from the gonad or germ or sex nucleus (micronucleus). See Micronucleus.

**Trophozoite** (G. *trope*, food, + *zoon*, an animal). The growing or feeding stage of protozoa.


**Trypanosoma** (G. *trypanon*, auger, + *soma*, body). Flagellates of the genus *Trypanosoma* which have both vertebrate and invertebrate hosts. They resemble those of the genus *Herpetomonas* in evolving through four flagellate types: leishmania, crithidia, leptomonas, and trypanosome. In nature they pass from vertebrate to vertebrate through an intermediary insect host.

**Trypanosomiasis** (G. *trypanon*, auger, + *soma*, body). Any of the several diseases due to infection with the various species of Trypanosoma.

**Tularemia.** A specific infectious disease due to *Pasteurella (Bacterium) tularense*, transmitted from rodents to man by the bite of a bloodsucking insect or the handling of infected rodents.

**Tumbler** (A.S. *tumbien*, dance). The aquatic pupa of *Culicidae*. In this non-feeding though very active stage, there is a pair of breathing “trumpets” situated dorsally on the cephalothorax. The pupa is sensitive to disturbances of the water, letting go suddenly and darting with a tumbling motion to shelter and after a few moments rising with little motion to the surface where the breathing trumpets break the surface film and contact with air is re-established. The pupal stage is quite short, usually two to three days under optimum conditions.

**Uncinaria** (L. *uncimus*, a hook). A genus of nematode worms, now only employed synonymously. See Necator americanus.
Undulating membrane (L. unda, a wave; membrana, membrane). A delicate lateral membranous expansion of trypanosomes and similar organisms.

Urticaria (L. urtica, nettle). A lesion of the skin characterized by the development of wheals, which give rise to sensations of burning and itching. These are indicative of vasomotor disturbances, due to direct action of substances of an allergic or purely chemical nature, or the ingestion of certain foods, etc. syn. Hives.

Uterine pore (L. uterus, uterus; G. poros, pore). An opening through which eggs are discharged from the uterus.

Vacuole (L. vacuolum, an empty space). A cavity or vesicle in cell protoplasm.

Vagina (L. vagina, sheath). The musculomembranous canal extending from the vulvar opening to the cervix uteri.

Vagina dentis (L. vagina, sheath; dens, tooth). A sheath covering the fang of viperine snakes when folded back against the roof of the mouth.

Vas deferens (L. vas, vessel; deferre, to carry down). The excretory duct of the testis.

Venenosalivary duct (L. venenum, poison, + saliva, saliva; ducere, to lead). Applied to the common salivary duct in the hypopharynx of female anopheline mosquitoes. Through it pass the malaria plasmodia sporozoites in saliva to be injected into the wound-puncture made at time of biting.

Venom (L. venenum, poison). Poison, especially a poison secreted by certain reptiles and insects.

Ventral (L. ventrum, the belly). Pertaining to the front surface of an animal. See Ventrum.

Ventrum (L. ventrum, the belly). The front portion of an animal. See Ventral.

Vermicide (L. vermis, worm, + caedere, to kill). A substance which kills worms; a drug to kill parasitic worms of the intestines. See Vermifuge.

Vermifuge (L. vermis, worm, + fugare, to chase away). A medicine that expels worms from the bodies of animals. See Vermicide.

Vermiculus (L. vermiculus, dim. of vermis, worm). See Ookinete.

Verminous (L. vermis, worm). Infested with worms, or caused by worms, as verminous diseases.

Villi (L. villus, a tuft of hair). Tufts of hair, or hairlike processes or projections of a mucous membrane giving it a velvety appearance.

Viperidae (L. vipera, viper). A family of thick-bodied poisonous snakes with movable maxillary bones which carry the large tubular fangs.

Viperinæ (L. viperus, pertaining to a viper). A subfamily of the Viperidae in which "loreal pits" are absent. They constitute the true vipers and are confined to the Old World, being most abundant in Africa.
Virus (L. virus, poison). A minute agent (often invisible) responsible for various pathological conditions of animals including man, birds, fish, insects, plants, and bacteria. They range in size from 8 to 275 millimicrons (μm), are usually filterable, and cannot be cultivated in the absence of living, susceptible cells.

Vitelline membrane (L. vitellus, yolk; membrana, membrane). A membrane enclosing the egg proper. In hen's egg it is represented by a very thin transparent membrane enclosing the ball of yolk. In many invertebrates, it is not present until the egg is fertilized, then it is immediately secreted by the egg in order to prevent other spermatozoa from entering.

Vitellus (L. vitellus, yolk). The yolk of egg; the germinal portion of the ovum together with the substance destined for the nutrition of the embryo.

Viviparous (L. vivus, alive, + parere, to bear). Producing living young by true birth, as in mammals, and not by hatching from eggs, as in oviparous and ovoviviparous animals; also often applied to the bringing forth of young which have been hatched from eggs within the body of the parent.

Volutin (L. voluta, roll, a spiral scroll). A cytoplasmic chromatin-like substance occurring in bacteria, certain other lower plants, and protozoa. It is a nucleoprotein and probably related to or synonymous with metachromatic granules.

Vulva (L. vulva, a wrapping or covering). The external organs of generation in the female.

Wiggletail. The aquatic larvae of various insects especially that of the mosquito. Also called wiggler.

Wuchereria. The genus of filarial worm which causes elephantiasis in man (the definitive host). The larvae are carried to him by the mosquito (Culex fatigans and others).

Xenopsylla (G. xenon, a stranger, + psylla, a flea). A genus of fleas belonging to the family Siphonaptera. X. cheopis is a carrier of plague bacilli (Pasteurella pestis) from rat to rat and from rat to man.

Ziemann's stippling (Ziemann, German scientist). A name suggested for quartan stippling when the red corpuscles are stained by the Romanowsky method. It consists of dots and points smaller and less distinct than those described for the benign tertian parasite.

Zoophilous (G. zoön, animal + philos, love). Preferring the blood of lower animals to that of man as applied to feeding habits of insects, particularly mosquitoes.

Zoophyton (G. zoön, animal, + phyton, plant). An animal resembling a plant. See Phytozoon.
Zygosis (G. zygosis, a joining). True conjugation or sexual union of two unicellular organisms, consisting essentially in the fusion of the nuclei of the two cells.

Zygote (G. zygosis, a joining). A body or cyst formed by the conjugation of two gametes. syn. Oospore.
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