STUDIES ON THE PATHOLOGY OF *IPOMOEA CARNEA* PLANT TOXICITY IN GOATS

By
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September, 1992

CERTIFICATE

Smt.Ch. Srilatha has satisfactorily prosecuted the course of research and that the thesis entitled **"STUDIES ON THE PATHOLOGY OF *IPOMOEA CARNEA* PLANT TOXICITY IN GOATS"** submitted is the result of original research work and is of sufficiently high standard to warrant its presentation to the examination. I also certify that the thesis or part thereof has not been previously submitted by her for a degree of any university.

Date: 18-9-92

Major Advisor

CERTIFICATE

This is to certify that the thesis entitled **"STUDIES ON THE PATHOLOGY OF *IPOMOEA CARNEA* PLANT TOXICITY IN GOATS"** submitted in partial fulfilment of the requirements for the degree of **'DOCTOR OF PHILOSOPHY'** of the Andhra Pradesh Agricultural University, Hyderabad is a record of the bonafide research work carried out by **Smt.Ch.Srilatha** under my guidance and supervision. The subject of the thesis has been approved by the Student's Advisory Committee.

No part of the thesis has been submitted for any other degree or diploma. The published part has been fully acknowledged. All the assistance and help received during the course of the investigations have been duly acknowledged by the author of the thesis.

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ABSTRACT

In summer and drought periods animals may be forced to eat the poisonous plants which are adopted to semi-arid conditions. *Ipomoea carnea* has long been incriminated as toxic to livestock particularly goats. Goats which graze on *I. carnea* during dry periods become prone to its toxic effects. The available information on the toxicity of *I. carnea* was scanty. Hence, an attempt was made to study the haematological, biochemical and pathological changes in goats on feeding *I. carnea* leaves.

Eighteen goats of 4-5 months age were utilized in the present work. Goats were randomly divided into three groups of six animals each. Group I and II constituted the experimental groups, while group III constituted the control. Animals in group I and II were fed with fresh leaves of *I. carnea* at the rate of 50 g and 5 g/kg body weight, respectively. In addition to that all the animals

including control were fed with green fodder and concentrates *ad libitum*. Experiment was continued for a period of 16 weeks.

Animals were monitored for clinical symptoms regularly and body weights were recorded weekly throughout the experiment. Haematological and biochemical studies were carried out weekly using standard procedures. Detailed postmortem examination was carried out on goats died during the experiment and those sacrificed alongwith the control group at the end of the experiment.

Clinical manifestations such as increased body temperature (after 10 days), anorexia, dullness, diarrhoea (after 23 days), marked decrease in body weight, respiratory distress (after 42 days), nervous symptoms (after 18-20 days) and death (between 15-70 days) were noticed in group I animals. The nervous symptoms like incoordination, awkward posture, lassitude, somnolence and paralysis of hind limbs with knuckling movement were observed. Emaciation, rough hair coat, alopecia and nervous symptoms (after 54-60 days) were pronounced in group II animals apart from general symptoms. Characteristic swinging and shaking of the head, severe muscular spasms and recumbency were also recorded in group II.

The values of RBC, WBC, Hb, PCV, MCV and MCH were significantly ($P < 0.01$) decreased during the period of study in both experimental groups. ESR values were significantly ($P < 0.01$) increased in both groups. These changes might be due to bone marrow depression and hepatic injury leading to hypoproteinaemia.

Significant decrease ($P < 0.01$) in total proteins and albumins was observed in experimental groups and this can be attributed to liver damage. Cholesterol levels were not affected by *I. carnea* feeding. Significant ($P < 0.01$) increase in enzyme levels such as Asparate amino transferase (AST) and serum Alkaline phosphatase (ASP) observed was indicated to the tissue injury in the organs.

At necropsy hydrothorax and hydropericardium were noticed in both experimental groups. Gelatinization of the coronary and renal fat was observed only in group II. Severe congestion of the meningeal blood vessels was observed in both groups. In group I animals congestion and haemorrhages were prominent in all the organs. In group II lesions were of chronic nature.

In group I congestion and haemorrhages were observed in cerebrum, cerebellum and spinal cord apart from neuronal degeneration, satellitosis and neuronophagia. In group II patchy serous effusions with loose textured appearance in the white matter of cerebrum were observed. Most prominent lesions were observed in the cerebellum. In group I focal loss of Purkinje cells was observed with oedema of the cell layer. Total loss of Purkinje cells with reticulated appearance in between molecular and granular layers was observed in group II. Thinning of molecular and granular layers was observed in both groups Congestion and focal haemorrhages were seen in spinal cord of group I animals, whereas demyelinative changes with swollen axons were observed in group II. These lesions can be correlated with the nervous symptoms observed.

Sinusoidal congestion and haemorrhages were prominent in the liver of group I animals. In group II mild bile duct proliferation with fibrosis and focal leucocytic infiltration was seen.

Congestion and haemorrhages throughout the parenchyma of kidneys in group I animals besides mild degenerative changes in the tubules were observed. In group II chronic changes such as thickening of the capsule, shrinkage of the glomeruli and fibrosis around the Bowman's capsules were noticed. Desquamation and cast-formation were seen in the tubules.

Congestion and oedema of lungs was seen in both the groups. In group II the alveolar walls were thickened. Interlobular septa and subpleural layers of connective tissue were also thickened.

In spleen congestion was observed in both groups. In addition, marked thickening of the connective tissue in the walls of the sinuses, reticulum of the pulp and fibrous trabeculae was observed in group II.

In lymph nodes severe congestion was observed in both the groups. Mild lympoid depeletion was seen in group II.

Ipomoea carnea appeared to affect chiefly the CNS i.e. brain and spinal cord. Purkinje layer of cerebellum was primarily affected resulting in incoordination and ataxia. *I. carnea* toxin affected the blood vessels resulting in haemorrhages in different organs. The toxic lesions appeared to be more pronounced in group II because of prolonged feeding probably due to cumulative effect.

CHAPTER 1

INTRODUCTION

CHAPTER - I

1. INTRODUCTION

Goat population in India is about 105 millions of which Andhra Pradesh alone has 38 millions (FAO 1988). Goats are reared by small farmers because of their effective utilization of feed resources. The goat is a versatile animal. It is known as the "poor man's cow" in India and as "wet nurse" of infants in Europe. Rural economy of the country is very much dependent on the welfare of the goat population because of its valuable contribution to economy in terms of the value of milk, meat, hide and hair. Goats kept in villages are solely maintained on grazing. They have a set of feeding habits. They utilize a variety of feed resources such as lucerne, napier grasses, cowpea, cabbage and cauliflower leaves, shrubs and weeds of different kinds and leaves of trees such as babul, neem, tamarind etc.

Due to their feeding habits at times goats may have accessibility to the feeding of certain plants which are poisonous in nature. It was reported that among the various causes of sickness in goats, poisonous plants were responsible to the extent of 8.7% (Keeler *et al* 1978). The toxicity of poisonous plants to farm animals was investigated in many countries (Gopinath and Ford 1969, Adam *et al* 1973 and Hofstee 1989).

Ipomoea carnea, a tropical plant of the family Convolvulaceae, has for long been incriminated as the cause of several instances of poisoning in domestic animals, particularly goats. The plant grows extensively in almost all parts of the country mostly as a hedge plant. Indeed, it is so resistant to drought that it can grow lavishly anywhere along valleys, near ponds or open lands even in localities where pasture plants can hardly be detected. Ease propagation, luxuriant growth and extensive availability of the plant made it easily accessible to livestock. During periods of drought, goats have a tendency to eat the leaves of *I.carnea* due to acute scarcity of fodder. They develop varying degrees of

neurotoxic symptoms depending on the quantity of leaves consumed and the duration of consumption (Tirkey *et al* 1987).

The literature on toxicity of *I. carnea* leaves was scanty. Hence, it was considered worthwhile to study the effects of the toxicity of *I. carnea* leaves in goats with the following parameters.

1. Haematological changes like haemoglobin (Hb), total erythrocyte (RBC) count, total leucocyte (WBC) count, packed cell volume (PCV) and erythrocyte sedementation rate (ESR).

2. Changes in total serum proteins, albumins and cholesterol.

3. Changes in the enzyme levels of Asparate amino transferase (AST) and Alkaline phosphatase in serum (SAP).

4. Extent of damage through pathological changes in organs.

CHAPTER 2

REVIEW OF LITERATURE

CHAPTER - II

2. REVIEW OF LITERATURE

2.1 HABITATION OF *IPOMOEA CARNEA*

Haines (1922) described *Ipomoea carnea* as a large straggling shrub with milky juice. It belongs to the family Convolvulaceae. It is said to be native of South Africa and introduced to India as an ornamental plant (Wealth of India 1959). The plant usually grows in tropical and subtropical countries as a hedge plant which can be as high as ten feet (Michaiel 1965). The plant is so resistant to drought that it can grow lavishly any where along valleys, near ponds or open lands, even in localities where pasture plants can hardly be detected. It is commonly known as "morning glory". It is prostrate in habit and develops elongated lateral stems up to 4 or 5 feet in length. The leaves arise singly at intervals along the stem. Leaf is heart shaped with two large rounded basal lobes and apex. The plant produces masses of green buds from which protrude the shiny funnel shaped flowers which have no perfume. The flowers are pink or violet in colour and borne in short clusters of one to three (Fig.1).

The plant has long been incriminated as toxic to livestock, particularly goats (Idris *et al* 1973). Goats and Sheep which graze on the leaves of *I.carnea* during summer and drought period become prone to its toxic effects (Tirkey *et al* 1987).

Other plants of the genus *Ipomoea* such as *I. batatas, I. muelleri* and *I. calobra* were also found to contain a toxic principle. These plants have been regarded as a probable cause of mortality in livestock (Everist 1947, Gardner and Bennetts 1956, Watt and Breyer - Brandwijk 1962 and Gardiner *et al* 1965).

Figure 1. *Ipomoea carnea* plant.

2.2 CHEMICAL NATURE

Crusellas (1946) studied the botanical, chemical and pharmacological aspects of the plant in detail. The author could detect in the plant a water soluble carbohydrate -Ipomose and a glucoside in the leaf extract whose aglucone component contained an anthracine. Starch or free reducing sugars were not present. It also contained a gum that gave galactose on hydrolysis and saponins containing considerable portions of sapotoxins. Alkaloids were absent. In the contrary, the presence of alkaloids both in the aqueous and the ether extracts of *I. carnea* leaves was observed by Arora and Ahmad (1974), Agrawal and Upadhyaya (1978) and Tirkey *et al* (1988). They identified the presence of other substances such as reducing sugars, tannins and glycosides only in the aqueous extract.

2.3 PHARMACOLOGICAL AND TOXICOLOGICAL STUDIES

The fifty per cent ethanolic extract (excluding roots) of *I.carnea* plant exerted gross behavioural effects and potentiated barbiturate induced hypnosis in mice. The LD_{50} of the extract was 1 g/kg body weight when administered intraperitoneally in albino mice (Bhakuni *et al* 1971).

Toxicological studies on the aqueous extract of *I. carnea* roots showed general depression in mice at a dose of 3 g/kg body weight. Administration of 1 g/kg body weight for 21 days did not produce any change in body weight or internal organs. The aqueous extract produced a marked non-specific hypotensive effect (Rana *et al* 1978).

The LD_{50} of the aqueous extract of the leaves of *I. carnea* was determined to be 250 mg/kg body weight by intraperitoneal administration and more than 4 g/kg body weight when administered orally in mice (Arora and Ahmad 1974).

The non-alkaloidal, non-saponifiable fraction of *I. carnea* leaves revealed non specific central nervous system (CNS) depressant activity with muscle relaxant property. The fraction had no tranquillizing activity. (Bhattacharya *et al* 1975).

2.4 BIOCHEMICAL EFFECTS

Adam *et al* (1973) have studied the biochemical responses elicited by *I. carnea* leaves toxicity in cattle, sheep and goats. All animals showed increased concentration of Asparate amino transferase (AST). The concentration of AST in the serum of goats started to rise 10 to 15 days after the commencement of dosing, reaching a peak on 18th day. The concentration of enzyme in the serum returned to normal at the time of death. There was no change in the Asparate alanine transferase (APT). The levels of AST increased to a peak in the serum of sheep on the 22nd and 32nd day after dosing and the levels returned to normal by 107th day. There was no significant change in the enzyme activity in calves.

2.5 HAEMATOLOGICAL CHANGES

Tartour *et al* (1974) observed severe leucopenia in goats when fed with fresh leaves of *I. carnea* at the rate of 5 g/kg body weight/day. Findings of their study indicated that both short and protracted courses of toxicity were characterized by marked haematological changes from the begining of administration. In early stages there was normocytic and hypochromic anaemia. A sharp fall in red blood cell count was observed. A progressive and gradual reduction in leucocytes from 9.75 to 3.88 thousands/cu.mm was also observed. Levels below 4 per cent for haemoglobin and 15 per cent for packed cell volume were sometimes recorded.

Gardiner *et al* (1965) reported that *I. muelleri* plant intoxication in sheep causes severe leucopenia with leucocyte count reaching levels of 3 to 4 x thousand/cu.mm after 3 or 4 weeks.

2.6 NATURAL INCIDENCE

Maselle *et al* (1989) reported an epidemic of posterior weakness in Norwegian goats with high fever, collapse and death in *I. carnea* intoxication. Clinical, post mortem and histopathological findings indicated gastroenteritis, with necrotic foci in liver and soft kidneys.

2.7 EXPERIMENTAL STUDIES

The symptoms and lesions of *Ipomoea carnea* toxicity were studied in different livestock species by different authors.

2.7.1 Symptoms

Adam *et al* (1973) administered an aqueous suspension of *I. carnea* fresh leaves to goats at a dose level of 5 g/kg body weight and observed initially anorexia, dullness and depression. Different degrees of paresis of hind limbs, abduction, incoordination and staggering were noticed as the toxicity progressed. Respiratory distress and mortality were also observed with different degrees of individual susceptibility.

Tirkey *et al* (1987) fed fresh leaves with the petiole of the plant *I. carnea* to three goats at a dose of 0.5 kg per animal daily and reported death of one goat on third day due to acute neurotoxicity after showing dullness, ataxia, incoordination of gait and paralysis of hindquarters, diarrhoea, lacrimation and nasal discharge. The other two goats died on 27th and 55th day of feeding of the leaves by showing well defined neurotoxic symptoms such as ataxia and incoordination, whereas Misra and Misra (1965) failed to record any adverse

symptom when crude leaf extract of *I. carnea* was given orally to sheep and goat at a dose of 800 ml per animal.

Tokarnia *et al* (1960) noticed lack of appetite and listlessness in goats on feeding of *I. fistulosa* leaves. The goats died between 29 and 81 days after having been sick for 2 to 14 days.

Gardiner *et al* (1965) recorded a progressive deterioration in condition, jerky gait, knuckling of hind feet, awkward swaying, rapid panting respiration, and tiring easily in sheep fed on *I. muelleri*. Weaners were more susceptible than adults and exhibited marked salivation with froth and knuckling of the hind feet leading to death.

Adam *et al* (1973) observed profound depression, anorexia, arching of back and weakness of hind limbs accompanied by staggering upon feeding of *I. carnea* fresh leaves at the rate of 5 g/kg body weight. Dyspnoea, pallor of the visible mucous membranes, wasting, weakness of the lumbar region and paralysis of hind limbs followed by weakness and later paralysis of the fore limbs, rough hair coat, progressive weakness, emaciation, recumbency and finally death were observed.

Dobereiner *et al* (1960) noticed nervous symptoms in sheep upon feeding of *I. asarifolia.*

Variation in the individual susceptibility of goats when fed with *I. fistulosa* was noticed by Tokarnia *et al* (1960).

2.7.2 Lesions in Goats

Specific gross lesions in the liver, heart, kidneys and brain together with serous effusions in serous cavities were observed by different authors on feeding *I. carnea* leaves at different dose levels.

2.7.2.1 Liver. Pale, enlarged and haemorrhagic livers with irregular surfaces were noticed grossly. Congestion of the sinusoids, fatty changes, reduction in cytoplasmic basophilia of the hepatocytes and portal fibrosis with infiltration of mononuclear cells and few polymorphonuclears were observed microscopically (Adam *et al* 1973).

In *I. carnea* toxicity Tirkey *et al* (1987) observed mild degenerative changes in livers of goats died on 27th day. Vacuolar degeneration around the central vein of hepatocytes was found. The vacuoles were microscopic in size. Hypertrophy of the Kupffer cells and bile duct proliferation were also noticed. Diffuse fatty changes were reported in hepatocytes of goats died on 56th day. Pronounced proliferation of connective tissue and congestion of the blood vessels were also observed.

2.7.2.2 Kidneys. Dobereiner *et al* (1960) reported pronounced hyperaemia in kidneys of goats fed with *I. asarifolia*. Grossly, congestion and ecchymotic haemorrhages at cortico-medullary junctions in both kidneys were observed by Adam *et al* (1973).

Wilson *et al* (1970) found toxic cellular changes in liver and kidneys of mice when ether extract of *I. batatas* was administered.

Microscopically, tubular casts and vacuolation and desquamation of tubular epithelial cells were observed by Tirkey *et al* (1987). Nephrosis in the convoluted tubules, albuminous casts in the proximal and distal convoluted tubules and in collecting tubules, and desquamation in some of the convoluted tubules were also observed in a goat which died on the 56th day. But no change was evident in the glomeruli of the goats died on 27th day of feeding 0.5 kg of *I. carnea* leaves daily.

2.7.2.3 Heart. Flabby heart, petechiae and ecchymoses in the subendocardium were reported by Adam *et al* (1973) in *I. carnea* plant intoxication in goats.

Tirkey *et al* (1987) indicated that the histological alterations in myocardium were of mild nature. They observed focal disintegration and fragmentation of the myofibrils and mild mononuclear cell infiltration in between the muscle fibres in goats died on 27th day of feeding *I.carnea* leaves at the rate of 0.5 kg/animal daily. Some of the cardiac muscle fibres showed degenerative changes characterized by loss of sarcoplasm and fatty changes.

2.7.2.4 Intestines. Lesions in gastrointestinal tract were oedema of the abomasal mucosa, petechial haemorrhages in the first portion of the small intestine and thickening of the mucosa of the second part of the small intestine on feeding of *I. asarifolia*. Histopathological studies revealed lymphocytic infiltration and oedema in the mucosa and submucosa of the intestines (Dobereiner et al 1960).

2.7.2.5 Lungs. Oedematous lungs were detected by Adam *et al* (1973) in *I. carnea* plant toxicity in goats.

2.7.2.6 Spleen. Soft spleen and mild degree of splenic hypertrophy were observed in sheep in *I. muelleri* intoxication (Gardiner *et al* 1965).

2.7.2.7 Brain and Spinal cord. Tirkey *et al* (1987) noticed significant changes in the grey matter of the spinal cord. Perineuronal vacuolation, satellitosis and neuronophagia were observed. In the central canal a small amount of eosin-stained homogenous material was seen.

Dobereiner *et al* (1960) observed small haemorrhages in the central nervous system upon feeding of *I. asarifolia* in ruminants.

2.7.3 Lesions in Sheep

Adam *et al* (1973) also observed the toxic changes in the organs of sheep upon feeding *I. carnea*. Grossly blood in the peritoneal and thoracic cavities; congestion in lungs, brain and kidneys and flabby heart were reported.

Liver was firm and pale with pale spots on the surface. Peritoneal, pericardial and thoracic cavities contained small quantities of straw coloured fluid. Congestion in kidneys was observed, particularly at the cortico-medullary junction.

Gardiner *et al* (1965) observed mild degree of adrenal and splenic hypertrophy in *I. muelleri* intoxication.

Microscopically, cytoplasmic fatty vacuolation in few hepatocytes, necrosis of isolated cells, infiltration of mononuclear and polymorphonuclear cells and focal portal fibrosis were observed by Adam *et al* (1973) in *I. carnea* toxicity.

In *I. muelleri* intoxication Gardiner *et al* (1965) observed livers containing swollen Kupffer cells laden with bilirubin. Marked siderosis was noticed in the spleen. Nuclear pyknosis, atrophy and fatty vacuolation of the muscle cells of the heart were reported. Heart tissues also revealed focal areas of lymphocytosis, necrosis and proliferation of sarcolemmal nuclei. There was an increased cellularity of the glomeruli. A little or no fat in the usual depots after a few weeks of feeding of *I. muelleri* was reported.

Tokarnia *et al* (1960) observed progressive weakness and rough hair coat in sheep fed on *I. fistulosa* plant.

2.7.4 Lesions in Cattle

Clinical responses to *I. carnea* differed in the two calves according to Adam *et al* (1973). A calf appeared dull with loss of appetite after 76 days of dosing. The other calf did not exhibit any obvious abnormality.

CHAPTER 3

MATERIALS AND METHODS

CHAPTER III

3. MATERIALS AND METHODS

Eighteen non descriptive goats of 4-5 months age, of both sexes and weighing 8 to 9 kg were used in this experiment. These were screened for any helminthic and protozoal infections for about a week. The goats were randomly divided into three groups of 6 animals each and housed separately. Group I & II constituted the experimental groups while group III constituted the control. All the animals in the group I, II and III were stall fed with green fodder and concentrates *ad libitum*. Accessibility to drinking water was provided round the clock. In addition, group I animals were fed with *I. carnea* fresh leaves at a rate 50 g/kg body weight whereas group II animals were given *I. carnea* leaves at a rate of 5 g/kg body weight daily. The experiment was continued for a period of 16 weeks.

3.1 FEEDING OF *IPOMOEA CARNEA* LEAVES

The fresh leaves of *I. carnea* were procured daily from its natural habitations located in and around Tirupati.

The required quantity of fresh leaves of *I. carnea* was provided during morning hours before giving fodder and concentrates to experimental animals. In order to ensure correct dosing the leaves were hand fed to each animal until the total calculated quantity was consumed.

The body weights of animals for all the three groups were recorded at weekly intervals. The animals were regularly monitored for clinical symptoms.

3.2 HAEMATOLOGICAL STUDIES

The blood for the haematological studies was collected at weekly intervals using EDTA as anticoagulant at 1 mg per ml. Total RBC, WBC, ESR, Hb and PCV were studied by routine methods described by Jain (1986).

3.3 BIOCHEMICAL STUDIES

The blood was collected at weekly intervals and at the time of sacrifice in sterile test tubes and allowed to clot in a slanting position. The serum was separated and was used for the following biochemical studies using Diagnostic kits*.

1. Total serum proteins - BCA method (Reinhold 1953).
2. Serum albumins - BCA method (Reinhold 1953).
3. Serum cholesterol - Wybenga and Pileggi one step method (Wybenga *et al* 1970)
4. Asparate amino transferase - Reitman and Frankel (1957)
5. Serum Alkaline phosphatase - Kind & King (1954).

3.4 HISTOPATHOLOGICAL STUDIES

Detailed post mortem examination was carried out on goats died during the experiment and those sacrificed along with the control group at the end of the experiment. Representative portions of tissues viz., liver, kidney, lungs, spleen, abomasum, intestine, lymph nodes, brain and spinal cord were

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* *Diagnostic kits: Manufactured by Span Diagnostics Private Limited, Surat, India*

collected in 10% formol saline and processed conventionally and stained by Haematoxyline and Eosin (H&E) for histopathology. Cerebrum was divided into two halfs transversely, from each half a representative piece was collected for histopathology. Cerebellar part was collected from the middle of the cerebellum. Spinal cord was divided into cervical, thoracic and lumbar portions and tissue from each portion was collected for histopathology. Representative portions were collected from brain and spinal cord and fixed in acetone, formalin - ammonium bromide for frozen sectioning (Humason 1972). As the majority of animals showed nervous symptoms special attention was given to the nervous system. In the nervous system no single stain is capable of revealing the necessary histologic details. The approach to the use of special stains in neuropathology is relatively simple and it involves integrating the image obtained from various stainings of a single region (Robbins and Cortan 1979). Hence, special staining methods like oil red 'O' and Sudan black B for fat, Thionin and Toludine blue for Nissl, Cajal's goldchloride and Hortega's silver carbonate for astrocytes and phosphotungstic acid-haematoxylin and Luxol fast blue method for myelin were used (Disbrey and Rack 1970, Humason 1972 and Drury and Wallington 1980).

3.5 STATISTICAL ANALYSIS

Statistical analysis of the data was made as per the methods described by Snedecor and Cochran (1967).

CHAPTER 4

RESULTS

CHAPTER IV
4. RESULTS

Clinical manifestations, haematological and biochemical changes and pathological lesions noticed in goats on experimental feeding of *I. carnea* have been detailed.

4.1 SYMPTOMS

At first goats were disinclined to eat the leaves, but this reluctance was soon overcome and they got accustomed to eating of *I.carnea* leaves within three days. The goats began to lose condition and there was progressive weakness with loss of body weight.

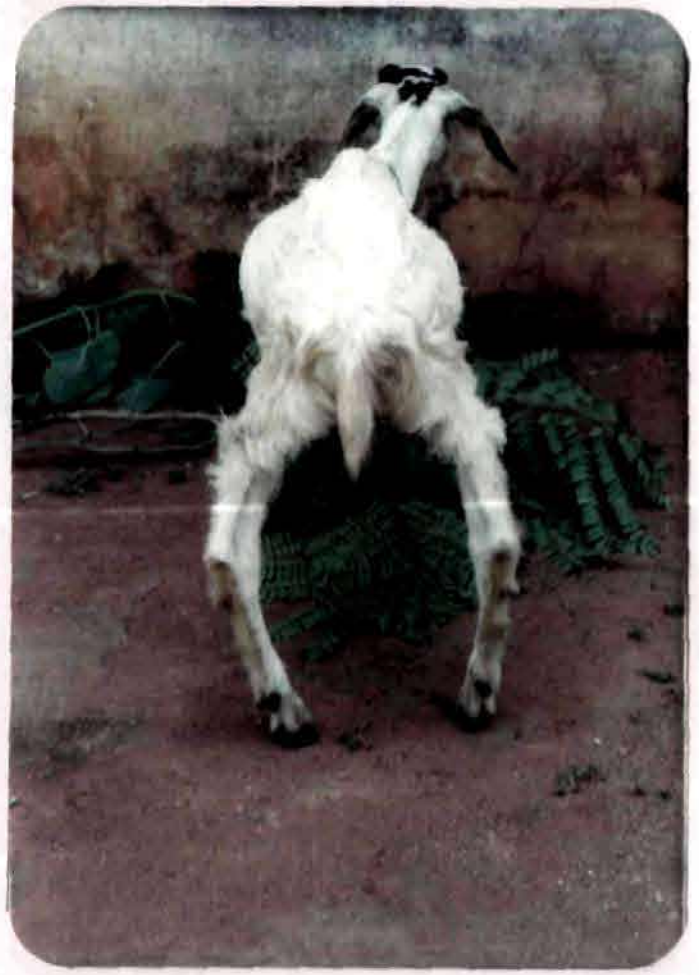
4.1.1. Group I

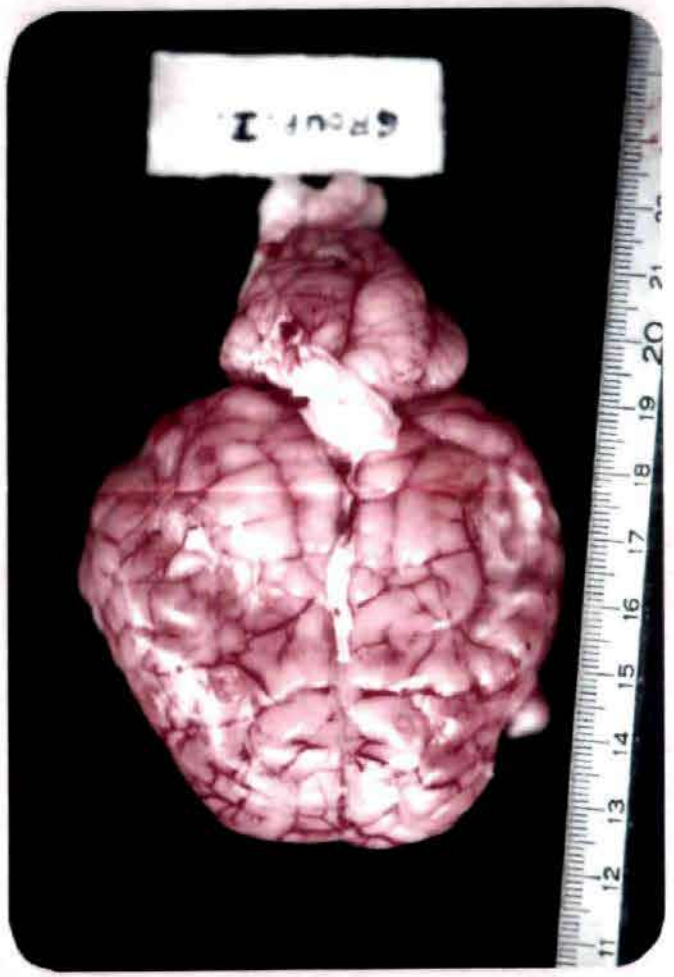
Initially there was a rise in body temperature reaching 103 to 105°F after 10 days of feeding and by 15th day the temperature returned to normal. Feed intake and water consumption were severely affected and the goats became anorexic. Loss of appetite and dullness were observed in all the animals in a fortnight. Marked decrease in body weight was observed. The animals exhibited nervous symptoms after 18-20 days of feeding *I.carnea*. There was a defect in the movement and animals exhibited aimless walk. The animals were in a state of lassitude and somnolence with little spontaneous activity. Some animals remained standing for a long period in an awkward posture (Fig 2). Animals exhibited knuckling movement due to paralysis of hind libms. Later they developed stiffness and paralysis of forelegs.

Darrhoea was observed in four goats after 23 days of feding (Fig 3). Respiratory distress was developed in three goats after 42 days. Subsequently, paralytic symptoms culminated in recumbency and death (Fig 4). Four goats died

Figure 2. *Ipomoea carnea* poisoning - Goat showing awkward posture - Group I.

Figure 3. *Ipomoea carnea* poisoning - Goat showing diarrhoea indicated by soiling of the tail, perineum and hind limbs - Group I.




GROUP I

between 15-50 days after the commencement of the experiment. The remaining two died between 65-70 days of the experiment.

4.1.2. Group II

Anorexia, dullness, depression and rise in body temperature were observed in all the goats after 45 days. There was marked decrease in body weight and finally the animals were emaciated. Rough hair coat was observed in all the goats. In three goats there was a loss of hair in patches over the neck and head regions. Progressive and gradual development of weakness and nervous symptoms were observed in all the goats. Incoordinated gait was a conspicuous clinical manifestation in all the animals. The loss of control over the extension and flexion movements of the hind limbs was observed after 54-60 days. Swinging and shaking of head was also observed. Affected animals became worse after 90-110 days and fell down with extended hind limbs when allowed to move out of the pen. Some animals had severe muscular spasms and became recumbent and could not stand without support. They lost the awareness of the surroundings. Three goats died within 92 to 103 days. The remaining animals were sacrificed at the end of the experiment.

4.2 BODY WEIGHTS

The mean body weights of the three groups are shown in the Table 1. The body weights of experimental animals (group I and II) were significantly decreased ($P < 0.01$, Fig 5). In group I, the body weight decreased at a rate of 336 g/week, whereas in group II the body weight decreased at a rate of 243 g/week(Table 3). The body weights of control animals increased significantly ($P < 0.01$) during the period of study.

4.3 HAEMATOLOGICAL CHANGES

The haematological values of experimental and control animals are presented in Tables 1 and 3.

4.3.1 Red Blood Cell Count

The mean values of RBC (Millions/cu.mm) in the three groups of animals are shown in the Table 1. The RBC values of the animals in group I and II decreased significantly ($P<0.01$, Fig 6). The RBC values in group I decreased at the rate of 0.337 millions/cu.mm per week (Table 3). During the experimental study the RBC values of control animals did not change significantly.

4.3.2 White Blood Cell Count

The mean values of WBC (thousands/cu.mm) are represented in the Table 1. The WBC values of the experimental animals (group I and II) decreased significantly ($P<0.01$, Fig 7). In group I the WBC values decreased at the rate of 0.639 thousands/cu.mm per week, whereas in group II the decrease was at the rate of 0.37 thousands/cu.mm per week (Table 3). The WBC values in control animals were significantly changed during the period of study. There was no significant change in the differential count in all the groups.

4.3.3. Haemoglobin

The mean values for Hb (g/dl) are represented in the Table 1. In experimental groups (group I and II) the Hb values were decreased ($P<0.01$, Fig 8). In group I the haemoglobin values decreased at the rate of 0.534 g/dl per week, whereas in group II the decrease was at the rate of 0.258 g/dl per week (Table 3). The haemoglobin values in control group were not changed during the period of study.

4.3.4. Mean Corpuscular Haemoglobin

The mean values of MCH (μ μg) for the groups are shown in the Table 1. In group I and II the MCH values were significantly decreased ($P < 0.01$). In group I animals the MCH values decreased at the rate of 0.227 μ μg/week, whereas in group II animals the decrease was at the rate of 0.098 μ μg/week (Table 3). The MCH values in control group were not significantly changed during the period of study.

4.3.5. Packed Cell Volume

The mean values of PCV are represented in the Table 1. In experimental groups (group I and II) the PCV values were significantly decreased ($P < 0.01$, Fig 9). In group I the PCV values decreased at the rate of 1.629% per week whereas in group II the decrease was at the rate of 0.731% per week (Table 3). The PCV values in control group were not significantly changed during the period of study.

4.3.6. Mean Corpuscular Volume

The mean values of MCV (μ^3) for the groups are shown in the Table 1. In group I and II the MCV values decreased significantly ($P < 0.01$). In group I animals the MCV values decreased at the rate of 0.256 μ^3/week, whereas in group II animals the decrease was at the rate of 0.640 μ^3/week (Table 3). The MCV values in control group were not significantly changed during the period of study.

4.3.7 Erythrocytic Sedementation Rate

The mean values of ESR (mm/hour) for the groups are shown in the Table 1. The ESR values of the animals in group I and II decreased significantly ($P < 0.01$). In group I the ESR values decreased at the rate of 0.224

Table 1. ***Ipomoea carnea*** **toxicity: Haematological parameters of goats in experimental and control groups (Mean ± S.E.)**

S.No.	Parameter	Groups I (37)	II (67)	Control (102)
1.	Body weight (kg)	7.691 ± 0.168	7.550 ± 0.139	9.170 ± 0.053
2.	RBC count (millions/cu.mm)	12.565 ± 0.169	12.910 ± 0.109	14.021 ± 0.016
3.	WBC count (thousands/cu.mm)	7.591 ± 0.314	7.173 ± 0.214	9.286 ± 0.032
4.	Hb (g/dl)	9.770 ± 0.264	9.624 ± 0.153	11.376 ± 0.017
5.	MCH ($\mu\mu$g)	7.727 ± 0.122	7.429 ± 0.097	8.114 ± 0.015
6.	PCV (%)	29.465 ± 0.869	28.970 ± 0.512	32.653 ± 0.273
7.	MCV (μ^3)	23.329 ± 0.503	22.372 ± 0.278	23.300 ± 0.195
8.	ESR (mm/hour)	0.513 ± 0.138	0.701 ± 0.070	0.176 ± 0.038

Figures in parenthesis indicate the number of observations in that group

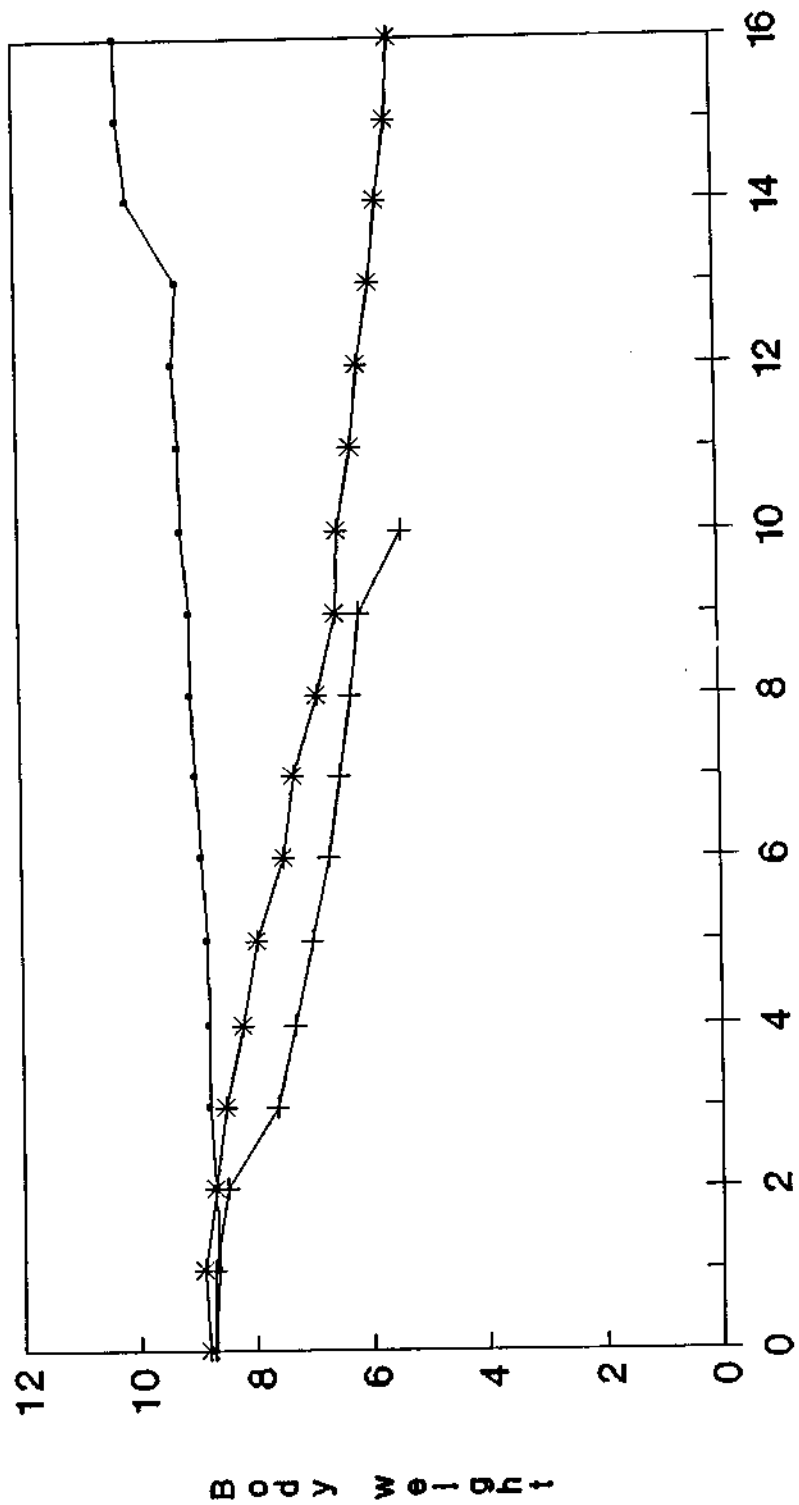


Fig.5. Ipomoea carnea toxicity: Graph showing mean body weights (kg) recorded at weekly intervals in control and experimental groups

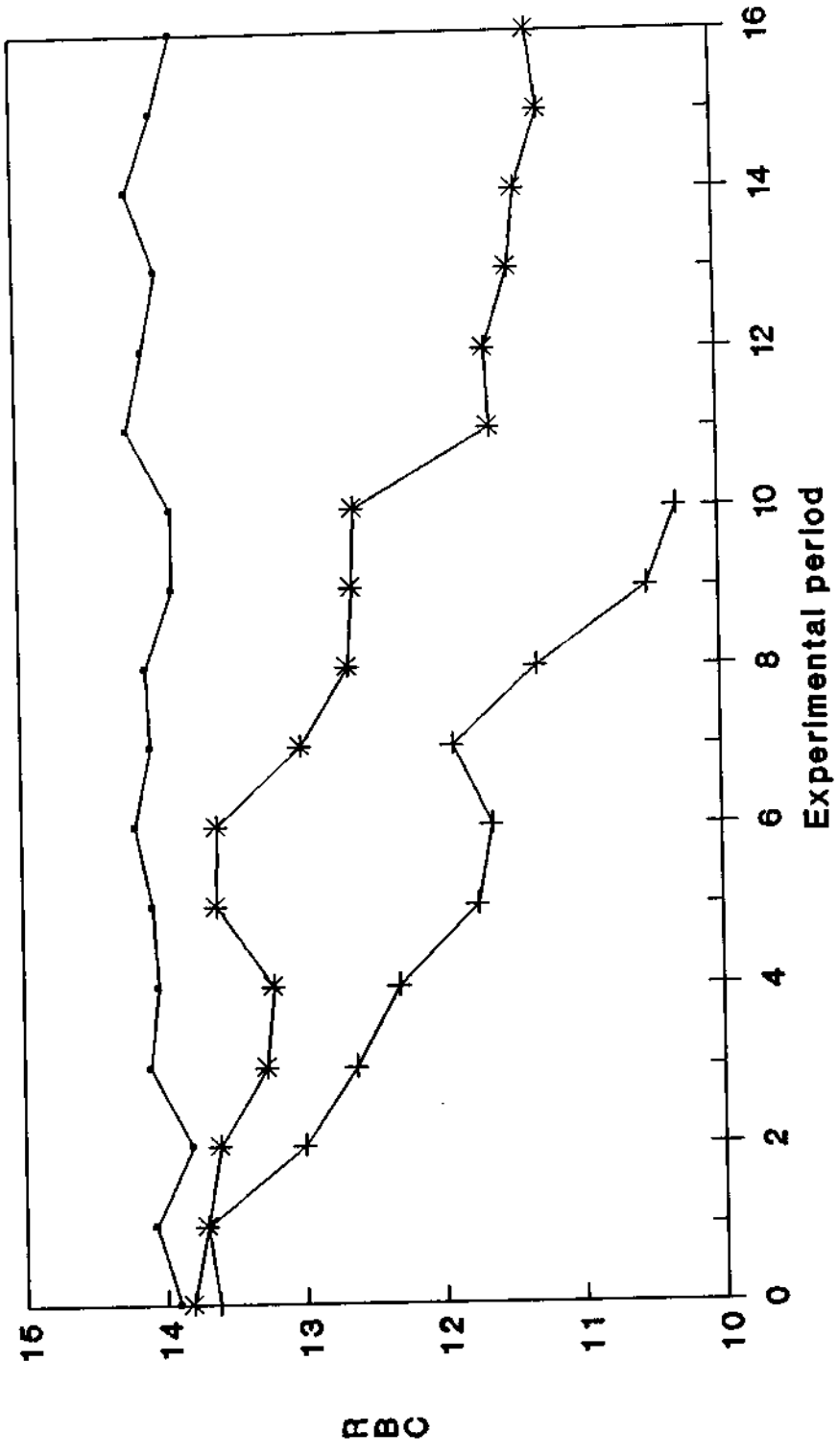


Fig.6. *Ipomoea carnea* toxicity: Graph showing mean RBC (millions/cu.mm) values recorded at weekly intervals in control and experimental groups

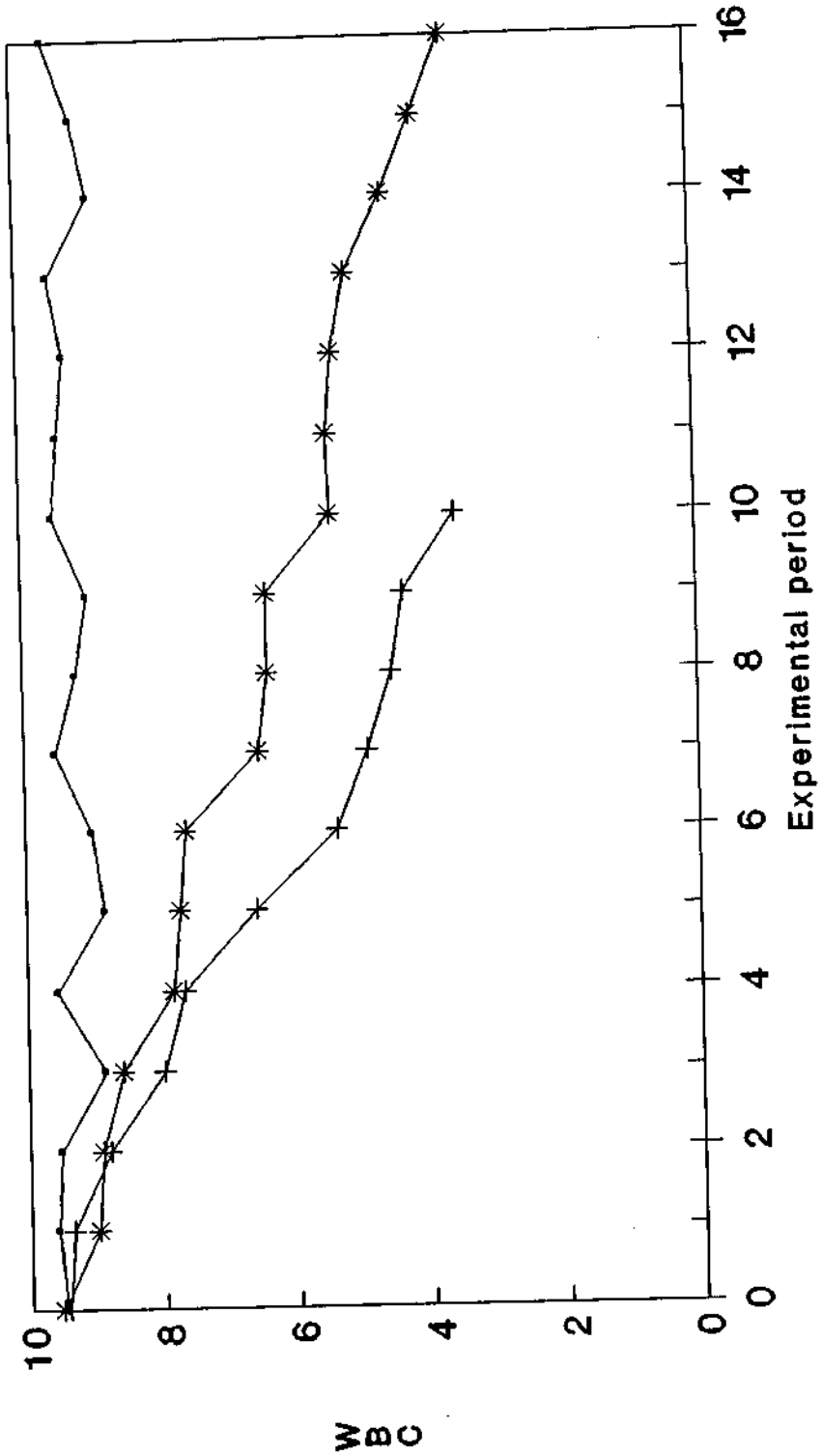


Fig.7. *Ipomoea carnea* toxicity: Graph showing mean WBC value (thousands/cu.mm) recorded at weekly intervals in control and experimental groups

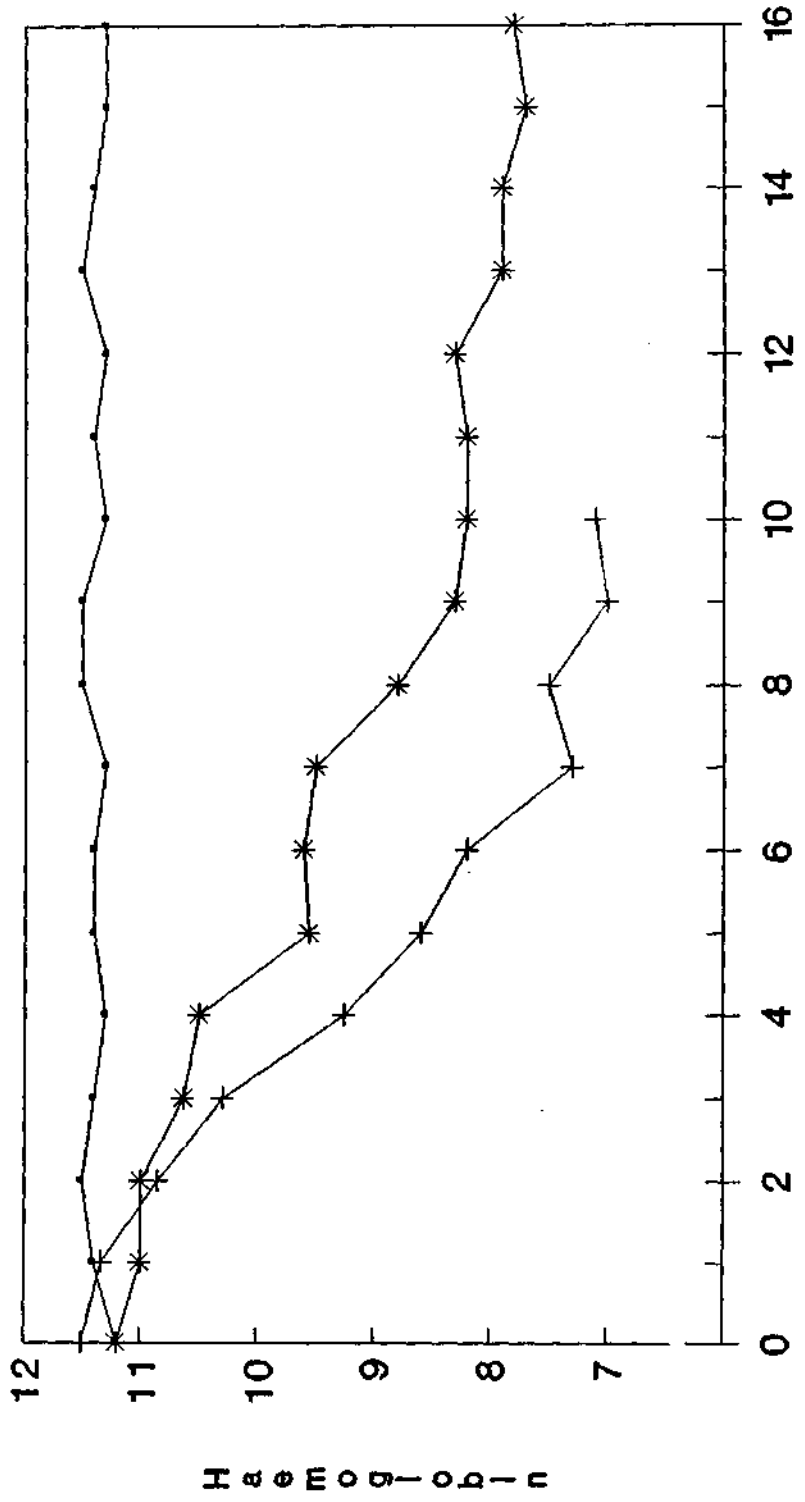


Fig.8. *Ipomoea carnea* toxicity: Graph showing mean haemoglobin values (g/dl) recorded at weekly intervals in control and experimental groups

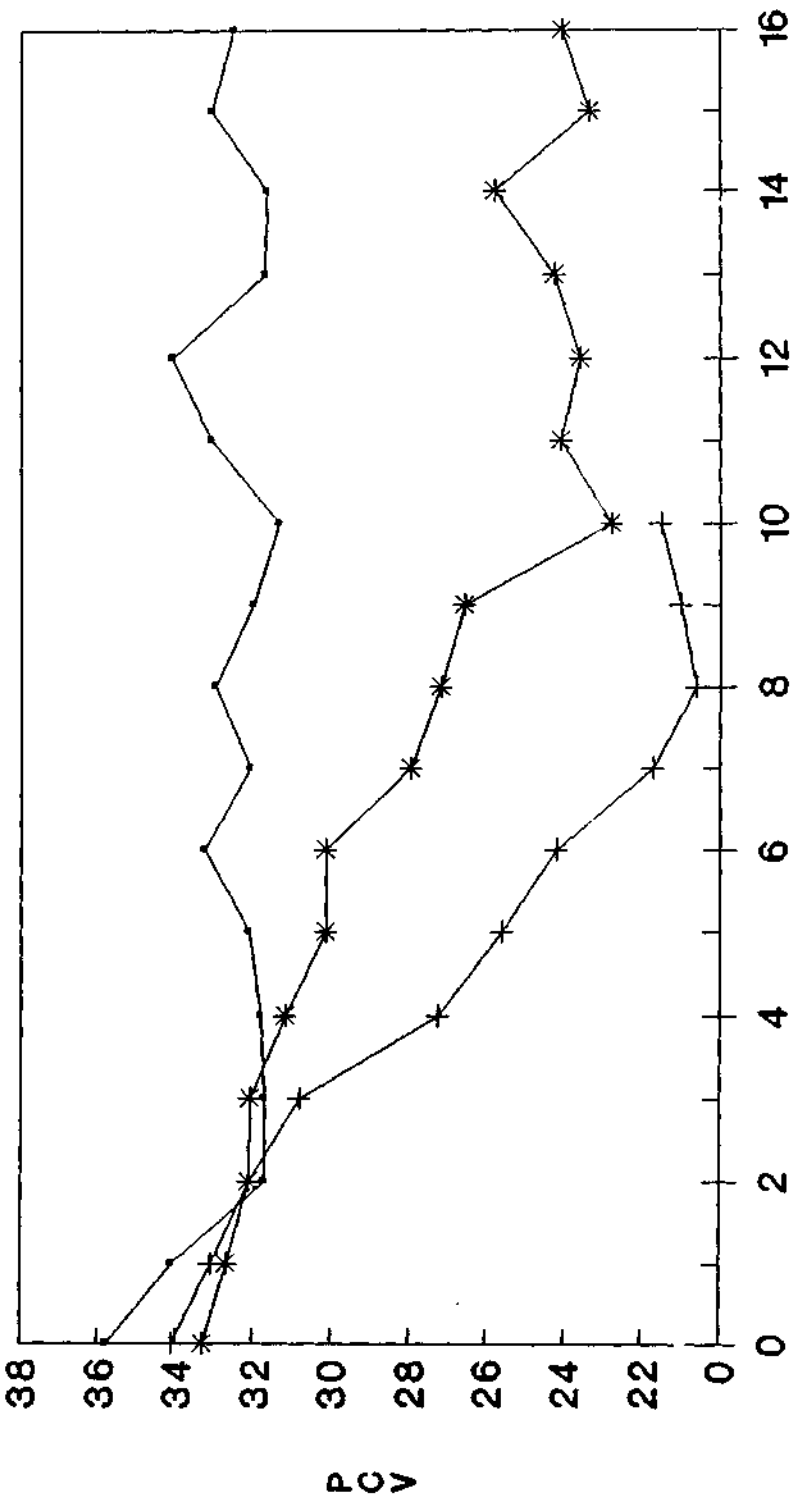


Fig.9. *Ipomoea carnea* toxicity: Graph showing mean PCV(%) recorded at weekly intervals in control and experimental groups

mm/hour per week, whereas the decrease in group II was at the rate of 0.69 mm/hour per week. The ESR values in control group not significantly changed during the experimental study.

4.4. BIOCHEMICAL CHANGES

4.4.1. Total Proteins

The mean values of total proteins (g/dl) for the three groups are shown in the Table 2. Total proteins of the group I and II decreased significantly ($P < 0.01$). In group I the total proteins decreased at the rate of 0.220 g/dl per week, whereas in group II the decrease was at the rate of 0.102 g/dl per week (Table 4). The values for total proteins in control animals increased significantly ($P < 0.01$, Fig 10) during the period of study.

4.4.2. Albumins

The mean values of serum albumins (g/dl) for the three groups are shown in the Table 2. In group I and II the albumin values decreased significantly ($P < 0.01$, Fig 11). In group I the albumin values decreased at a rate of 0.122 g/dl/week, whereas in group II the decrease was at a rate of 0.076 g/dl/week during the period of study (Table 4). The albumin values of the control animals increased significantly ($P < 0.01$) during the period of study.

4.4.3. Cholesterol

The mean values of cholesterol (mg/dl) for the groups are shown in the Table 2. The serum cholesterol levels in group I, II and control animals were not significantly changed during the experimental period.

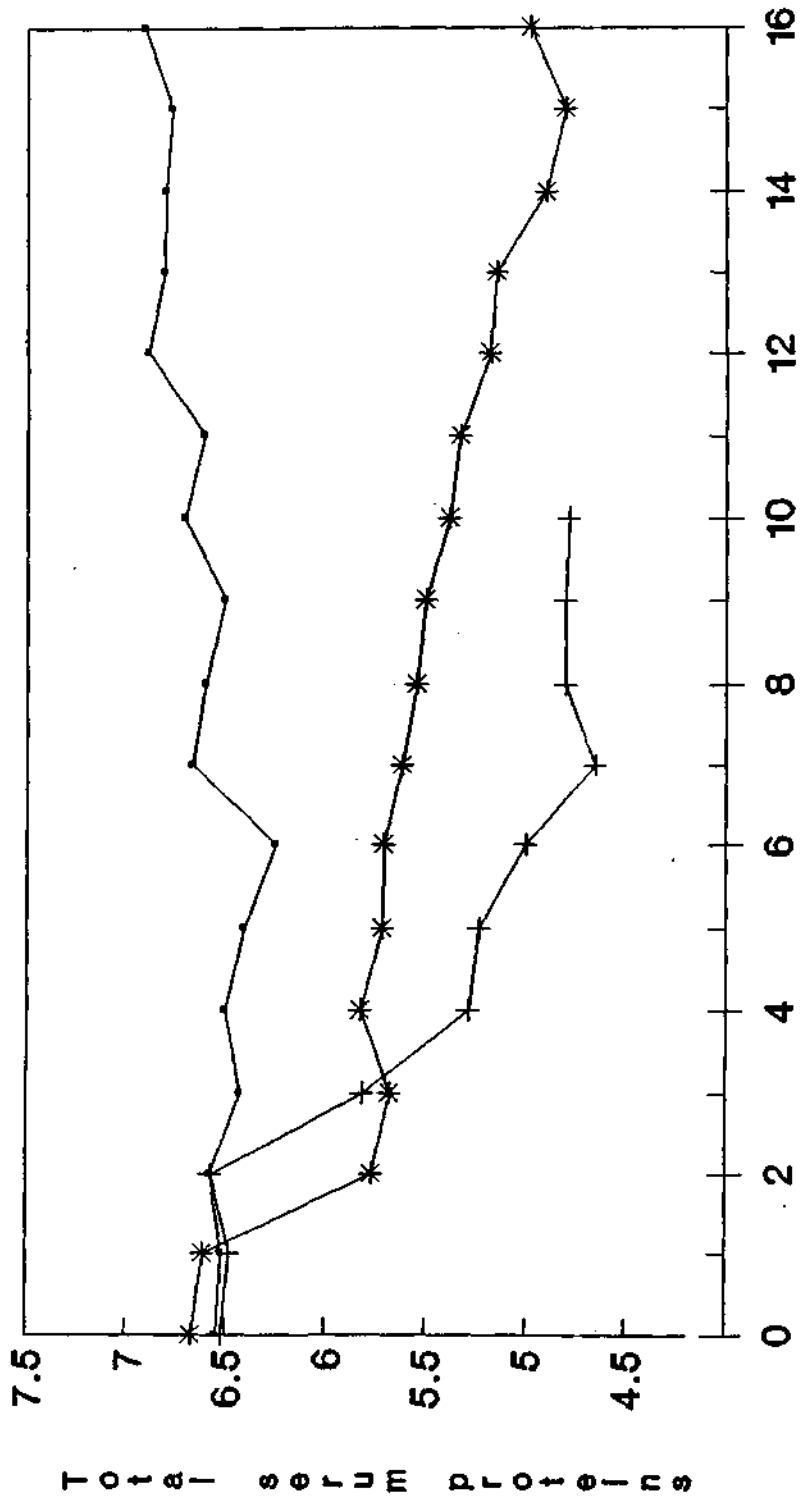


Fig.10. *Ipomoea carnea* toxicity: Graph showing mean total serum protein values (g/dl) recorded at weekly intervals in control and experimental groups

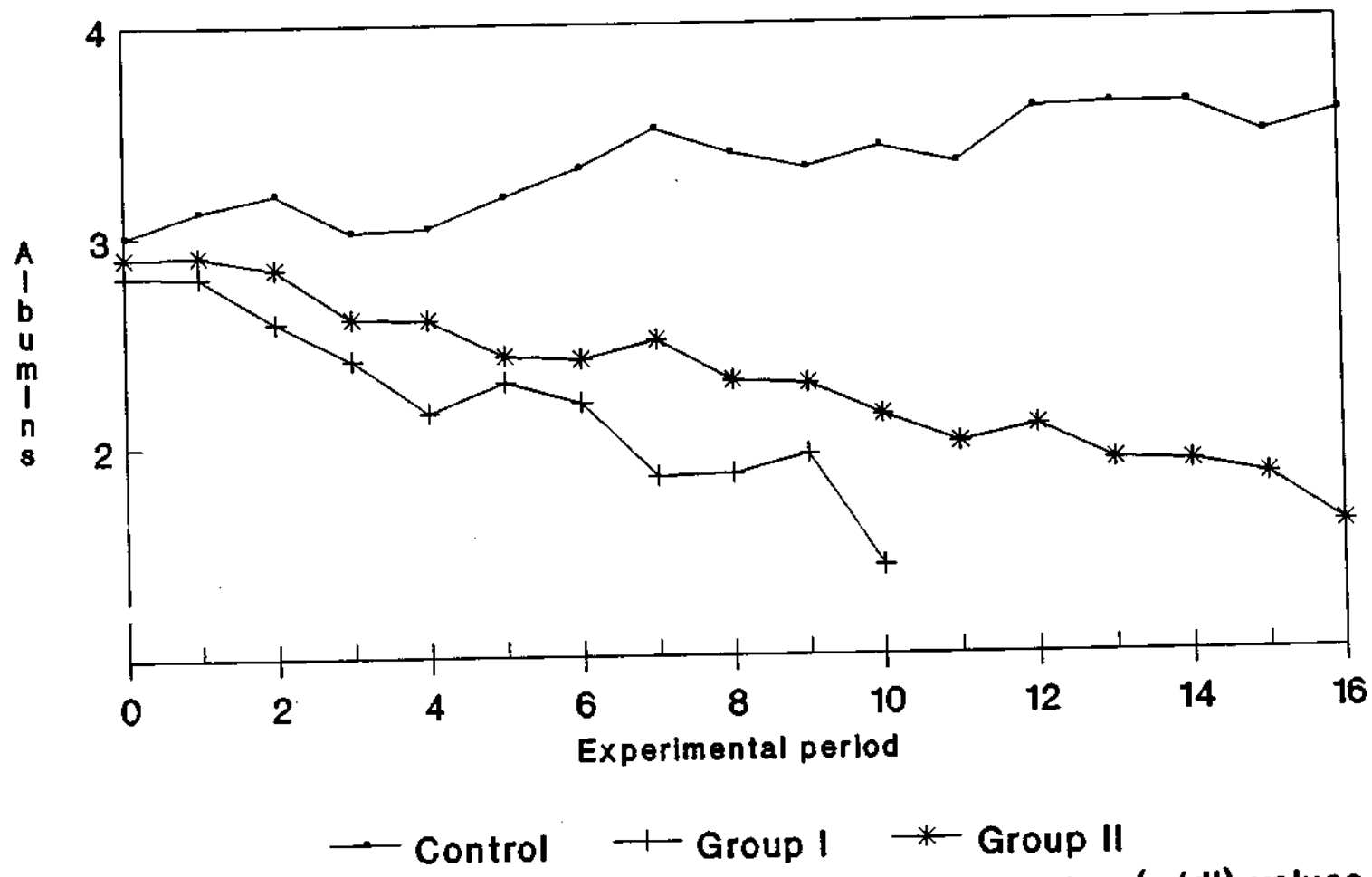


Fig.11. *Ipomoea carnea* toxicity: Graph showing mean albumins (g/dl) values recorded at weekly intervals in control and experimental groups

Table 2. Ipomoea carnea toxicity: Biochemical parameters of goats in experimental and control groups (Mean ± S.E.)

S.No.	Parameter	Groups I (37)	II (67)	Control (102)
1.	Total proteins (g/dl)	5.710 ± 0.066	5.790 ± 0.125	6.630 ± 0.031
2.	Albumins (g/dl)	2.450 ± 0.051	2.405 ± 0.072	3.310 ± 0.033
3.	Cholesterol (mg/dl)	78.209 ± 0.198	77.454 ± 0.275	77.583 ± 0.186
4.	Alkaline phosphatase (B.units/L)	22.472 ± 1.596	20.081 ± 0.331	18.616 ± 0.148
5.	Aspartate amino transferase (IU/L)	83.034 ± 3.200	72.506 ± 3.088	58.660 ± 0.234

Figures in parenthesis indicate the number of observations in that group

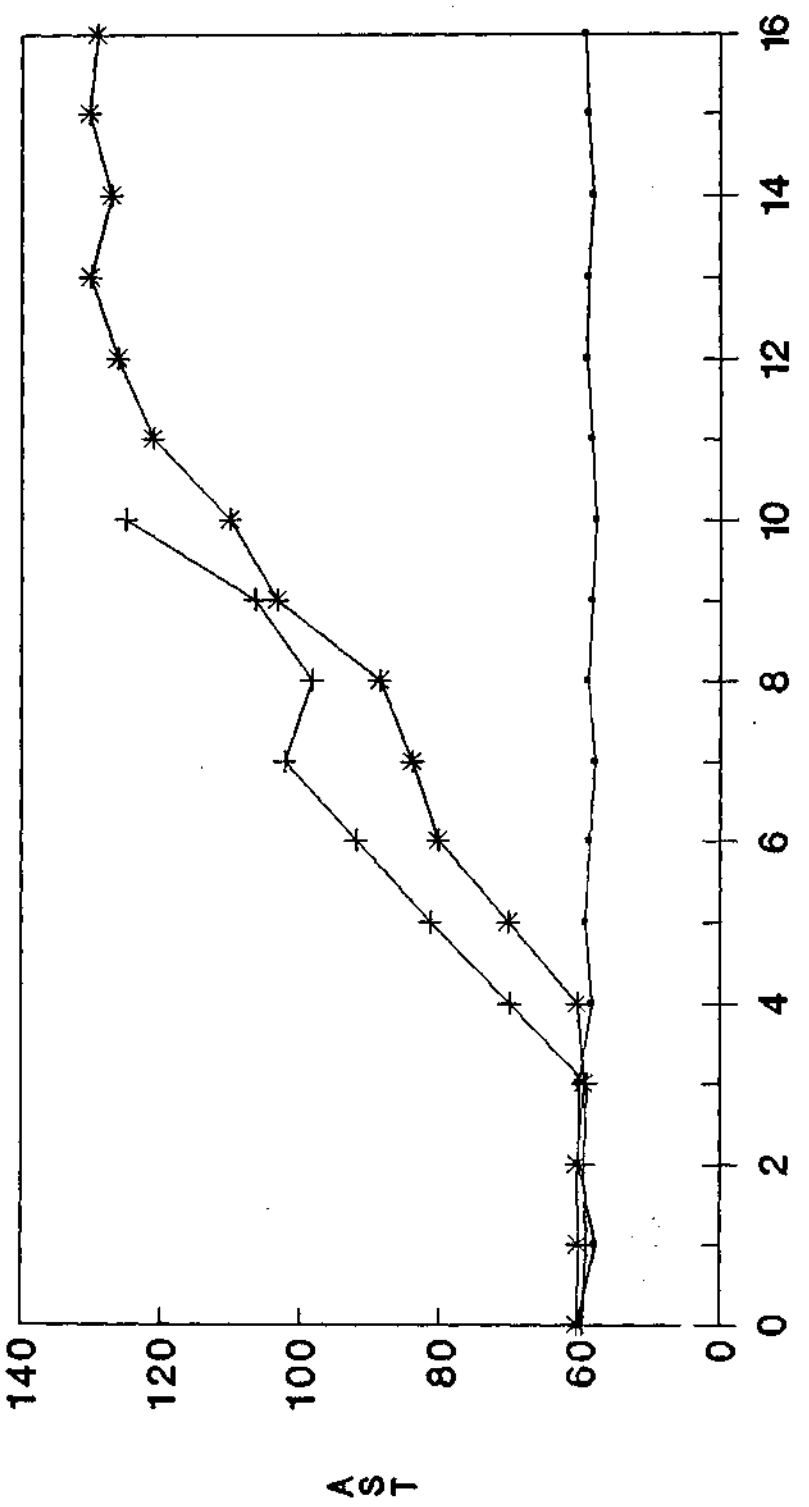


Fig.12. *Ipomoea carnea* toxicity: Graph showing mean AST (IU/L) values recorded at weekly intervals in control and experimental groups

Table 3. Correlation and regression between periods and haematological parameters among control and experimental groups

Groups	Body weight (kg)		RBC count (millions/ cu.mm)		WBC count (thousands/ cu.mm)		Hb (g/dl)		MCH ($\mu\mu$g)		PCV (%)		MCV (μ^3)		ESR (mm/hr)	
	r	b	r	b	r	b	r	b	r	b	r	b	r	b	r	b
I	-0.967	-0.336*	-0.962	-0.337*	-0.984	-0.639*	-0.978	-0.534*	-0.901	-0.227*	-0.906	-1.629*	-0.614	-0.640*	0.787	0.224*
II	-0.984	-0.243*	-0.934	-0.180*	-0.979	-0.370*	-0.957	0.258*	-0.766	-0.098*	-0.808	-0.731*	-0.519	-0.256*	0.556	0.069*
Control	0.842	0.091*	0.065	--	-0.134	--	0	0	-0.037	--	-0.109	--	-0.127	--	0.062	--

* P < 0.01

Table 4. Correlation and regression between periods and biochemical parameters among experimental and control groups

Groups	Total proteins (g/dl)		Albumins (g/dl)		Serum cholesterol (mg/dl)		Alkaline phosphatase (B.units/L)		Asparate amino transferase (IU/L)	
	r	b	r	b	r	b	r	b	r	b
I	-0.849	-0.220*	-0.816	-0.122*	0.036	-	0.231	-	0.927	5.925*
II	-0.874	-0.102*	-0.839	-0.076*	0.076	-	0.872	0.796*	0.955	5.404*
Control	0.404	0.026*	0.566	0.039*	0.112	-	-0.067	-	-0.136	-

* $P < 0.01$

4.4.4. Serum Alkaline Phosphatase

The mean values of SAP (B.units/L) for the groups are shown in the Table 2. In group II the SAP values significantly decreased ($P < 0.01$). In group II animals the SAP values decreased at the rate of 0.796 B.units/L per week (Table 4). The SAP values of group I and control animals were not significantly changed during the period of study.

4.4.5. Asparate Amino Transferase

The mean values of AST (IU/L) for the three groups are shown in the Table 2. In group I and II the AST values decreased significantly ($P < 0.01$, Fig 12). In group I animals the AST values decreased at the rate of 5.925 IU/L per week, whereas in group II animals the decrease was at the rate of 5.404 (IU/L) per week (Table 4). The AST values of control animals were not significant during the period of study.

4.5 PATHOLOGICAL CHANGES IN GROUP I

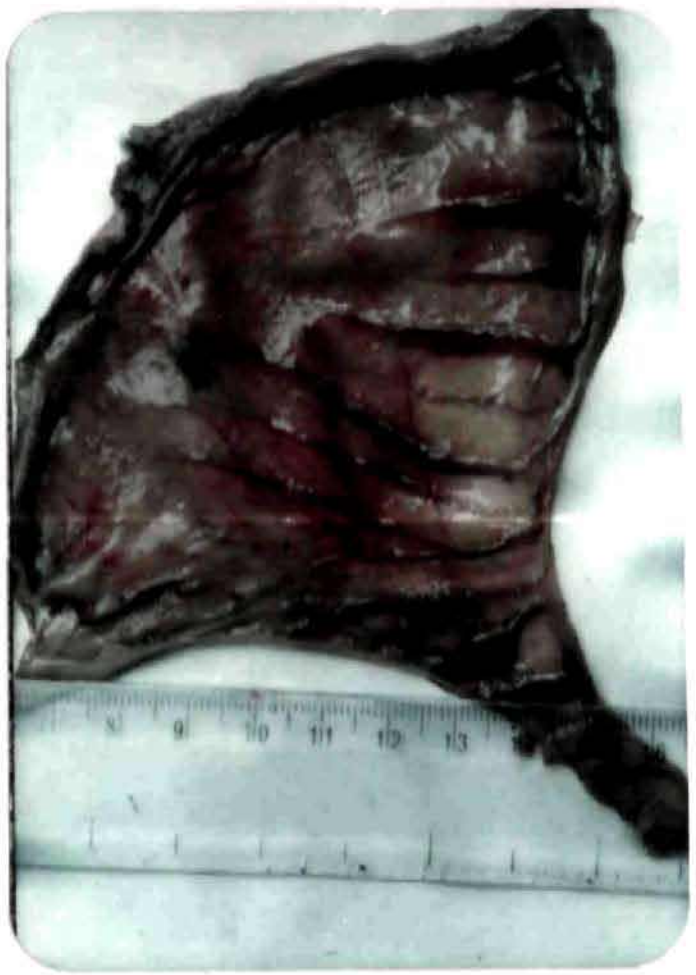
4.5.1. Gross Lesions (Group I)

A clear straw coloured fluid could be observed in the abdominal cavity of all the animals at necropsy. Hydropericardium and hydrothorax were consistently noticed. Grossly there was slight enlargement of the liver with rounded borders. On section the surface was mottled and blood oozed out. Gall bladder was distended with yellowish green thick bile. Heart blood vessels were engorged. Diaphragmatic lobes of lung were moderately congested and oedematous. Moderate amount of sanguineous froth was present in the bronchial tree. The interlobular septa was prominent.

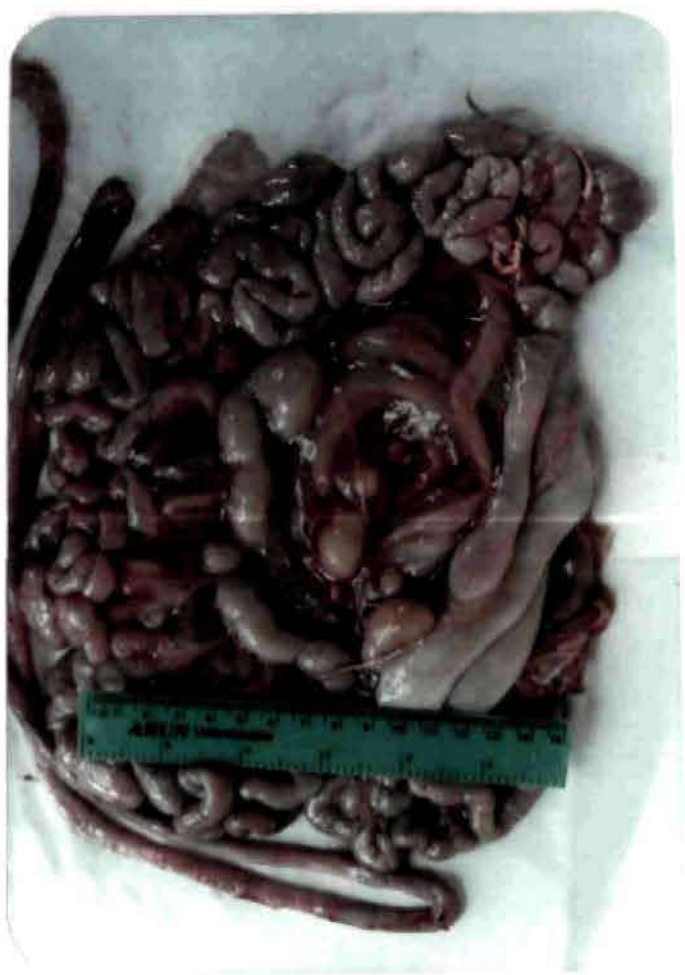
Kidneys were dark red in colour and swollen. On section the medullary region was filled with gelatinous fluid and was severely congested. The

Figure 14. Abomasum (Group I) - Mild congestion of the mucosa in fundic and pyloric regions and congestion and oedema of abomasal folds.

Figure 15. Mesenteric lymph nodes (Group I) - swollen and oedematous.



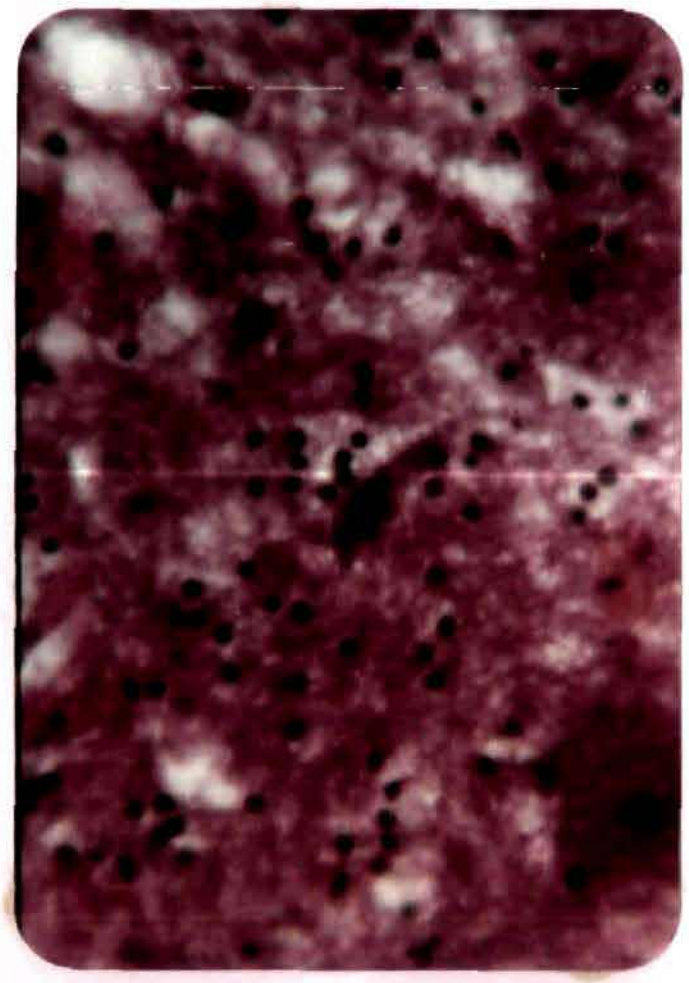
14.

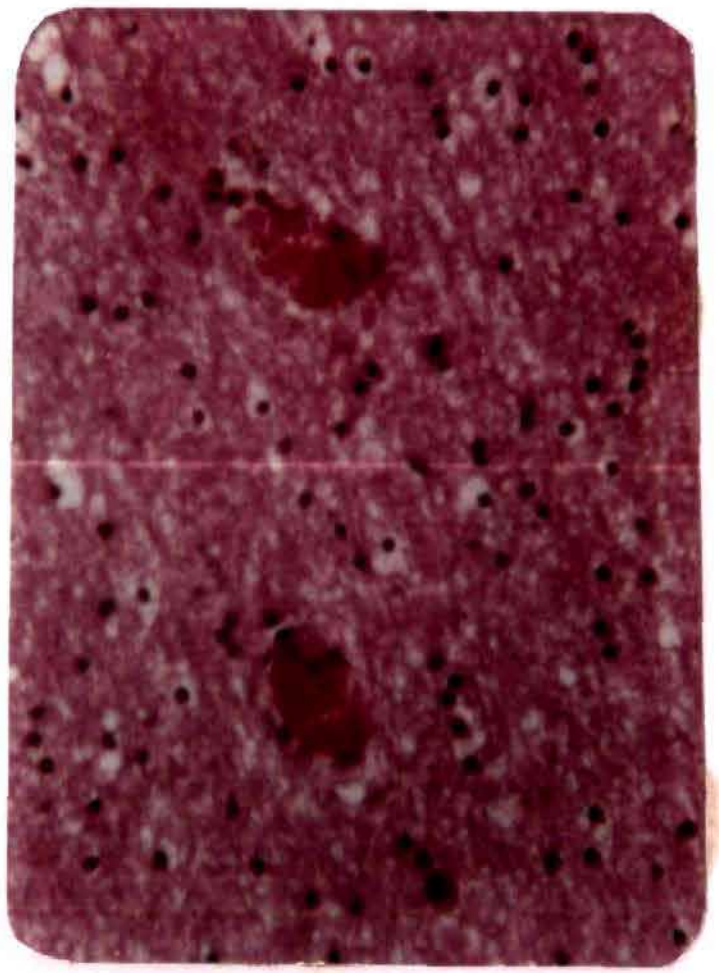


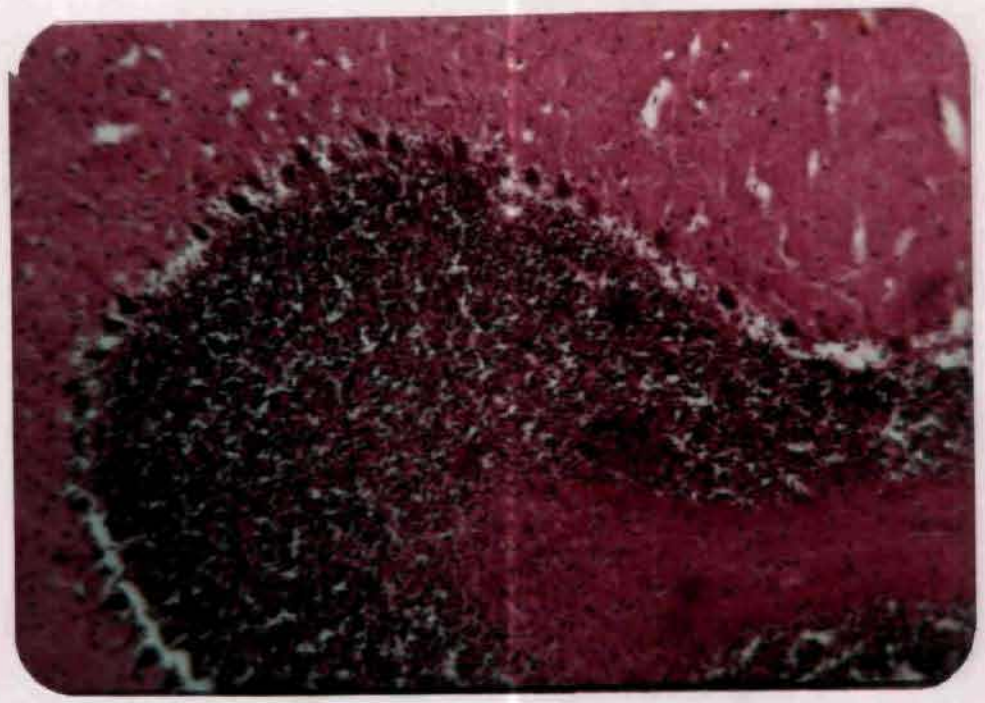
15.

Figure 18. Cerebellum (Control) - The cells of Purkinje layer showing normal staining. H & E x80.

Figure 19. Cerebellum (Group I) - Purkinje cell layer exhibiting pale staining compared with the Fig 18 above. H & E x320.







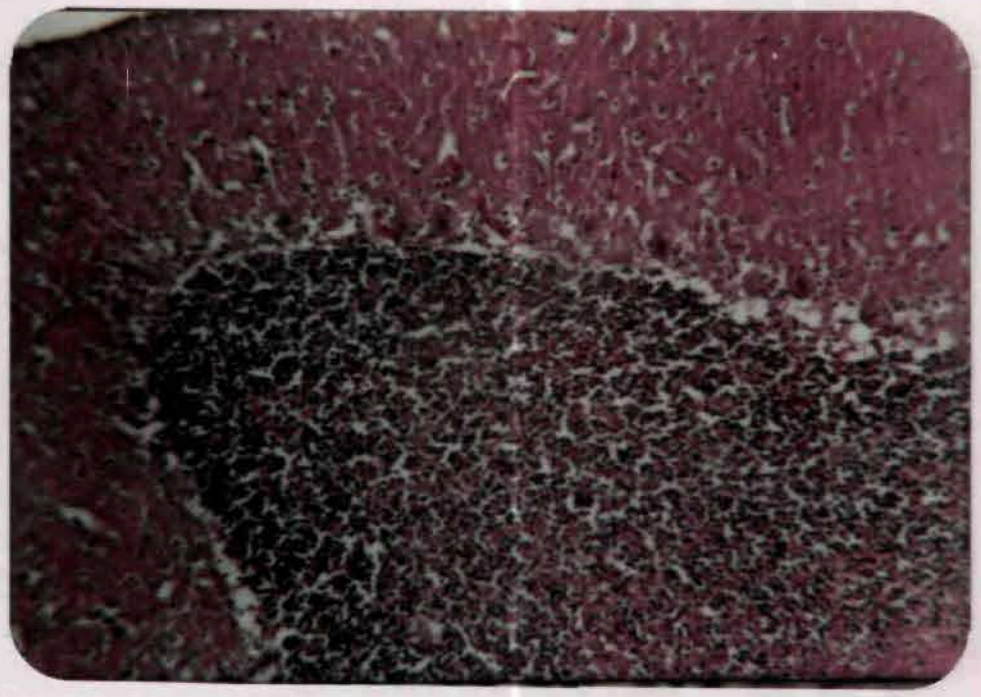
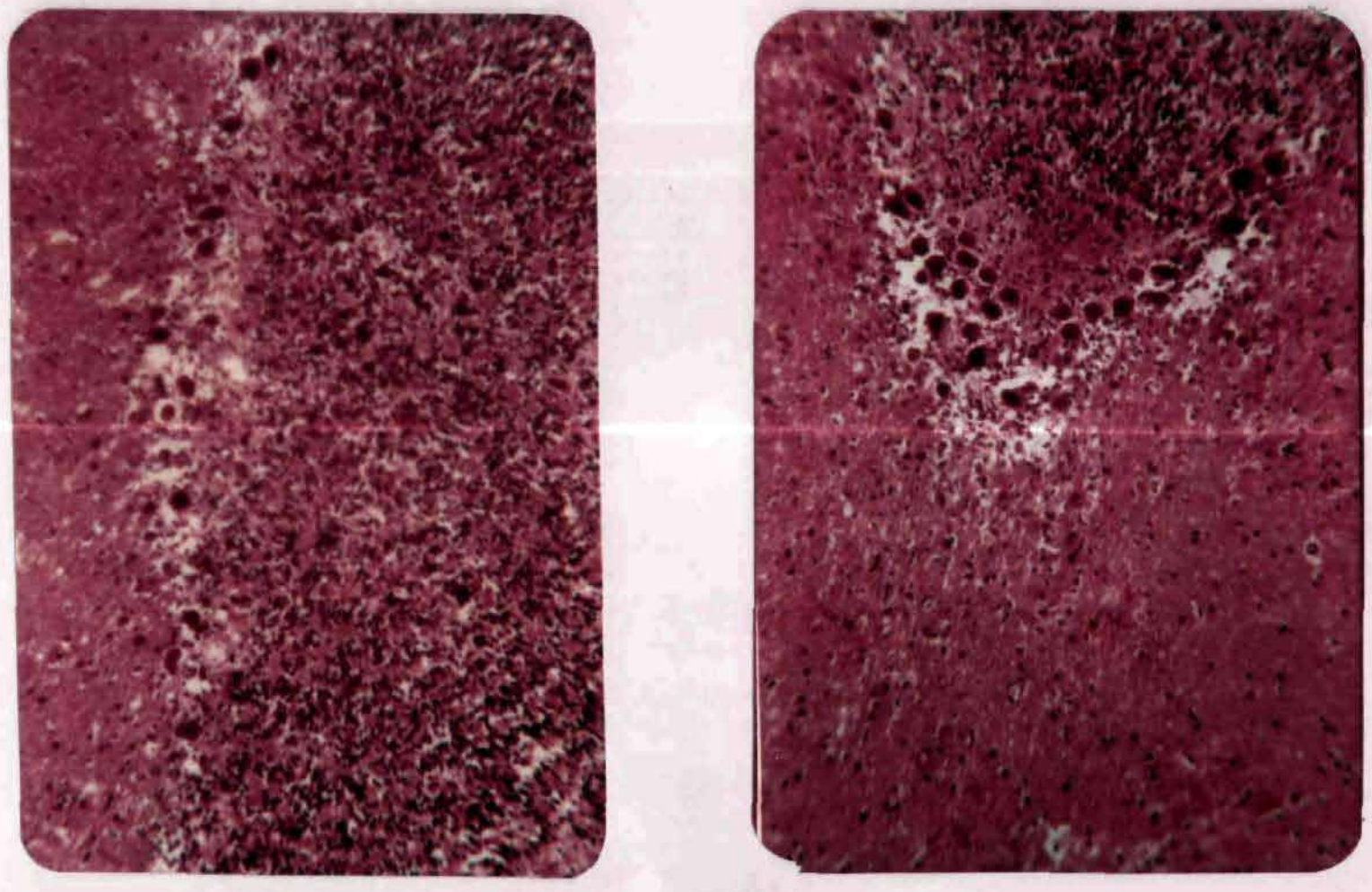


Figure 20. Cerebellum (Group I) - Aggregation of Purkinje cells with oedema between Purkinje cell layer and molecular layer. H & E x240.

Figure 21. Cerebellum (Group I) - Purkinje cells giving the impression of dipping into the granular layer. H & E x100.



cortico-medullary junction was dark red with petechiae. The capsule could be peeled off easily. Mild congestion of the mucosa of abomasum (Fig 14) and small intestine was observed. Mesenteric lymph nodes were swollen and oedematous (Fig 15). There was mild enlargement of spleen with rounded edges. The pulp was deep reddish and soft. The meningeal blood vessels of the brain and spinal cord were engorged (Fig 13). The sulci were narrowed and the gyri were flattened. The ventricles were compressed. On section the white matter appeared soft and gelatinous.

4.5.2. Microscopic Lesions (Group I)

4.5.2.1 Cerebrum. Congestion and haemorrhages were consistently observed both in the white and grey matter (Fig 16). Polymorphism of the neurons was observed and the affected cells were confined to the middle and deeper laminae of the cerebral cortex. Increase in the perineuronal space was observed in some neurons. Some nerve cells were enlarged with faintly stained cytoplasm. The lack of details resulting in shadow state of the cells was conspicuous. Shrinkage of neurons with pyknosis of nuclei was observed in a few cases. In some areas focal loss of neurons was observed. Vacuolization of cytoplasm at various degrees was observed in some of the nerve cells. The Nissl substance in most of the neurons in the perinuclear zone became fine, dispersed and disappeared resulting in a halo. This central chromatolysis was confirmed by Thionin staining. Satellitosis (Fig 17) and neuronophagia of some pyramidal cells were observed. The white matter was loosely textured with demyelination.

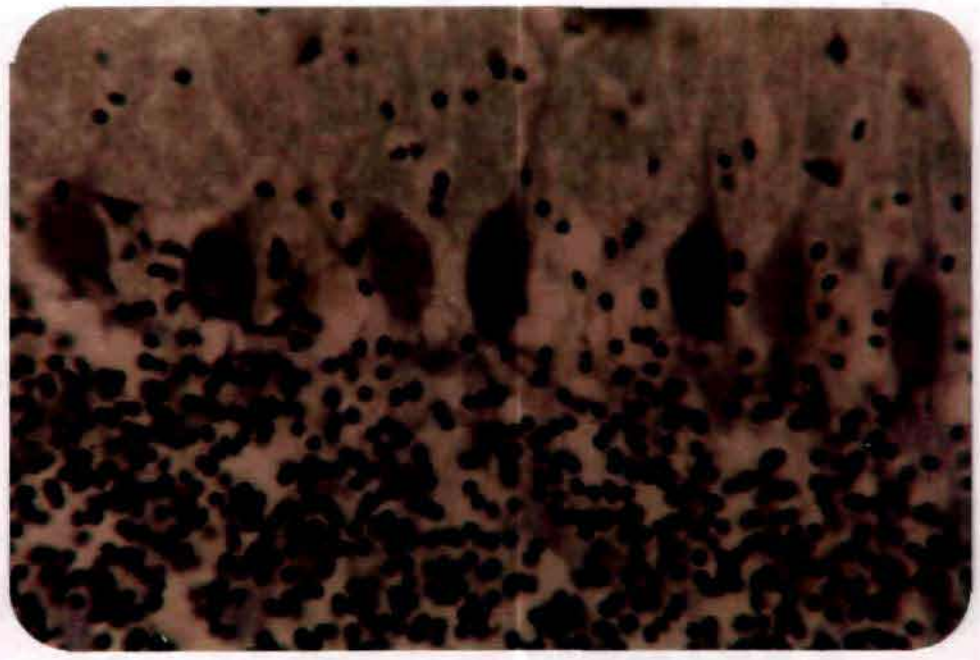
4.5.2.2 Cerebellum. The histologic changes were noticed chiefly in Purkinje cell layer. Some of the Purkinje cells had pale staining affinity (Fig 18 & 19) with accompanying astrocytic proliferation in molecular layer. The astrocytic proliferation was confirmed by Hortega's silver carbonate method. The Purkinje cells in some places were aggregated together with oedema between Purkinje cell layer and the molecular layer (Fig. 20). These cells appeared as though they got

displaced from the junction of molecular and granular layers. This gave the impression that these cells have dipped into the granular layer (Fig. 21). At some other foci the arrangement of the Purkinje cells gave the impression that they were in two layers. The cytoplasm of some Purkinje cells retained its normal staining properties but revealed small vacuoles mainly towards the granular layer. In goats that died after 25 days, the Purkinje cells were swollen and the processes were shortened with marginated nuclei. some Purkinje cells were stained pale and the differentiation between cytoplasm and nucleus was totally lost resulting in a ghost like appearance. The cellular outline was discernable in some, while in other it was indistinct. The cell body was amorphous and foam- like. In some Purkinje cells central chromatolysis was observed, which was confirmed by Thionin staining (Fig 22, 23 & 24). There was an accumulation of glial cells in the molecular layer of cerebellum in association with degenerating Purkinje cells. In some places loss of Purkinje cells was observed in goats that died after 56 days. The molecular layer of the cerebellum appeared thinner than normal and the cells of granular layer appeared to have entered into the molecular layer. Similarly, the cells in the granular layer also became less dense (Fig 25).

5.5.2.3 Spinal Cord. Mostly the changes were observed in the thoracic and lumbar portions of the spinal cord. Congestion and haemorrhages were consistent both in the white and grey matter. Neurons showed degenerative changes with margination of the nucleus and central chromatolysis (Fig 26). In some neurons the processes were shortened and they became swollen and rounded. In some others the cytoplasm was uniformly acidophilic and vacuolated with loss of cellular details giving a ghost (shadow) - like appearance. Moderate satellitosis and neuronophagia were observed (Fig 27). The distribution of the neurons in the affected part was less dense. There was increased cellularity due to proliferation of cells mostly astroglial cells which was confirmed by Hortega's silver carbonate method (Fig 28 & 29). Demyelination with swollen axons was observed in the white matter.

Figure 22. Cerebellum (Group I) - The Purkinje cells showing normal elongated processes. Thionin staining x320.

Figure 23. Cerebellum (Group I) - Swollen and rounded Purkinje cells showing shortened processes with chromatolysis. Thionin staining x320.



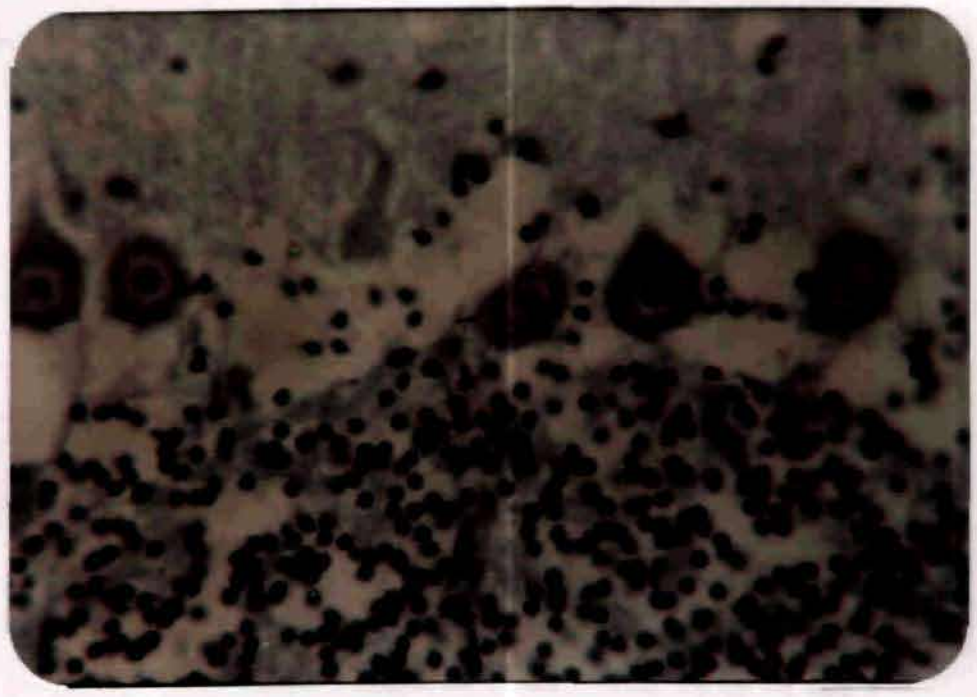
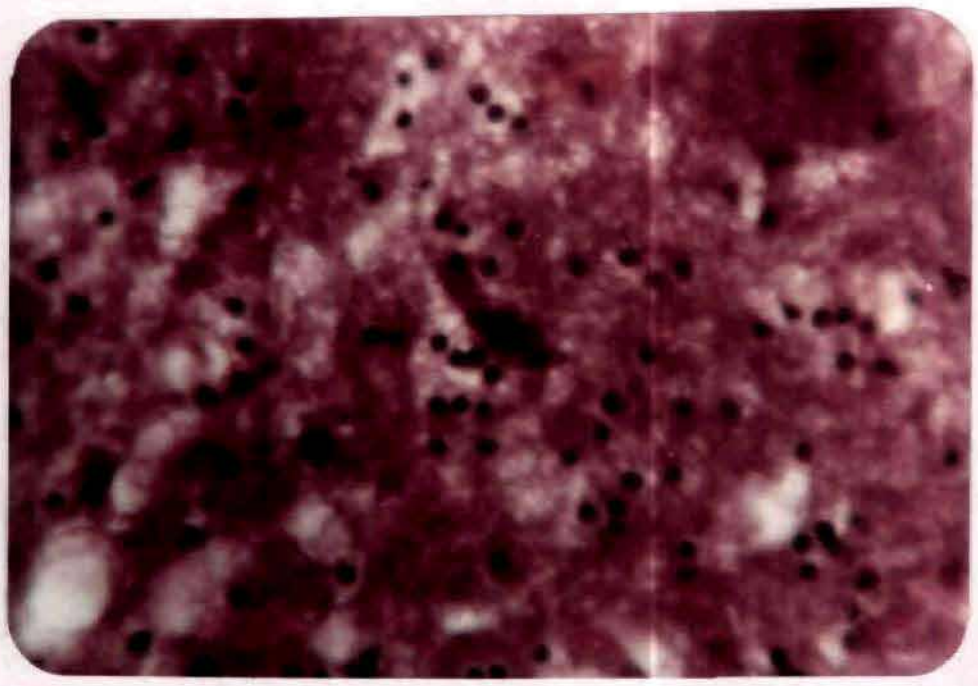


Figure 24. Cerebellum (Group I) - Swollen and rounded Purkinje cells with shortened processes and margination of nuclei. H & E x320.

Figure 25. Cerebellum (Group I) - Showing:
1. Thinning of molecular layer.
2. Loss of Purkinje cells.
3. Low density of granular layer.
4. Encroachment of cells of granular layer into the molecular layer H & E x100.



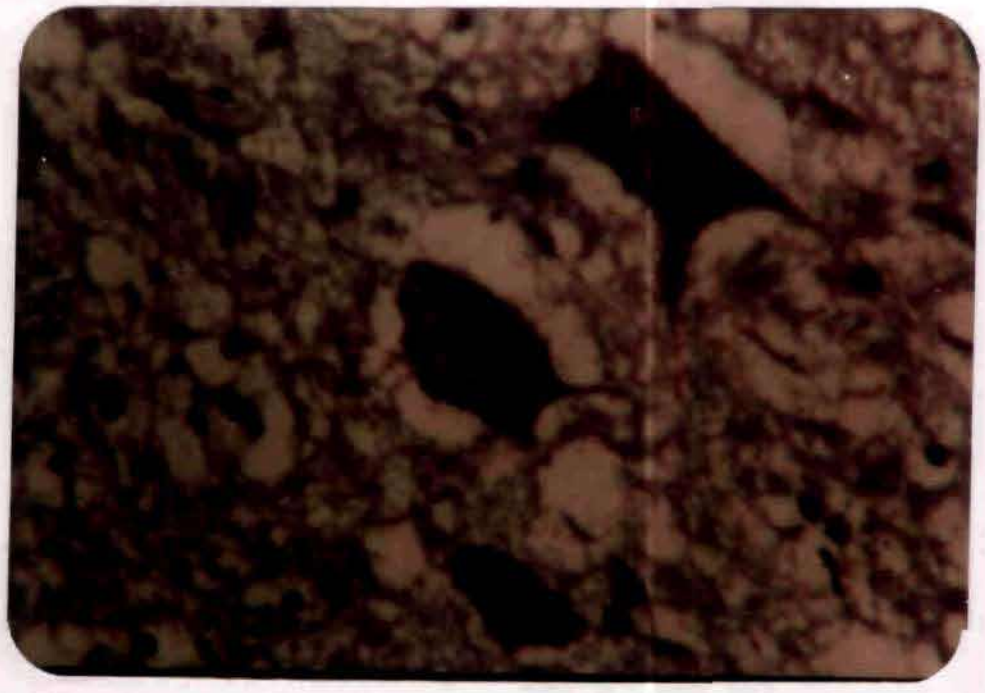
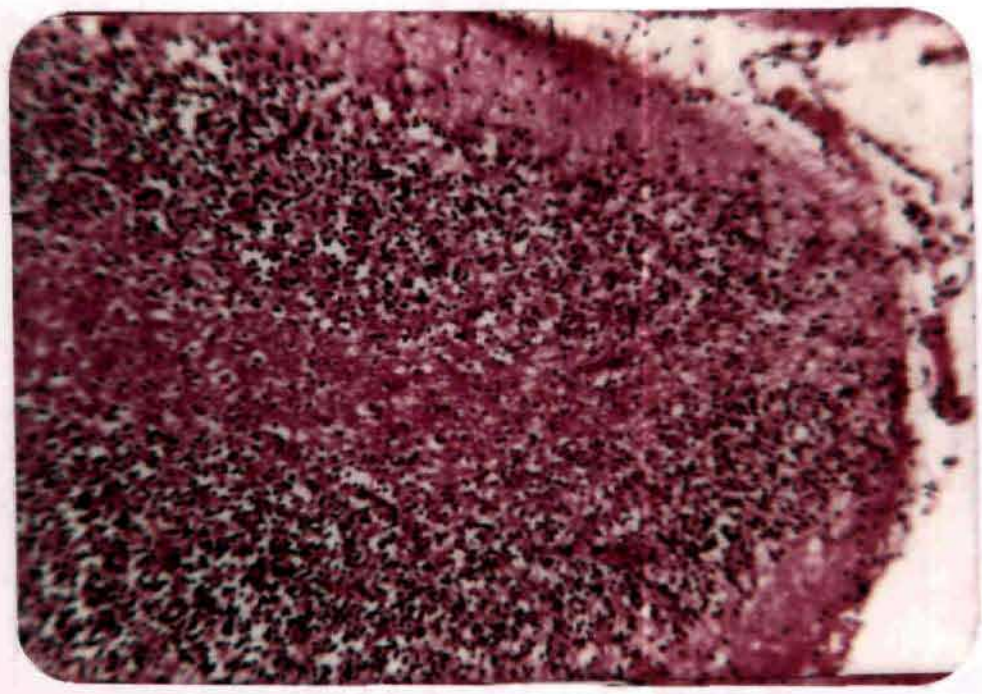


Figure 26. Spinal cord (Group I) - Neurons showing:
1. Margination of the nucleus.
2. Central chromatolysis.
3. Swelling and shortening of processes. Thionin staining x320.

Figure 27. Spinal cord (Group I) - Note satellitosis and neuronophagia. H & E x320.



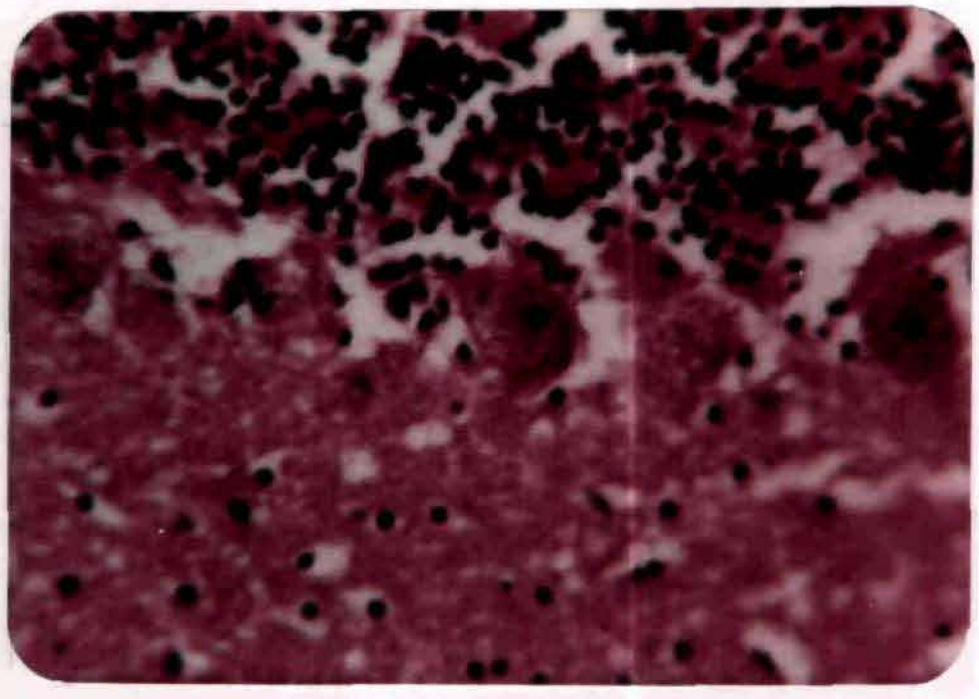
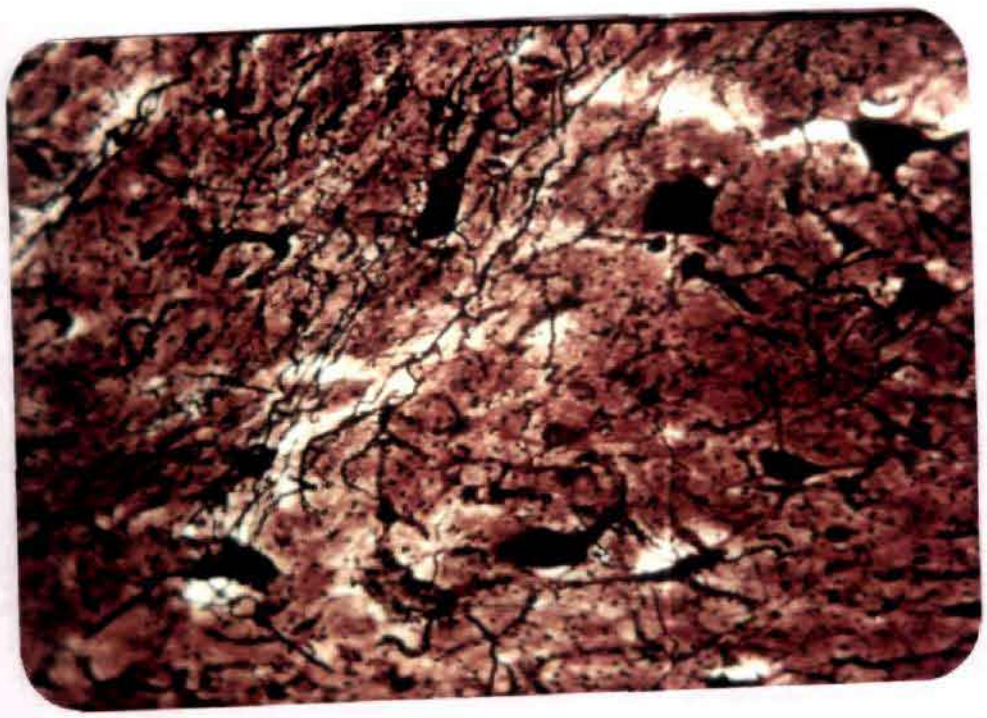
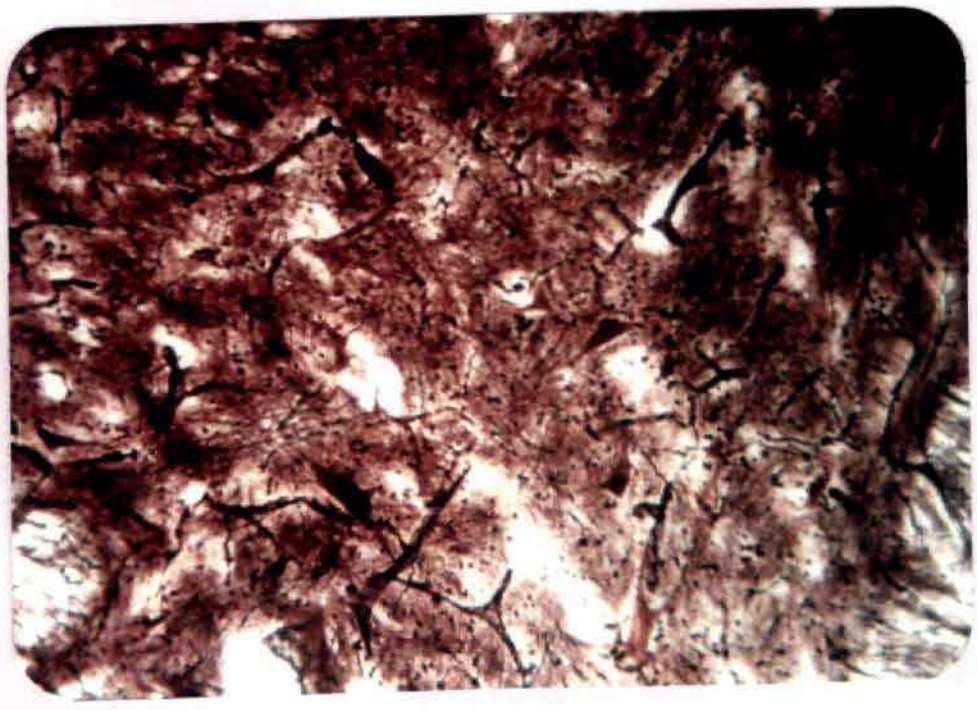


Figure 28. Spinal cord (control) - Astroglia cells with their processes Hortega's silver carbonate method x80.

Figure 29. Spinal cord (Group I) - Proliferation of glial fibres. Hortega's silver carbonate method x80.



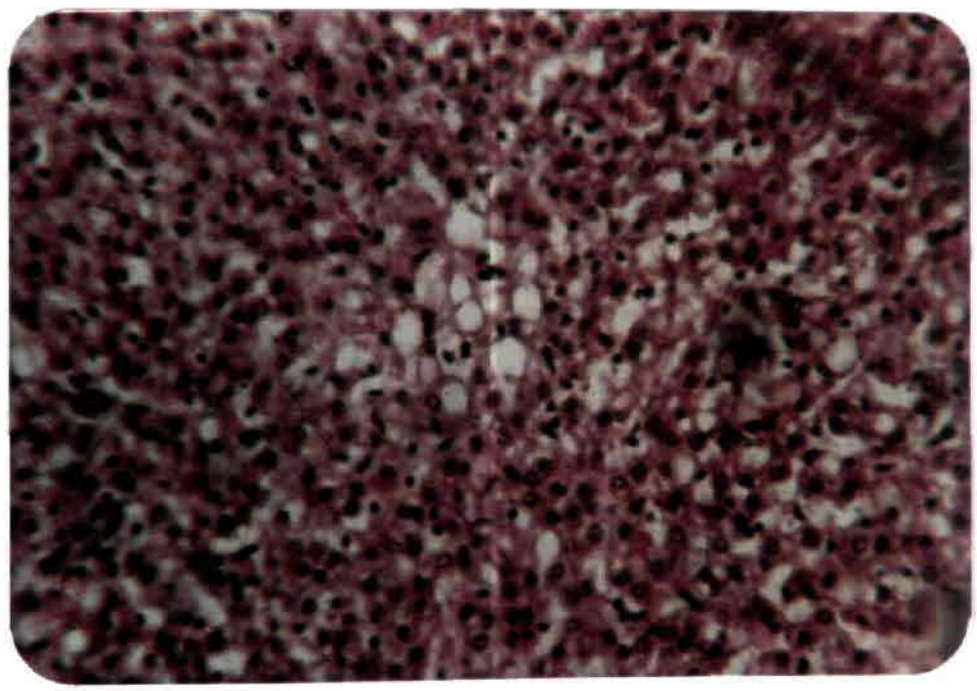


4.5.2.4 Liver. Degenerative changes in the liver parenchyma were observed in almost all the goats. Hepatic cells in the central zone showed more or less advanced degenerative changes. The cells were swollen and contained many pink stained granules. Sinusoids were occluded by the swollen hepatic cells. Vacuolar degeneration of hepatocytes around the central vein was prominent (Fig 30). The frozen sections were found to be negative for fat with Sudan black B and oil red 'O' and the alcohol fixed sections were negative for glycogen with Bests, carmine stain. Mild bile duct proliferation with Kupffer cell hyperplasia was observed in goats that died beyond 65 days. Sinusoids and portal vessels were engorged (Fig 31).

4.5.2.5 Kidneys. Congestion in the cortex and medulla was a constant finding (Fig 32). At places the vessels in between the tubules were ruptured and red blood corpuscles were found free in the interstitial tissue. The extent of intertubular haemorrhage ranged from mild to severe degree. In some cases the haemorrhages were diffuse and severe, while in others these were mild and focal in nature (Fig 33). The lesions were were observed mainly around the glomeruli and mostly at the cortico-medullary junction. In a few goats the glomerular tufts were congested and free erythrocytes were seen between the glomerular capillaries and Bowman's capsule. Haemorrhages were also observed between the tubules around the glomeruli. Vacuolation of epithelial cells, especially in proximal convoluted tubules was prominent (Fig 34). The forzen sections were found to be negative for fat and glycogen with oil red 'O' and Best's carmine stain, respectively. More severely affected kidneys showed dilated distal convoluted tubules and loops of Henle. Tubular epithelial lining showed vacuolization. The lumen of the tubules was narrowed with granularity of the cytoplasm of the epithelial cells. The cells lining these tubules were swollen and separated from each other and also from basement membrane. In some tubules the lining epithelium was denuded, while in others casts were observed (Fig 35).

Figure 30. Liver (Group I) - Showing vacuolar degeneration. H & E x20.

Figure 31. Liver (Group I) - Showing:
1. Mild bile duct proliferation.
2. Kupffer cell hyperplasia.
3. Engorged portal vessels. H & E x270.



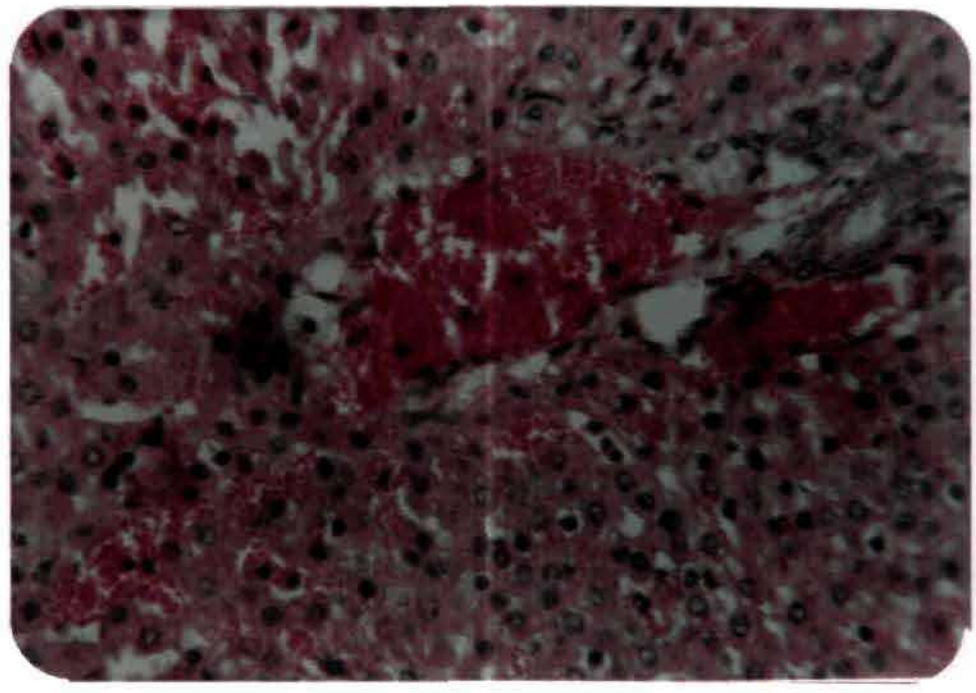
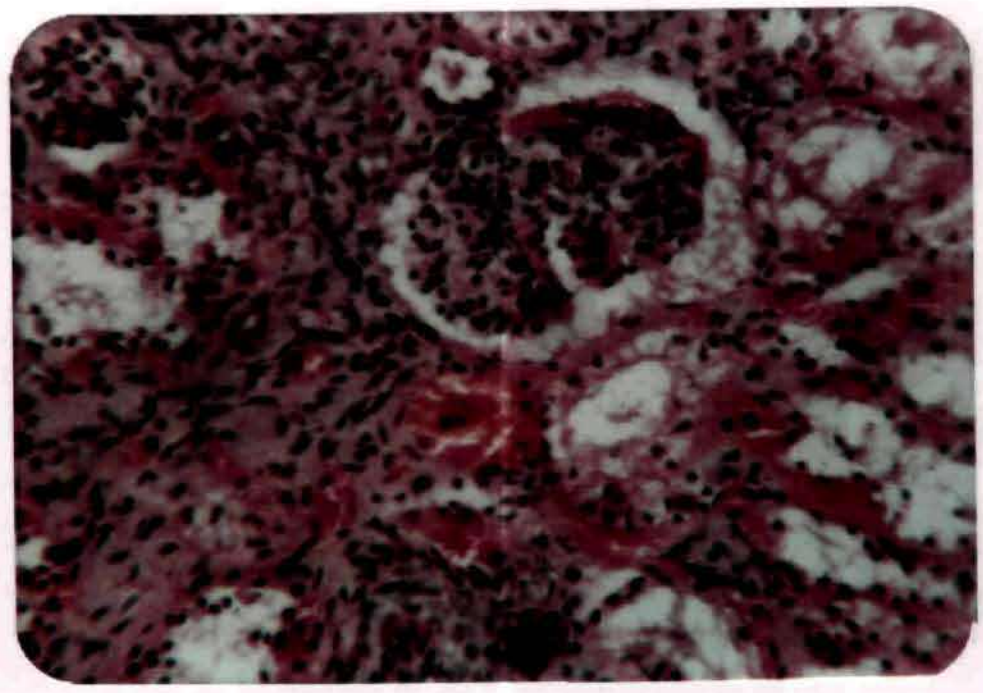


Figure 32. Kidney (Group I) - Showing:
1. Haemorrhages between the glomerular tuft and Bowman's capsule.
2. Intertubular haemorrhages. H & E x200.

Figure 33. Kidney (Group I) - Focal haemorrhages in the medulla. H & E x80.



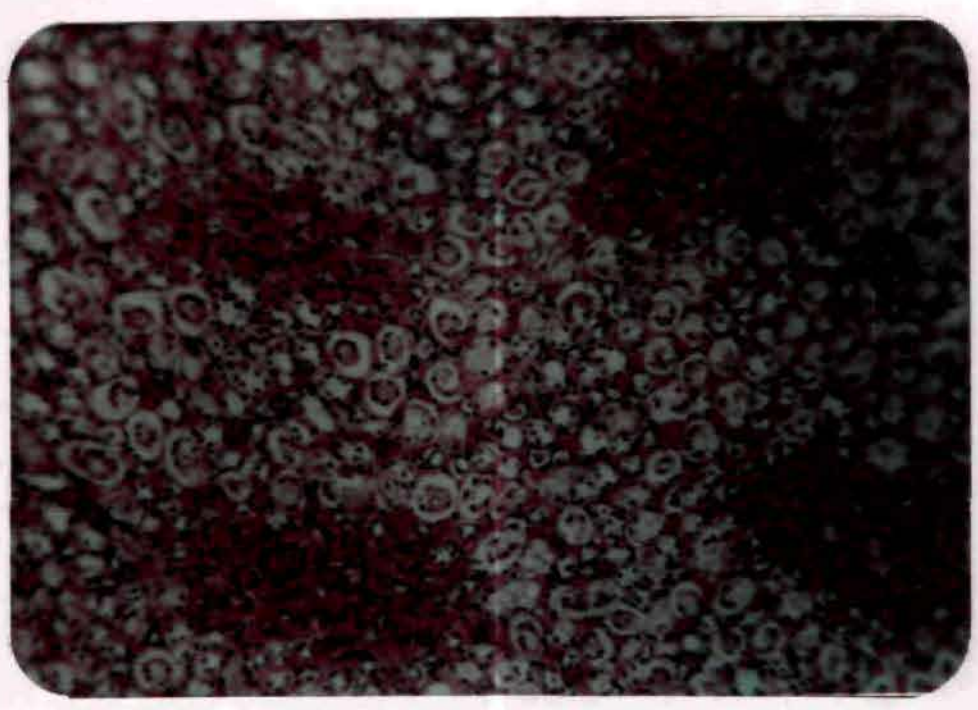
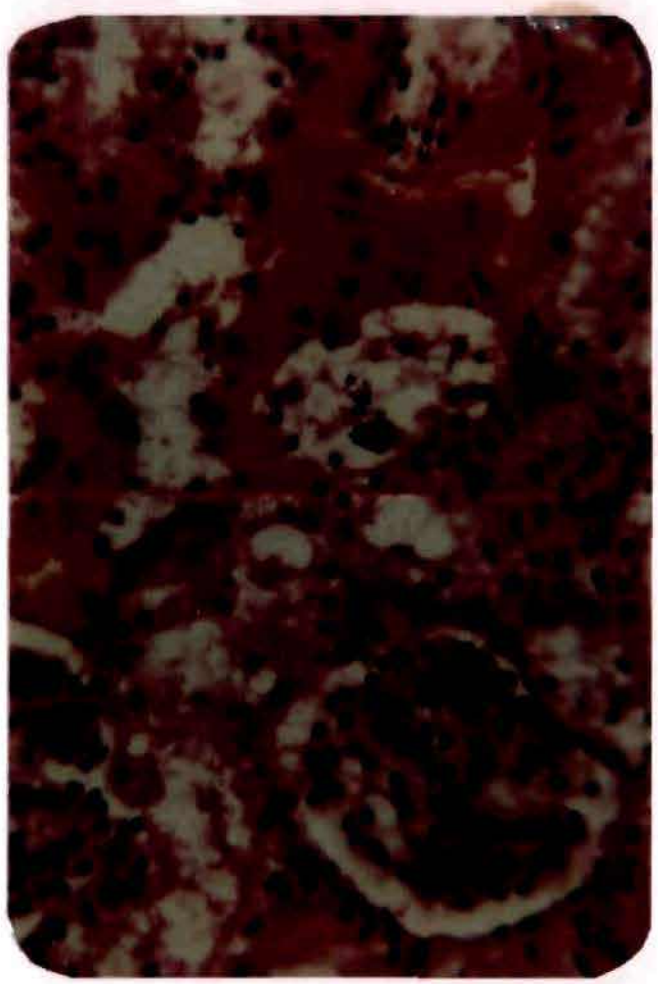
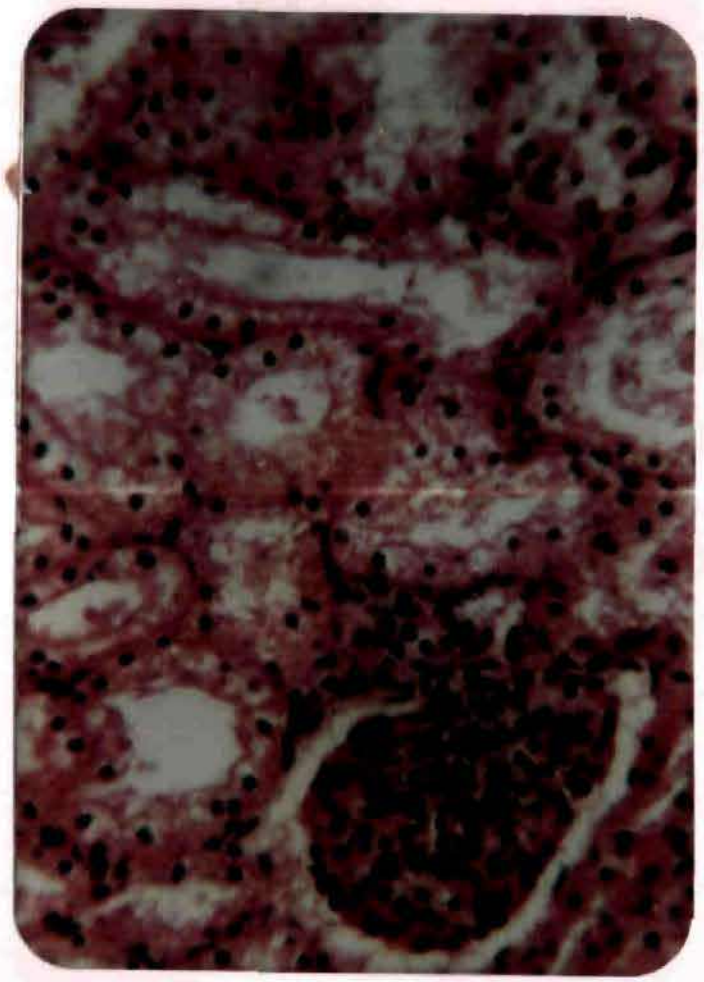


Figure 34. Kidney (Group I) - Showing:
1. Vacuolization and degeneration of tubular epithelium.
2. Oedema between Bowman's capsule and glomerulus.
3. Periglomerular haemorrhage. H & E x150.

Figure 35. Kidney (Group I) - Showing:
1. Vacuolization in tubular epithelial cells.
2. Casts in the lumina of the tubules. H & E x270.





4.5.2.6 Heart. Focal haemorrhages were seen in the interstitial tissue of myocardial fibres (Fig 36). The muscle fibres lost their striations with occasional leucocytic infiltration in the area. Muscle cells were swollen and pink stained.

5.4.2.7 Lungs. Alveolar capillaries were engorged with focal haemorrhages at places. Some of the alveoli and bronchi contained pink stained homogeneous material while some others revealed emphysematous changes (Fig 37).

4.5.2.8 Spleen. Splenic sinusoids were engorged. Trabeculae were thickened. Red pulp was engorged with atrophy of white pulp (Fig 38).

4.5.2.9 Digestive system. Capillaries in the lamina propria of the mucosa were engorged in abomasum and small intestine. Moderate desquamation of the superficial epithelium was observed.

4.5.2.10 Lymph nodes. Congestion (Fig 39) and reticuloendothelial cell hyperplasia were prominently noticed.

4.6 PATHOLOGICAL CHANGES IN GROUP II

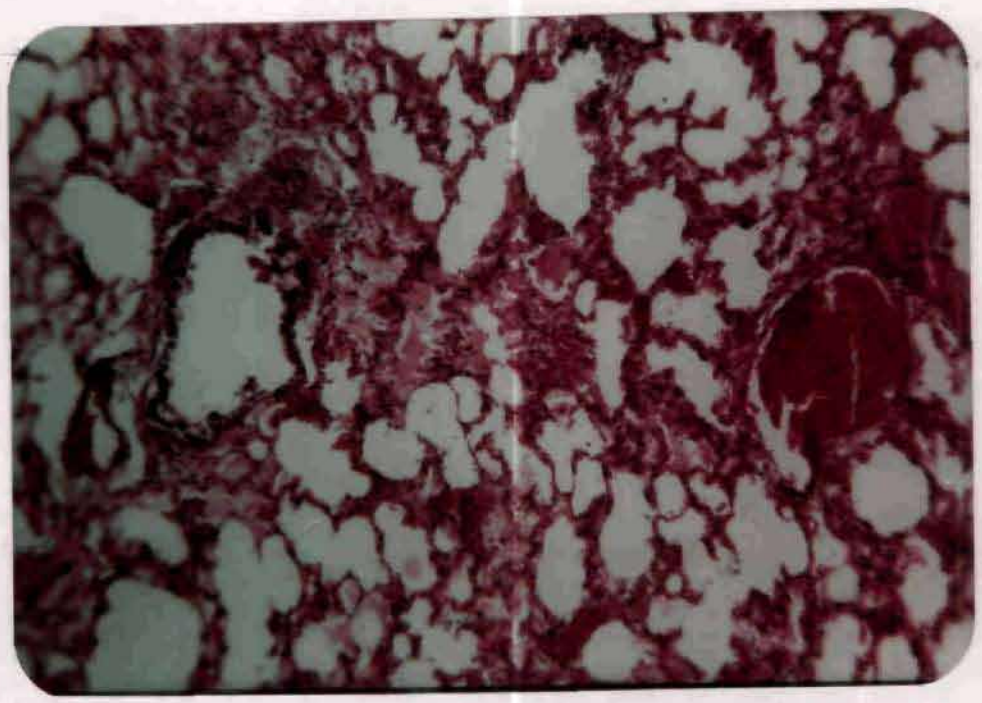
4.6.1. Gross Lesions (Group II)

At necropsy the animals were highly emaciated with severe wasting of the muscles. The organs were congested and serosanguineous fluid was observed in serous cavities of all animals.

Livers were enlarged with rounded borders. On section the cut surface bulged out with oozing of blood. In three animals greyish or yellowish white cricumscribed foci of 1 to 2 mm diameter were observed. Gall bladder was distended with yellowish thick bile.

Figure 36. Heart (Group I) - Note focal haemorrhages between the myocardial fibre bundles. H & E x240.

Figure 37. Lung (Group I) - Showing:
1. Congestion of alveolar capillaries and larger blood vessels.
2. Areas of emphysema and oedema. H & E x60.



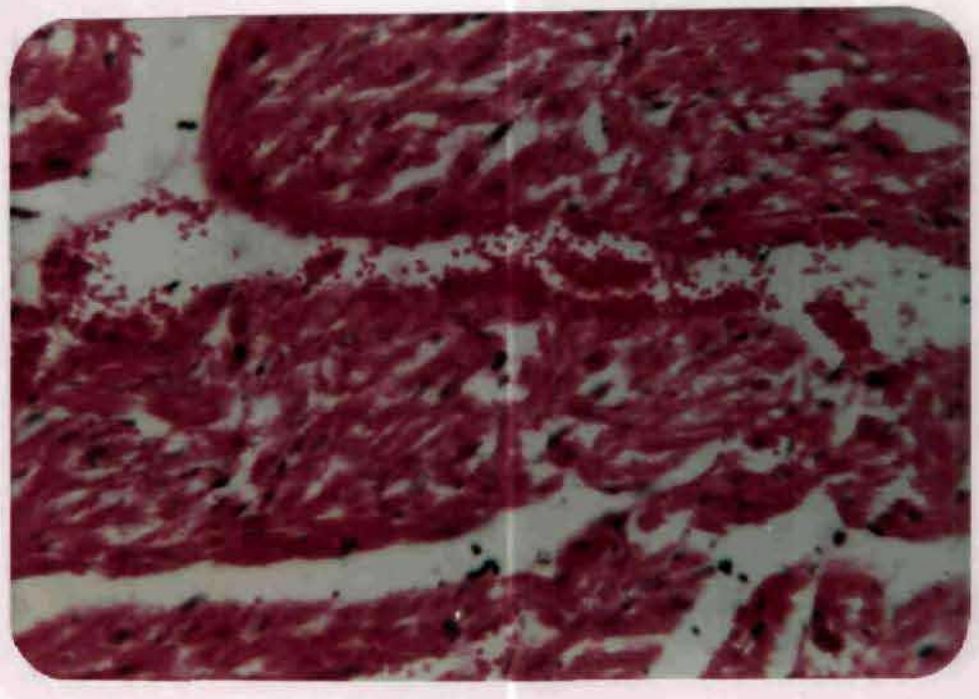
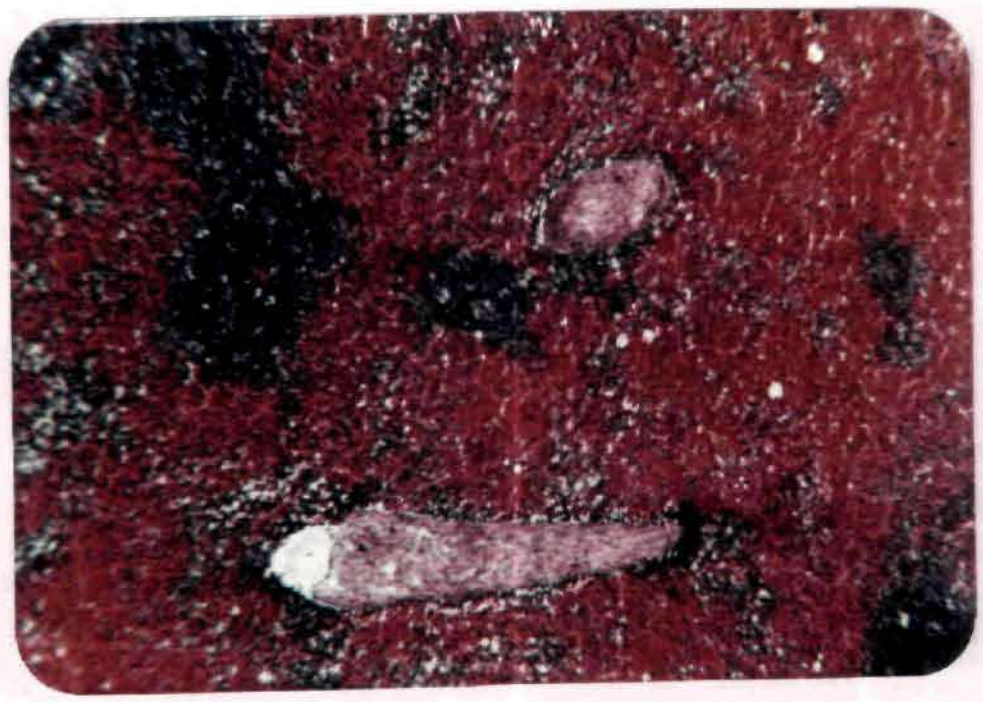
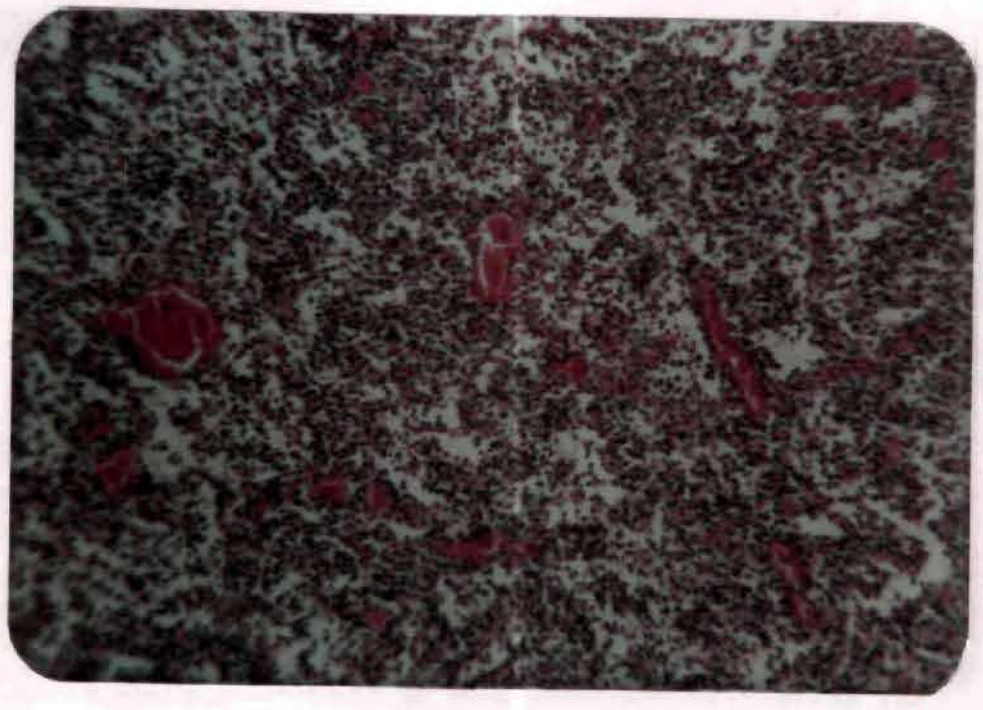


Figure 38. Spleen (Group I) - Showing engorgement of sinusoids and red pulp with atrophy of white pulp. H & E x60.

Figure 39. Lymph node (Group I) - Note engorgement of the vessels in the cortex. H & E x128.





Kidneys were congested and capsule peeled off easily. In some kidneys greyish foci of necrosis was observed on the surface. On cut section there was severe congestion of cortico-medullary junction with gelatinous fluid in the pelvis.

Heart was flabby and the blood vessels were engorged. There was gelatinization of the fat over the coronary groove (Fig 40). Petechial haemorrhages over the epicardium and endocardium were observed. The chambers were dilated. Myocardium was soft and frankly yellow in colour particularly in inner third of the wall of the left ventricle.

Lungs were heavier, dark brown and leathery. Oedema of the lungs was confined to ventral part of diaphragmatic lobes. Blood tinged frothy fluid was observed in trachea and bronchioles.

There was congestion and thickening of the intestinal mucosa. Severe enlargement of mesenteric lymph nodes was noticed (Fig 41).

Spleen was enlarged, deep reddish in colour with rounded broders. On section large amount of blood oozed out and pulp was soft and red.

Meningeal blood vessels of the brain and spinal cord were highly engorged (Fig 42). The sulci were narrowed. On section the white matter appeared soft and gelatinous.

The vertebral canal contained yellow straw coloured fluid in some animals and in other animals it was serosanguineous. The meningeal blood vessels over the lumbar and cervical regions of spinal cord were severely engorged.

Figure 40. Heart (Group II) - Note gelatinization of coronary fat.

Figure 41. Small intestine (Group II) - Severe congestion of the musoca.

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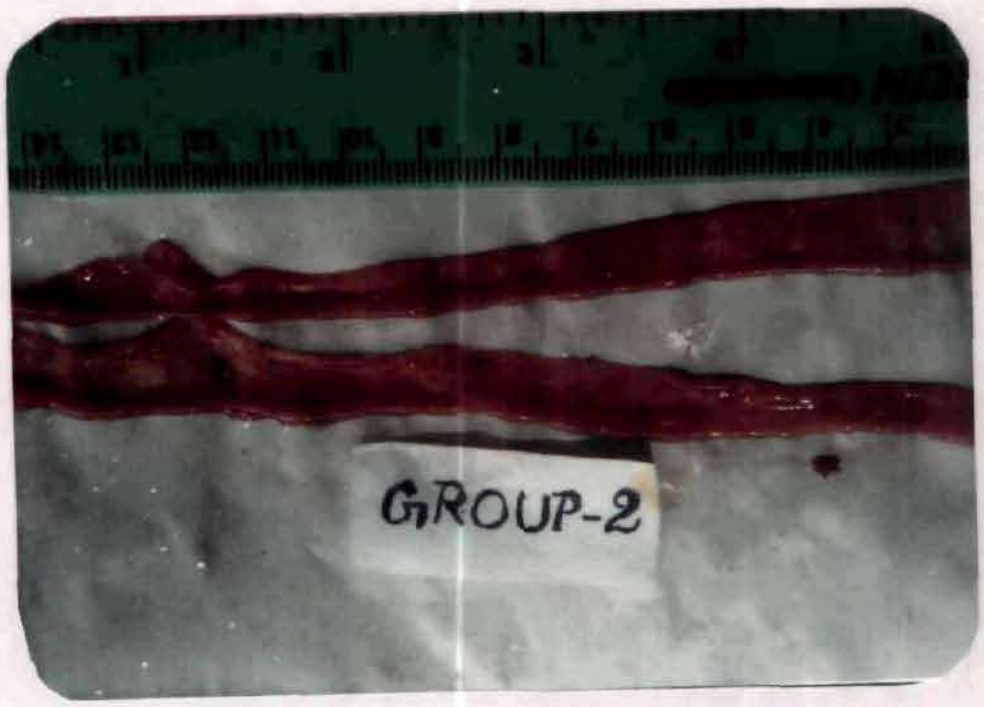

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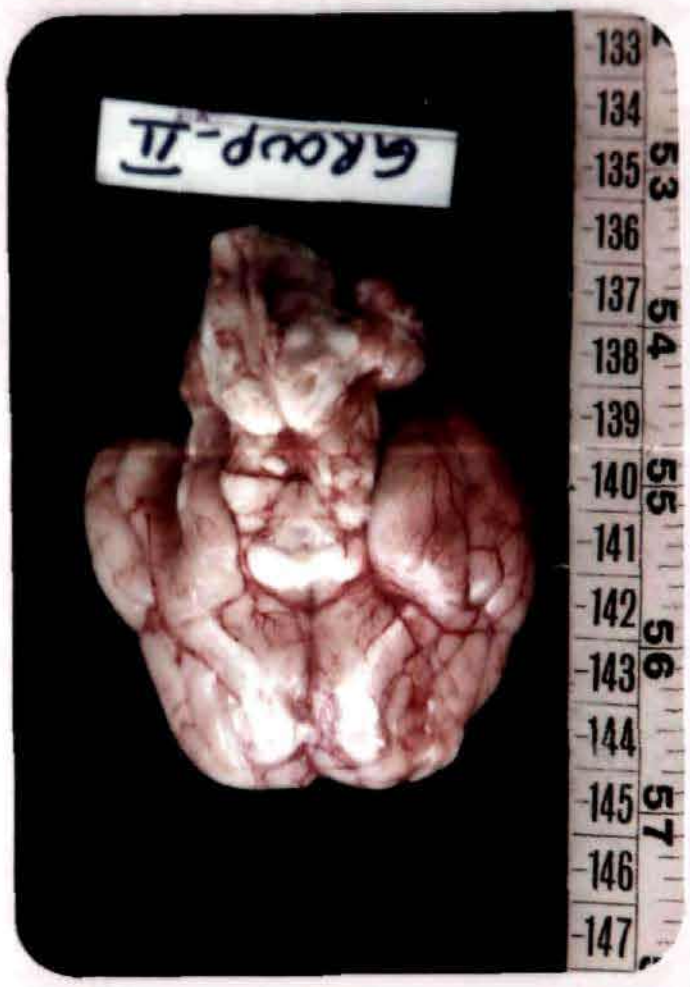
4.6.2 Microscopic Lesions (Group II)

4.6.2.1 Cerebrum. There was considerable widening of the interfibrillary space giving loose textured appearance to the white and grey matter. Blood vessels were constricted with clear halo around the vessels. Capillary congestion and haemorrhages were observed both in the white and grey matter (Fig 43). Perivascular haemorrhages and patchy serous effusions were consistently observed. The cells became irregular in their contour and nuclei were pyknotic. In some nerve cells the protoplasm was dark homogeneous with an obscure nucleus having indistinct contour. Some cells were swollen with pale cytoplasm, which was uniformly acidophilic and nuclei were pyknotic with irregular contours. Central chromatolysis was observed in most of the neurons. Satellitosis and neuronophagia of the nerve cells were seen (Fig 44). There was increased focal gliosis with proliferation of astroglial cells which was confirmed by Hortega's silver carbonate staining method (Fig 45). There was occasional presence of elongated glial cells. The myelinated fibres were spread apart widely with eosinophilic spaces. Focal areas of necrosis were occasionally observed in the molecular layer and amorphous layer of cerebrum (Fig 46 & 47)).

4.6.2.2 Cerebellum. The changes were observed mainly in the Purkinje cells. Focal and diffuse absence of Purkinje cells was observed (Fig 48 & 49). An occasional normal Purkinje cell was seen among many pale stained ghost forms which still retained the general outline of the Purkinje cells (Fig 50). The processes of the Purkinje cells were shortened and the cells were swollen assuming a rounded form. The margins of the cells became indistinct from the adjacent ground substance. In a few, the cell body became foam-like with loss of cellular details. Central chromatolysis was observed in some Purkinje cells. The nucleus was pushed to one side in some Purkinje cells (Fig 51). Focal absence of the cells in Purkinje cell layer presented a reticulated appearance. This finding was observed in goats died at later stage of the experiment (Fig 52).

Figure 42 Brain (Group II) - Moderate congestion of the vessels. H & E x60.

Figure 43. Cerebrum (Group II) - Showing:
1. Congestion of the capillaries and vessels.
2. Haemorrhages. H & E x80.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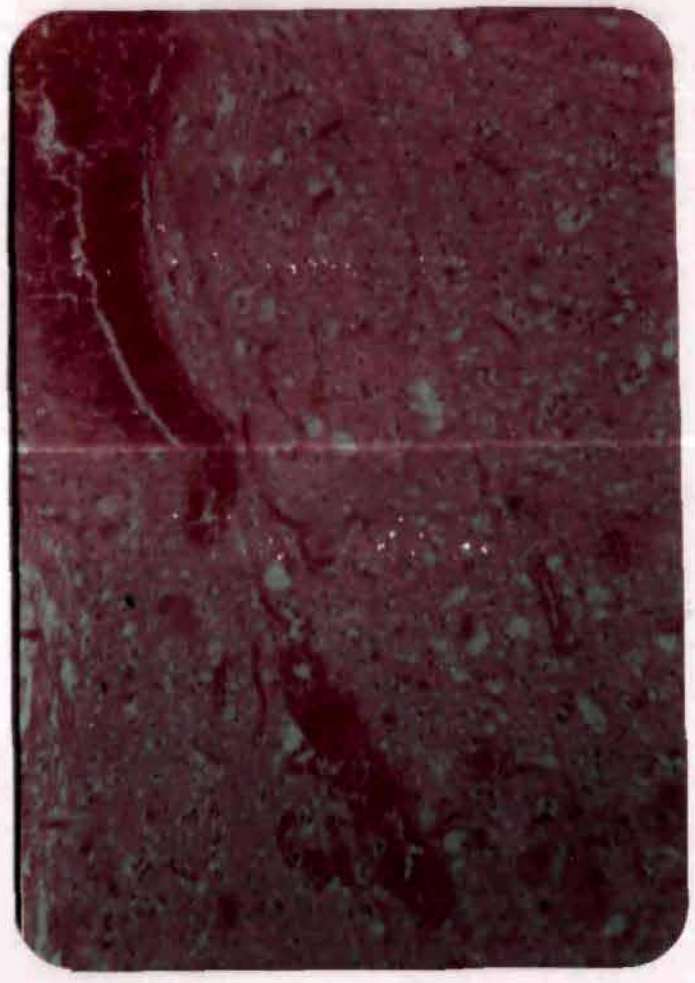

Figure 44. Cerebrum (Group II) - Showing satellitosis and neuronophagia. H & E x80.

Figure 45. Cerebrum (Group II) - Showing gliosis. Hortega's silver carbonate method x150.

Figure 46. Cerebrum (Group II) - Focal necrotic areas in the molecular layer. H & E x100.

Figure 47. Cerebrum (Group II) - Focal necrotic areas in the amorphous layer. H & E x100.

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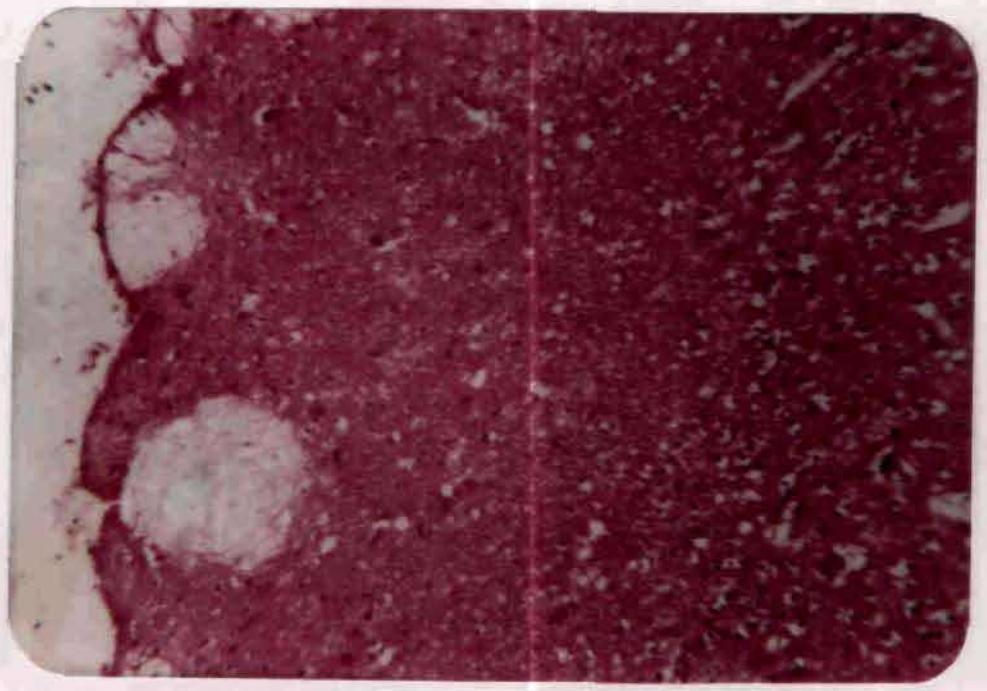


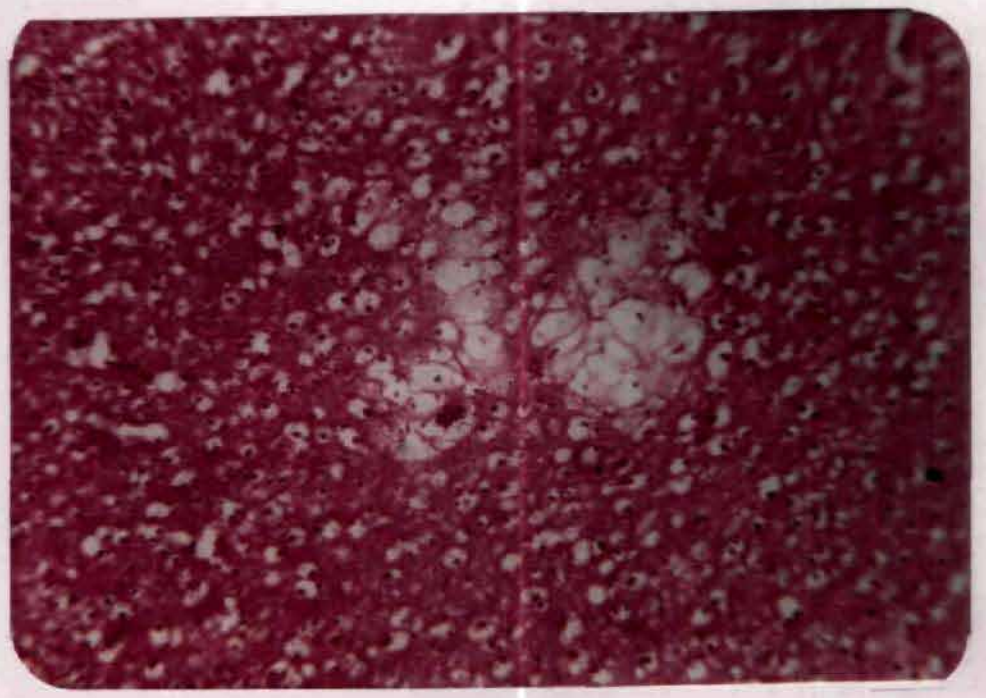
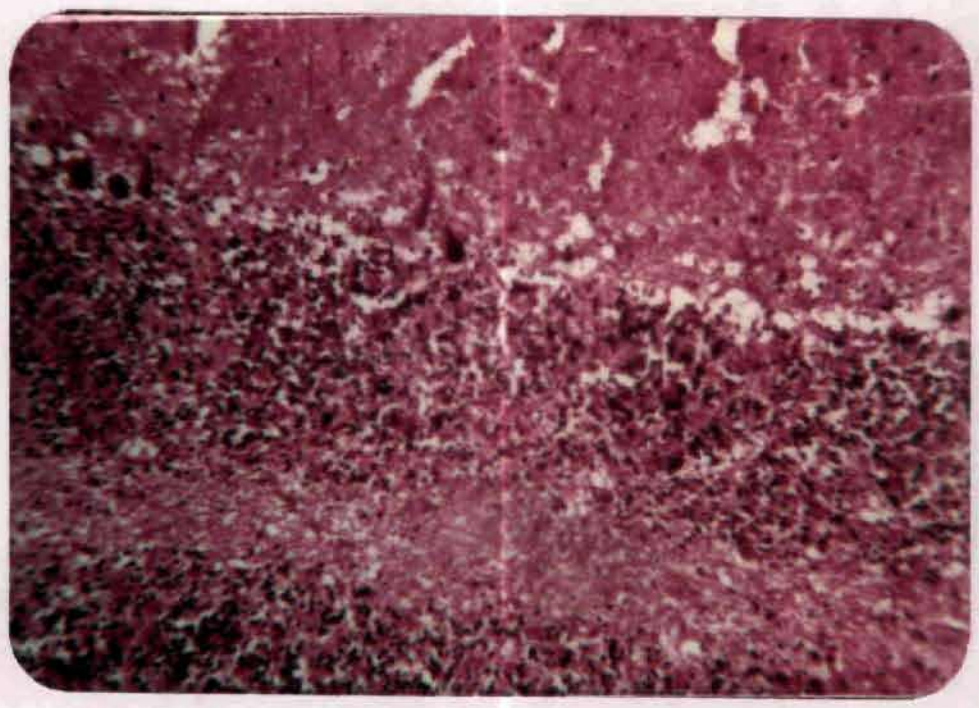


Figure 48. Cerebellum (Group II) - Note focal absence of Purkinje cells. H & E x100.

Figure 49. Cerebellum (Group II) - Diffuse absence of Purkinje cells. H & E x54.



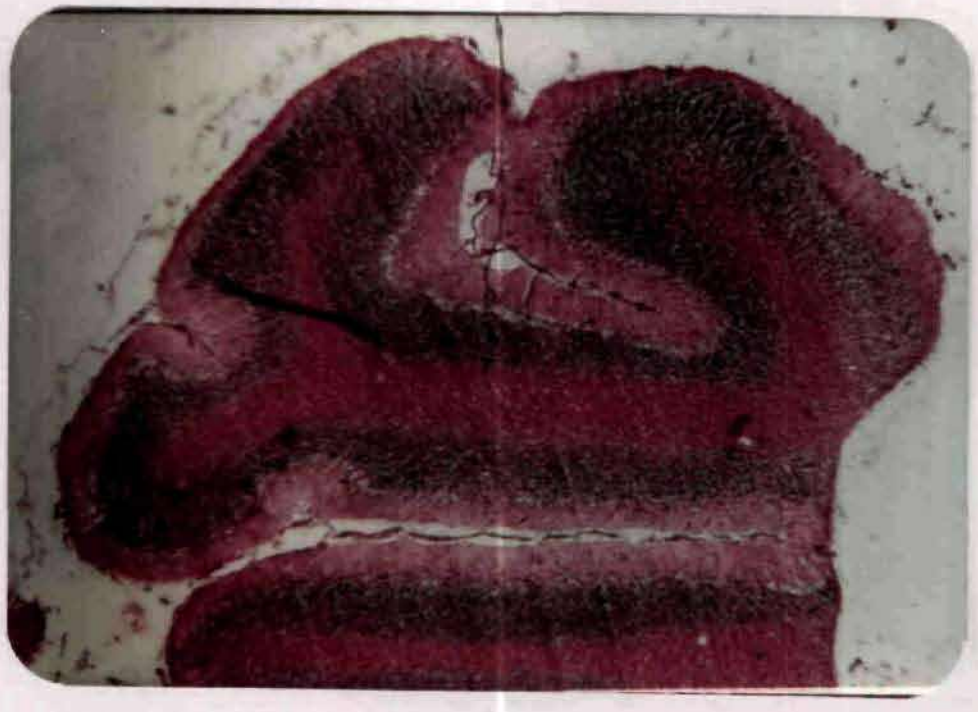
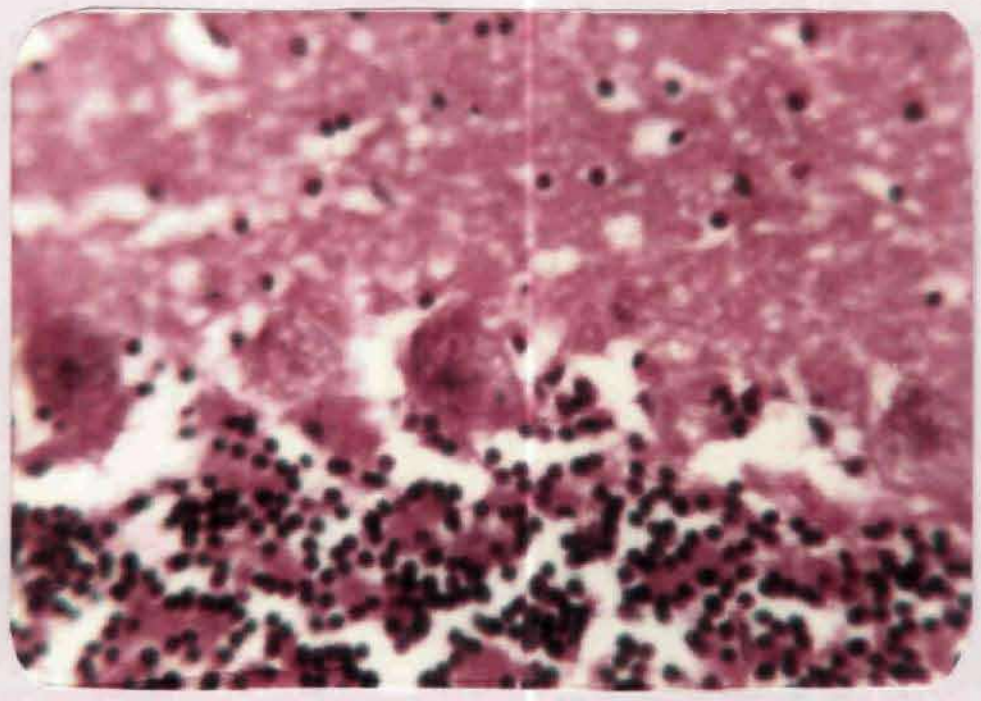
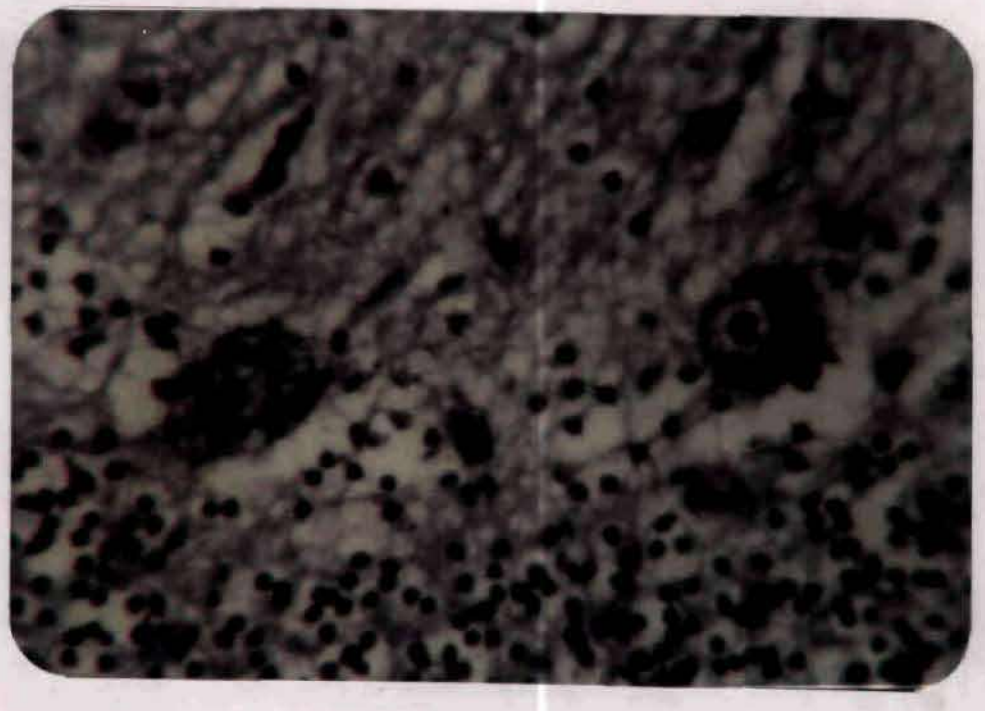


Figure 50. Cerebellum (Group II) - Ghost forms of Purkinje cells with indistinct cellular outlines, lightly stained cytoplasm and a distinct nuclei. H & E x320.

Figure 51. Cerebellum (Group II) - Purkinje cells showing central chromatolysis and margination of the nucleus. Thionin staining x320.





Some Purkinje cells were displaced into granular layer. The granular layer simultaneously showed a decrease in cellular density. Satellitosis and neuronophagia of the degenerating Purkinje cells were observed. There was proliferation of astroglial cells. Loose textured appearance in the white matter was commonly observed (Fig 53 & 54).

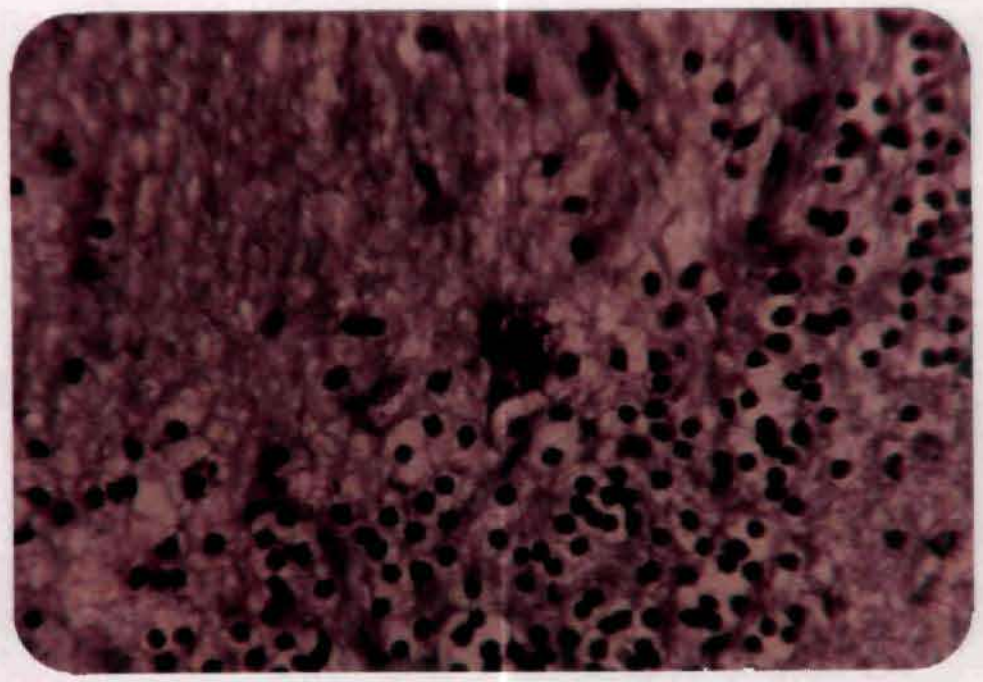
4.6.2.3 Spinal cord. The changes were observed mostly in the thoracic and lumbar portions of the spinal cord. Focal haemorrhages and capillary congestion were observed in both white and grey matter (Fig 55). Pale stained faded out neurons with indistinct borders were seen. In some the borders were absent and some neurons assumed round shape. Most of the cells showed shrinkage with irregular contours and pyknotic nuclei. There was an increase in the perineuronal space (Fig 56). In some neurons variable vacuolation of the cytoplasm was seen. Central chromatolysis was observed in most of the neurons and this was confirmed by Thionin and Toludine blue staining (Fig 57 & 58). Increased cellularity due to proliferation of glial cells, mostly astroglial cells of protoplasmic type were observed in the grey matter (Fig 59). Satellitosis and neuronophagia were observed.

4.6.2.4 Liver. Diffuse degenerative changes in the parenchyma were observed. The sinusoids were obliterated. In most of the cases the sinusoids, venules and capillaries were engorged with blood (Fig 60). The hepatic cells around the central vein were distended with a single large clear vacuole with indistinct eccentric nuclei. Occasionally the hepatic cells near the central veins and periportal areas were swollen and contained irregular eosinophilic bodies in the cytoplasm (Fig 62). Frozen sections stained with oil red 'O' and Sudan black B were negative for fat. The alcohol fixed sections were negative for glycogen with Best's carmine staining. Focal hepato-cellular necrosis was observed with infiltration of neutrophils (Fig 61). There was mild proliferation of bile ducts.

4.6.2.5 Kidneys. Congestion and haemorrhages were consistently observed throughout the parenchyma. There was an increase in the cellularity of the

Figure 52. Cerebellum (Group II) - Focal loss of Purkinje cells with a reticulated appearance of Purkinje cell layer. Thionin staining x320.

Figure 53. Cerebellum (Control) - Normal appearance of white matter. H & E x150.



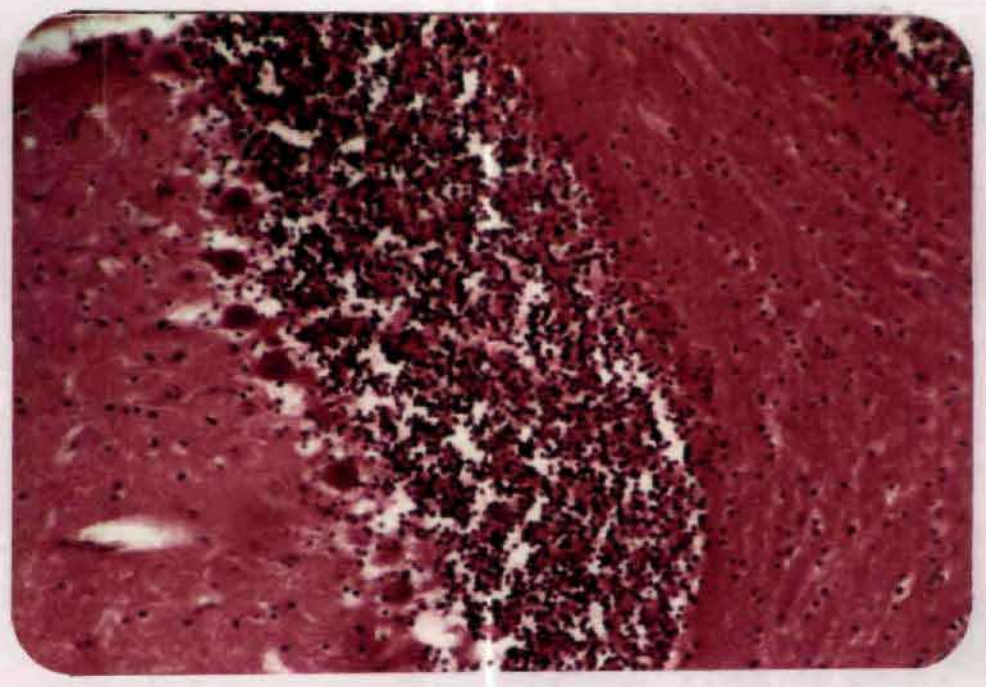
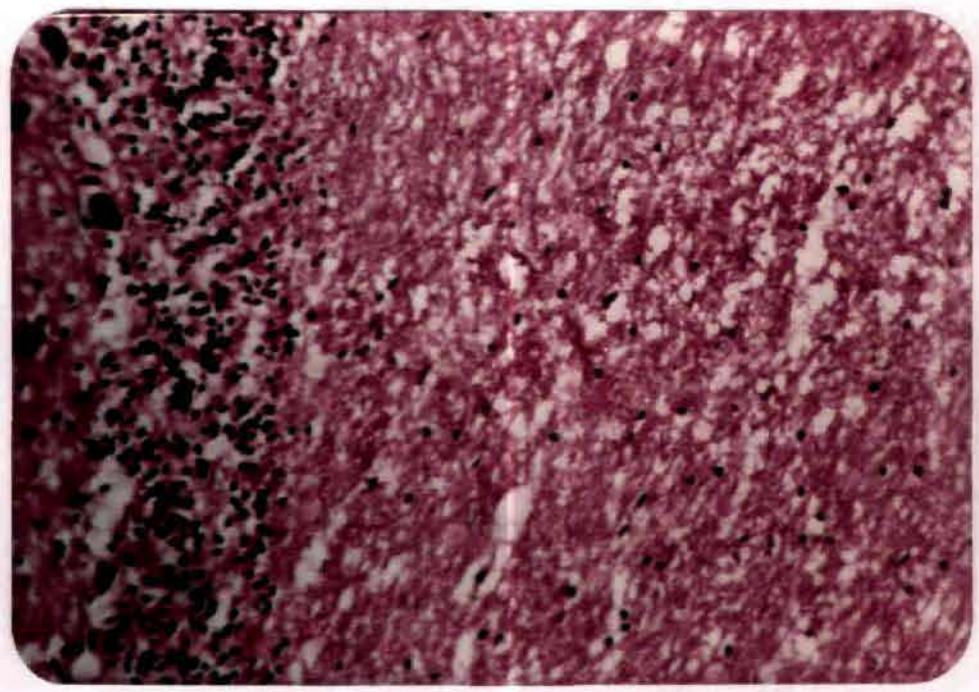


Figure 54. Cerebellum (Group II) - Note loose textured appearance in the white matter. H & E x150.

Figure 55. Spinal cord (Group II) - Focal haemorrhages between white and grey matter. H & E x240.



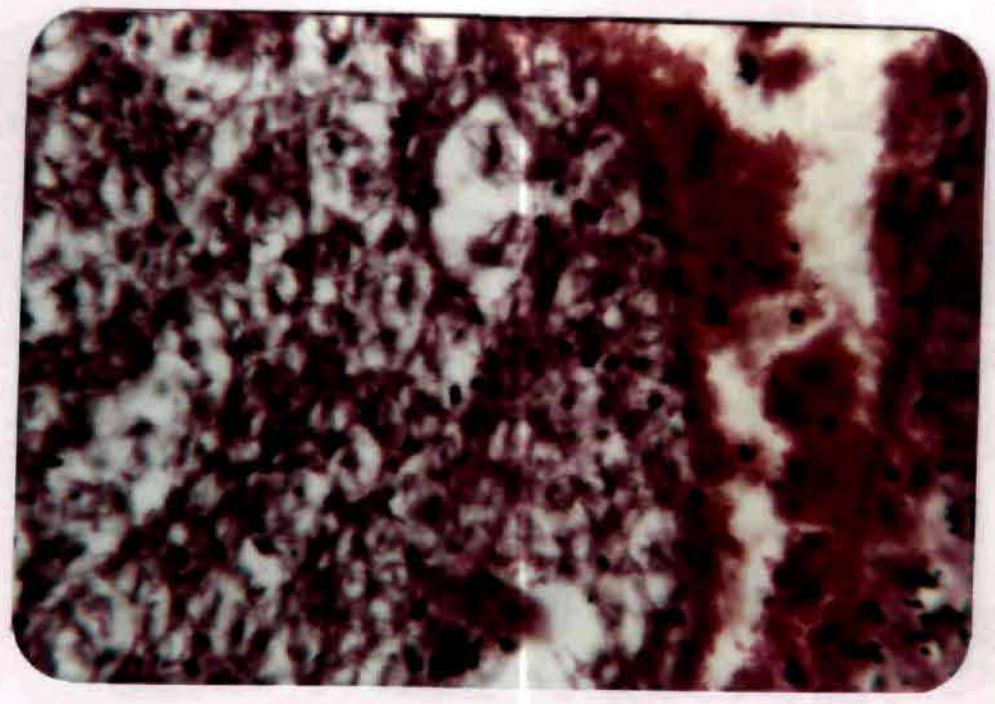
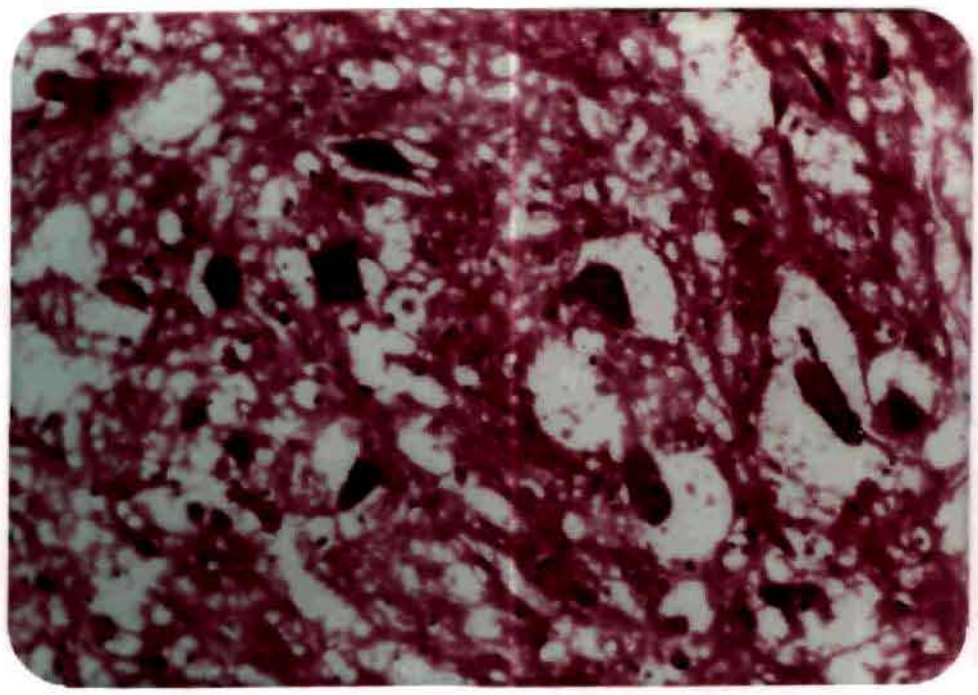


Figure 56. Spinal cord (Group II) - Increase in the perineuronal space. H & E x150.

Figure 57. Spinal cord (Control) - Normal neurons showing the nucleus and the Nissl bodies. Thionin staining x320.



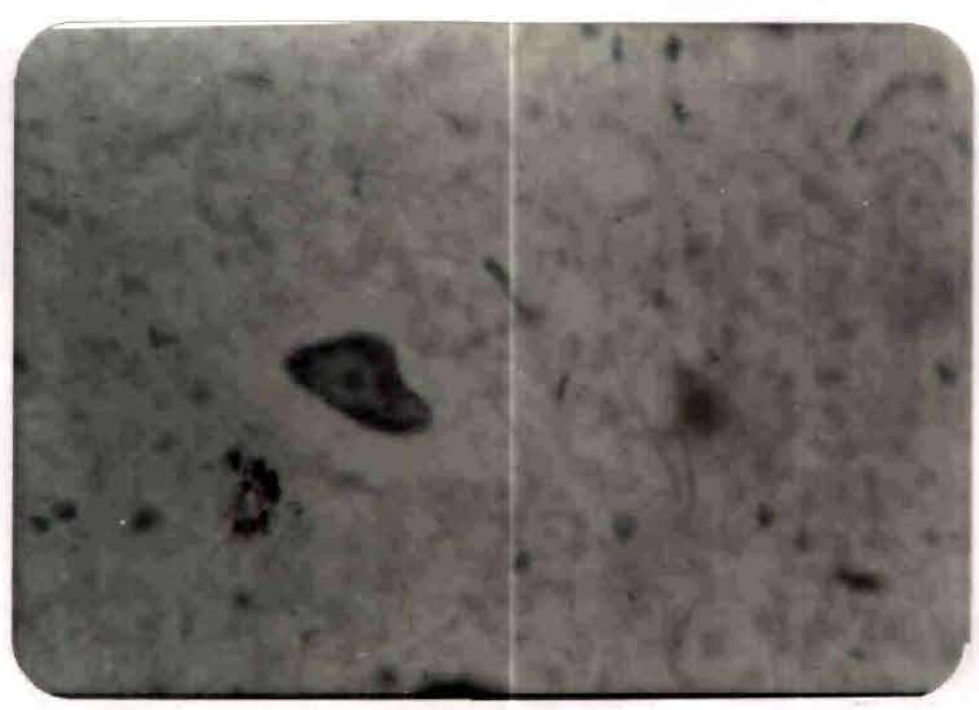
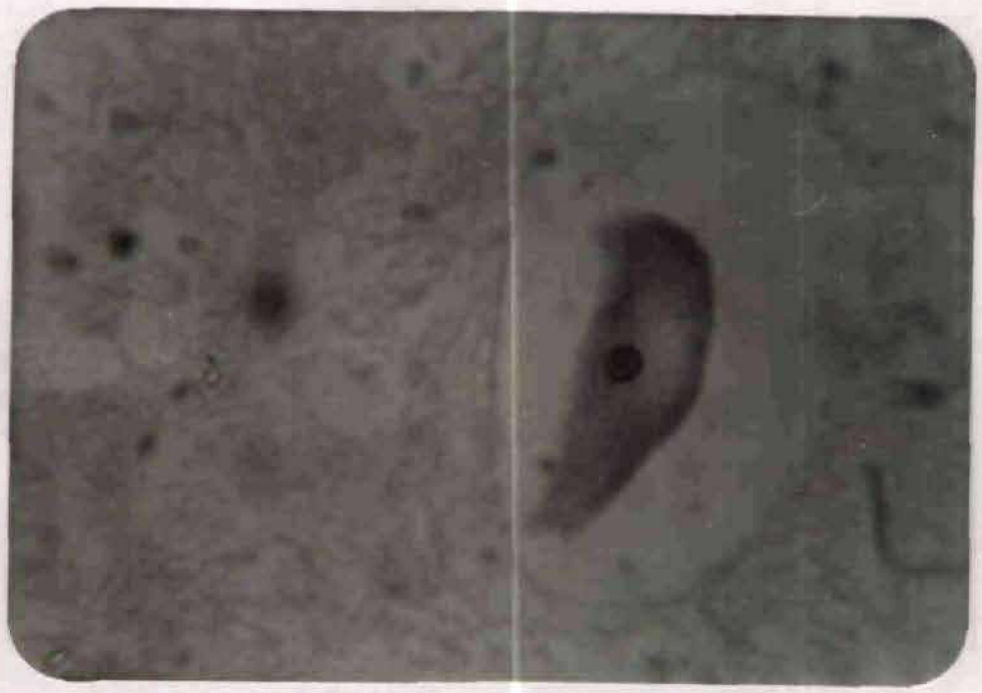


Figure 58. Spinal cord (Group II) - Note central chromatolysis. Thionin staining x320.

Figure 59. Spinal cord (Group II) - Note astroglial fibres proliferation. Hortega's silver carbonate method x 80.



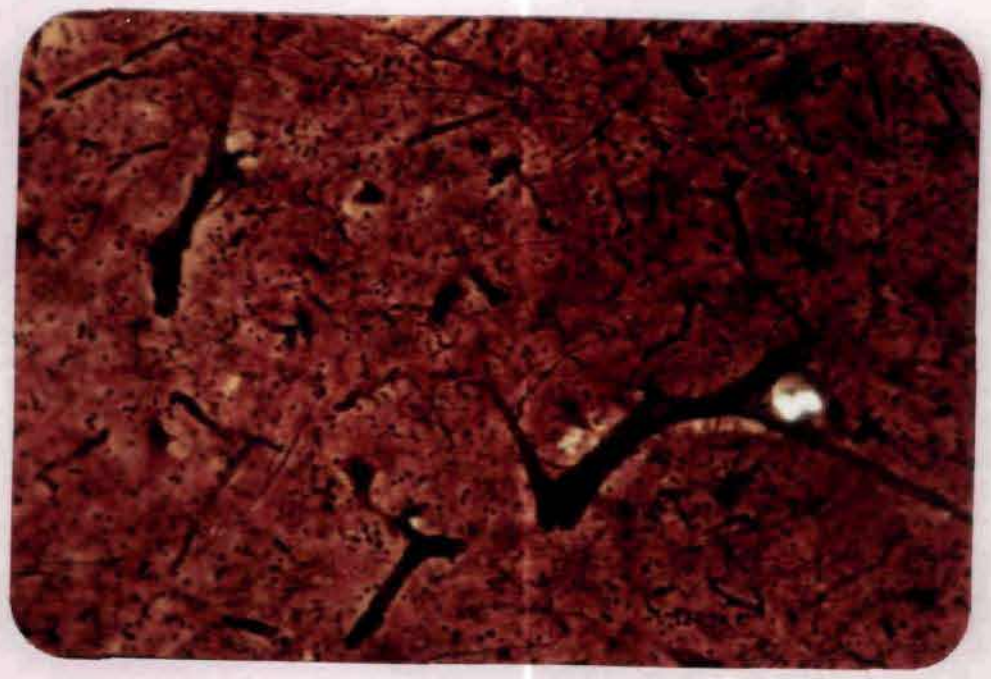
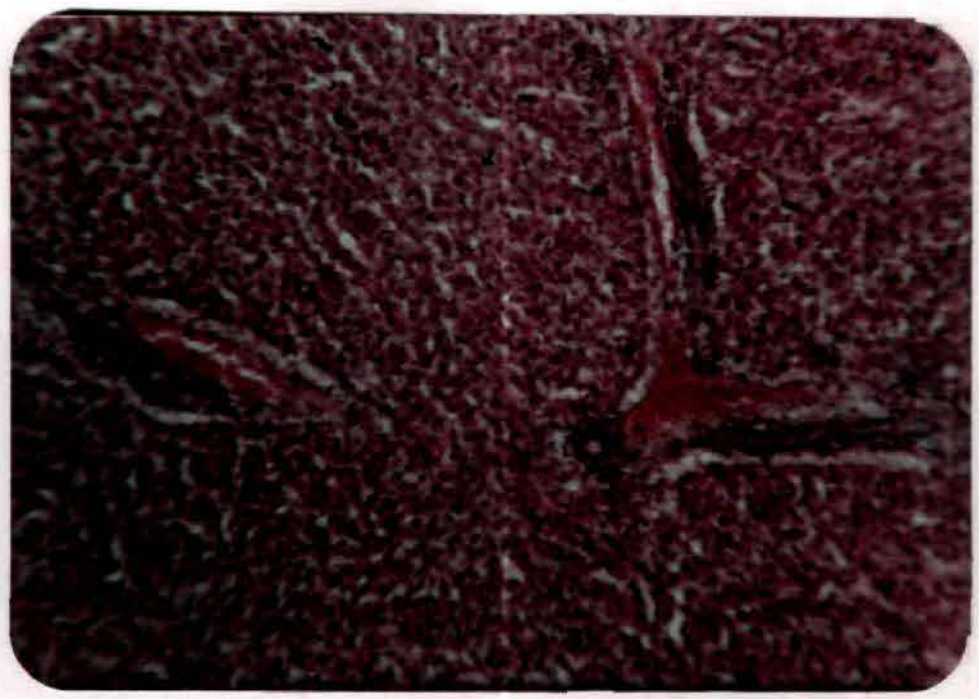


Figure 60. Liver (Group II) - Showing congestion of hepatic vessels in portal tract and mild proliferation of bile duct (arrows). H & E x100.

Figure 61. Liver (Group II) - Focal hepatic necrosis with infiltration by neutrophils and lymphocytes. H & E x450.



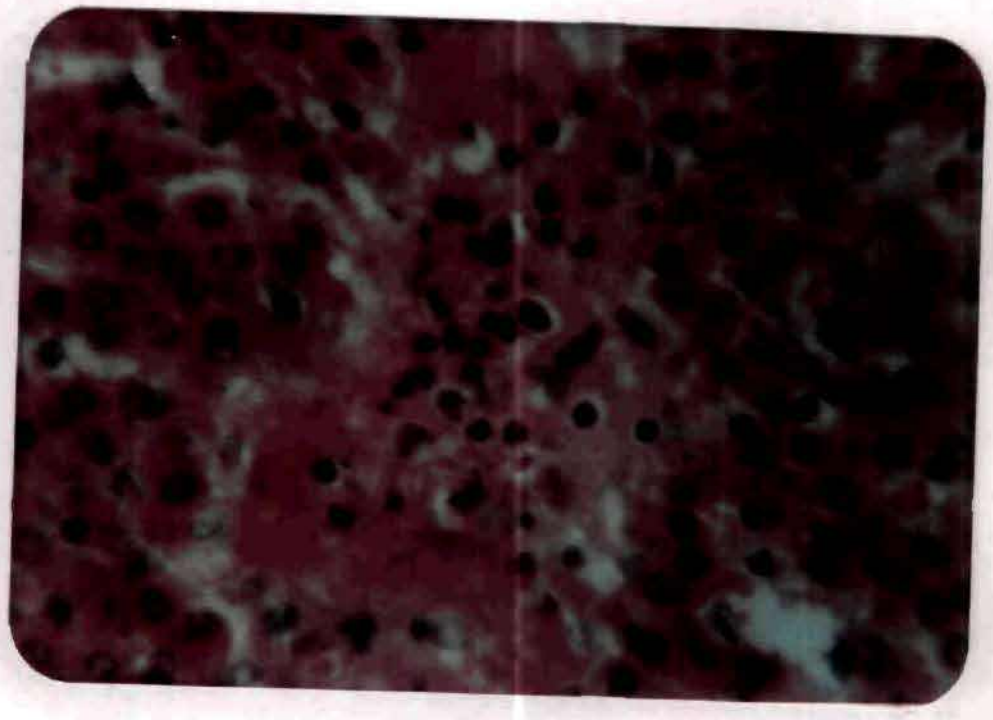
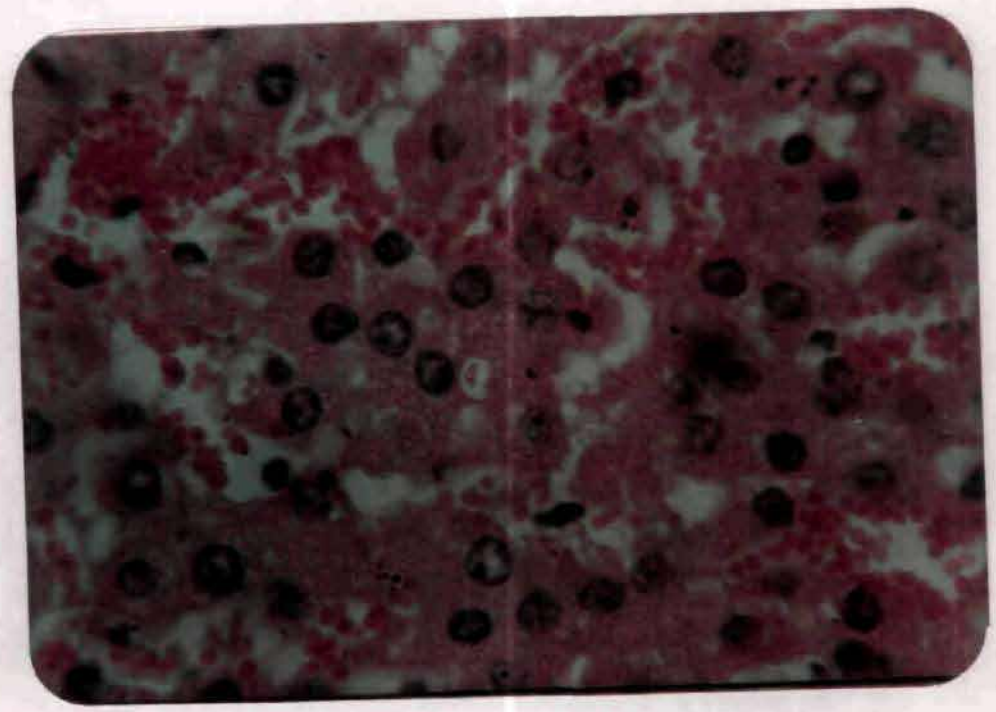


Figure 62. Liver (Group II) - Hepatocytes showing irregular eosinophilic bodies in the cytoplasm. H & E x600.

Figure 63. Kidney (Group II) - Showing:
1. Increase in the cellularity of glomerular tufts.
2. Tubular epithelial cells showing vacuolization with indistinct borders. H & E x270.



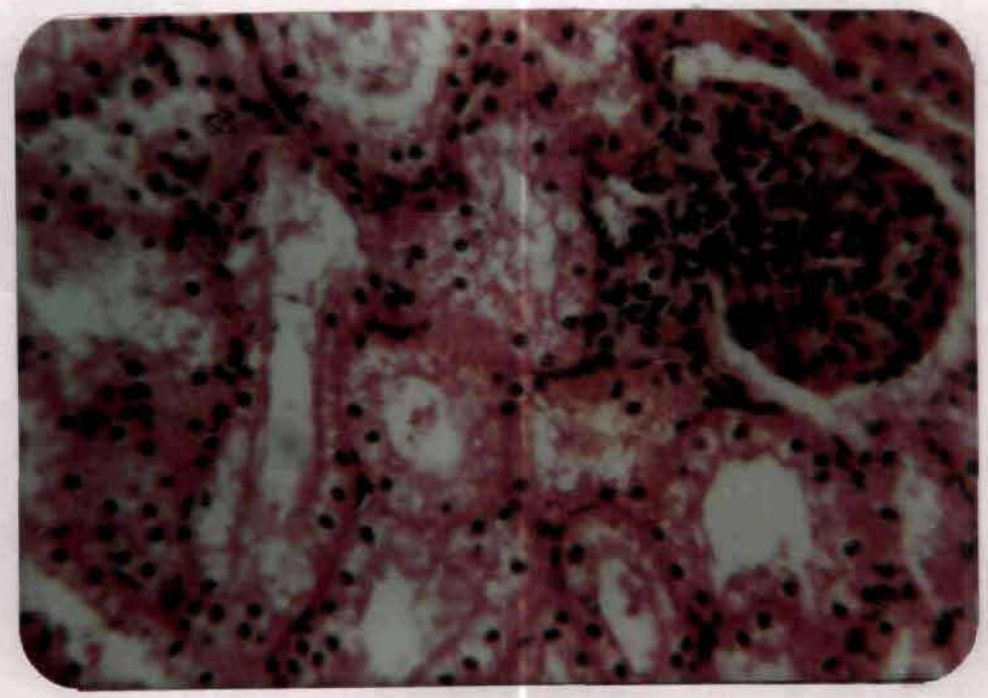
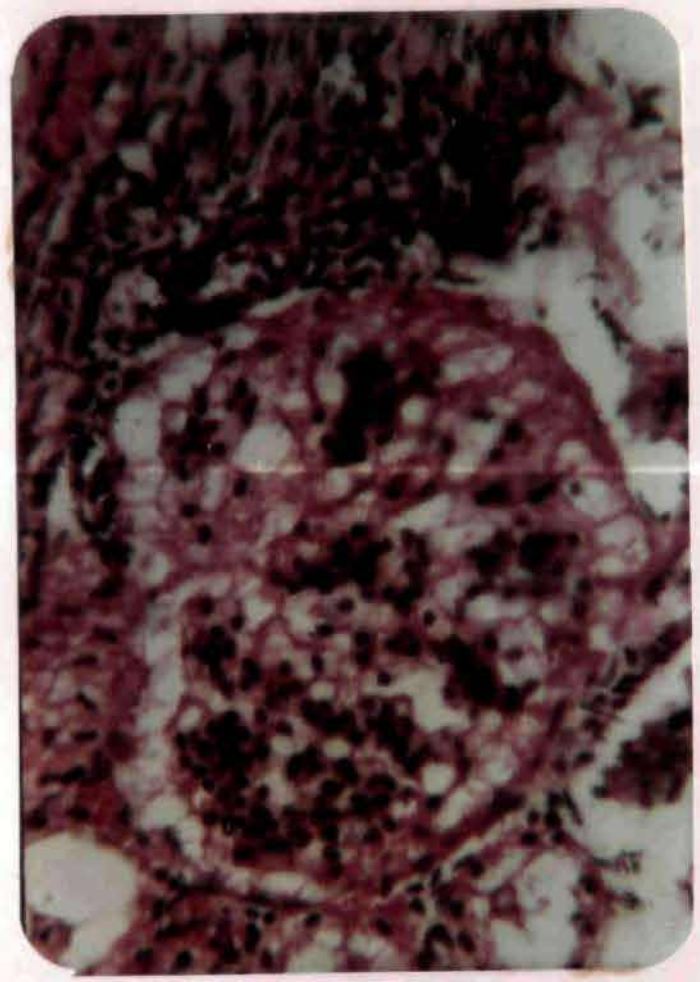
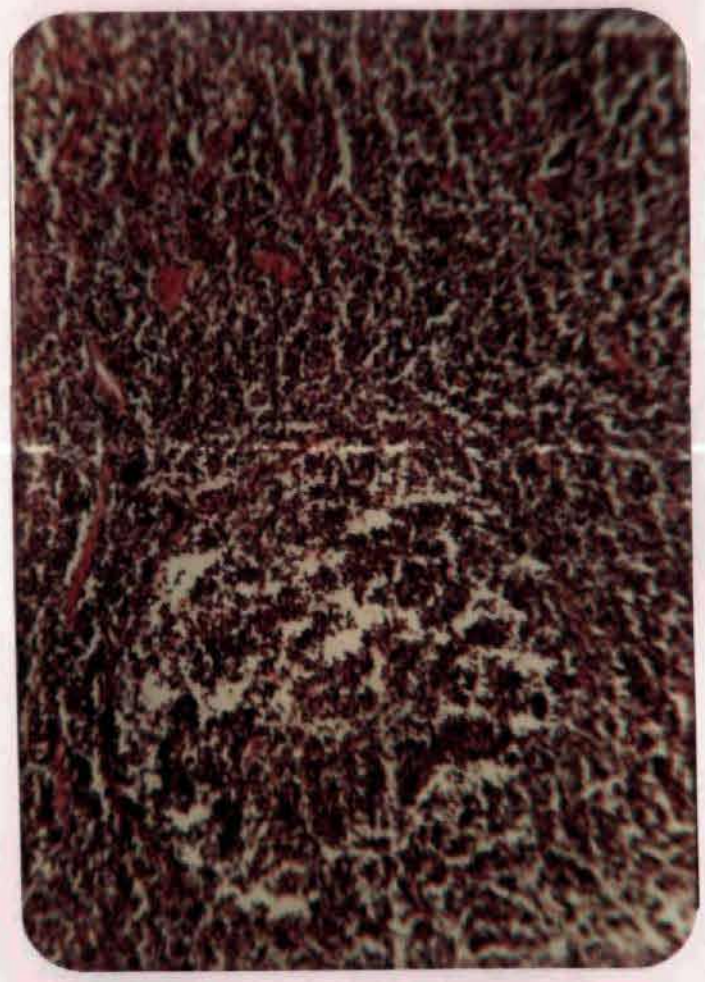


Figure 64. Kidney (Group II) - Showing periglomerular oedema and swelling of the glomerulus. H & E x270.

Figure 65. Lymph node (Group II) - Mild depletion of lymphoid cells in germinal centre. H & E x80.





glomerular tufts (Fig 62). Some of the glomeruli revealed severe atrophy. In most of the kidneys the glomerular capillaries were highly congested and free erythrocytes were seen outside the glomerular capillaries along with periglomerular oedema. In some kidneys the glomeruli were swollen (Fig 64).

There was diffuse tubular degeneration especially in the proximal convoluted tubules. The tubules revealed vacuolization in the epithelial cells and granularity in others. Frozen sections of kidneys stained with oil red 'O' and Sudan black B were negative for fat and the alcohol fixed sections stained with Best's carmine were negative for glycogen. Desquamation of the tubular epithelium and casts were seen in most of the tubules. Periglomerular fibrous tissue proliferation was most pronounced in majority of the animals.

4.6.2.6 Heart. The lesions were similar to that of group I. But the haemorrhages were extensive and mild degenerative changes were also observed. Muscle cells were swollen and contained numerous granules of varying sizes.

4.6.2.7 Lungs. Diffuse congestion and haemorrhages were observed. Alveoli contained eosinophilic homogeneous material. The alveolar walls as well as interlobular septa and pleura were thickened.

4.6.2.8 Lymph nodes. Congestion was a constant finding. Depletion in the germinal centres was seen in 50% of the cases (Fig 65).

4.6.2.9 Spleen. There was marked dilatation of venous sinuses. The endothelial cells lining the sinuses were swollen. Trabeculae were thickened.

The lesions in the digestive system were similar to that seen in group I.

DISCUSSION

CHAPTER V
5. DISCUSSION

In this experiment two dose levels viz. 50 g and 5 g per kg body weight of fresh leaves of *Ipomoea carnea* were used to assess the toxic effects. Similar dose levels were employed by Tirkey *et al* (1987) and Tartour *et al* (1974), respectively. The authors considered these experiments as acute and chronic based on dose levels and not in relation to the development of symptoms and lesions.

There was a rise in the body temperature reaching 103 to 105°F after 10 and 45 days of feeding *I. carnea* leaves respectively, in group I and II. These findings were not in agreement with Tartour *et al* (1974) who observed a temperature of 104.8°F between 6-10 days by feeding *I. carnea* leaves at the rate of 5 g/kg body weight. However, no information regarding rise of temperature was available when *I. carnea* leaves were fed at 50 g/kg body weight. The rise in temperature may probably be attributed to the action of toxin on hypothalamus.

Anorexia, dullness and depression were observed within fortnight in group I fed with *I. carnea* leaves at the rate of 50 g/kg body weight whereas it took 45 days in group II when fed 5 g/kg body weight. This suggested that development of anorexia and dullness was dose dependent. Similar findings were reported in *I. carnea* and *I. fistulosa* toxicity (Tokarnia *et al* 1960 and Adam *et al* 1973).

Progressive weakness and rough coat were noticed in most of the animals in both the experimental groups and this was in agreement with the findings of Tirkey *et al* (1987). Nervous symptoms like ataxia, aimless walking, somnolence, incoordination, awkward posture with stiffness of legs and paralysis of hind limbs with kunckling movements observed could be associated with the lesions in brain, spinal cord and liver. These symptoms were more pronounced

in group II. Similar nervous symptoms were reported by Idris *et al* (1973), Tartour *et al* (1974) and Tirkey *et al* (1987) in *I. carnea* toxicity. These nervous symptoms were also observed in other plant intoxications like *I. muelleri* (Gardiner *et al* 1965), *lolium* toxicity (Culvenor *et al* 1978), *cycad* poisoning (Hooper 1978) and in *lathyrus* poisoning (Innes and Saunders 1958).

In the present study the mortality was noted at different periods of the experiment in both groups which could be attributed to the variation in individual susceptibility of goats to *I. carnea* toxicity. In group I the mortality occurred earlier than group II.

A significant ($P<0.01$) decrease in the body weights was observed in group I and group II compared to control group. This was in concurrence with the observations of Adam *et al* (1973) and Tirkey *et al* (1987) in *I. carnea* toxicity. In group I the body weights decreased at a rate of 336 g/week, while in group II body weights decreased at a rate of 243 g/week. The difference between the group I and II can be attributed to difference in the onset of anorexia.

Significant decrease ($P<0.01$) in RBC, Hb, PCV was observed both in group I and II. The difference in 'b' values of group I and II can probably be attributed to the difference in the dose. In both the experimental groups there was significant decrease in MCV, MCH. So anaemia might be microcytic and hypochromic type.

The observations recorded for WBC study in group I & II denoted gradual development of severe leucopaenia which indicated depression of granulopoiesis in the bone marrow and also due to lymphoid depression of lymph nodes and spleen possibly due to effect of *I. carnea* feeding.

In group I the decrease in erythrocyte count and increase in ESR were significant ($P<0.01$) as evidenced from Table (3). In group II the decrease

in erythrocyte count and Hb levels and increase in ESR were highly significant. These findings were in accordance with Tartour *et al* (1974). The decrease in erythrocyte count and increase in ESR values (Table 3) correlate with each other in that the decrease in erythrocyte count was inversely proportional to the erythrocytic sedementation rate (Coles 1980 and Jain 1986). The enhanced ESR might also be due to decrease in plasma albumin content since albumin inhibits ESR. There was severe leucopaenia in group I and II and such severe leucopaenia was reported by Tartour *et al* (1974). Leucopaenia was usually an accompaniment of haemorrhagic anaemia as observed by Jain (1986).

The total serum proteins and albumins were significantly decreased ($P < 0.01$) in both the experimental groups and it could be traced to the impairment of synthesis of albumins consequent to hepatic damage which gains support from the observation of Kaneko and Cornelius (1971). Significant increase in AST was observed in both groups and this was in agreement with Adam *et al* (1973). The changes in the levels of AST more or less followed similar pattern in both groups (Fig 12). Kaneko and Cornelius (1971) attributed it to the release of enzymes subsequent to the disruption of the hepatic cell walls leading to an increase in the values of AST. The increasing trend observed in the levels of SAP in group II could be attributed to the cell damage as a result of prolonged feeding of *I. carnea* leaves.

At necropsy ascites, hydrothorax and hydropericardium with accumulation of clear straw coloured fluid consistently observed in both the experimental groups. Adam *et al* (1973) reported similar findings in sheep, goats and cattle in *I. carnea* plant toxicity. These findings could be related to hypoprotenemia.

The findings of congestion of the meninges and brain were in agreement with that of Adam *et al* (1973) in goats in *I. carnea* toxicity. Congestion

of the brain was observed in other plant poisoning like *Senecio* (Innes and Saunders 1958) and mushroom poisoning (Minckler 1971).

There was difference in the lesions of brain between the group I and II. In brief the changes of congestion, satellitosis and neuronophagia were seen in cerebrum and oedema and displacement of Purkinje cells in cerebellum of group I and II. In group I congestion and haemorrhages were observed in the cerebrum which were not seen in group II. Serous effusions with loose textured appearance of the white matter of cerebrum seen only in group II. In the cerebellum focal loss of cells in Purkinje cell layer was noticed in group I while there was total loss of cells in Purkinje cell layer in group II.

Congestion and haemorrhages were observed both in the white and grey matter of cerebrum consistently in both the groups, more pronounced in group I. In this case hypoxia as evidenced by anaemia and the toxic effects of *I. carnea* leaves on the thin walled vessels might be responsible for the rupture of vessels and haemorrhages. This gains support from the Bogolepov (1983) and Jubb *et al* (1985) who attributed that the thinness of walls and degenerative changes in the endothelial cells of capillaries and venules to be responsible for hyperaemia and haemorrhages. Minckler (1971) also opined that certain toxins were also capable of selectively affecting the permeability of the capillaries. Mild hyperaemia and small haemorrhages were observed in central nervous system by Dobereiner *et al* (1960) in *I. fistulosa* intoxication. Cerebral haemorrhages were reported in other poisonings like *Oleander* (*Nerium oleander*) leaves and in lead poisoning by Innes and Saunders (1958).

In group II patchy serous effusions were observed mostly in the white matter and to a lesser exent in the grey matter. The white matter was loosely textured and the myelinated fibres were spread apart. Loose textured appearance may be probably due to oedema. The microscopic lesions in cerebrum

were not reported in *I. carnea* poisoning although similar observations were reported by Innes and Saunders (1958) in locoism and lead posioning.

The serous effusions observed in the white matter might be due to hypoxia leading to increased permeability of capillaries and venules which was in concurrence with the observations of Romanova (1956). Jubb *et al* (1985) were of the opinion that the lack of resistance to the passage of fluids from the blood vessels of white matter was the primary cause for oedema.

Polymorphism, shadow state of cells, shrinkage with increased perineuronal space, pyknosis and central chromatolysis and gliosis (astrogliosis) were observed in the pyramidal cells of cerebrum. These changes were the result of direct toxic effects of the *Ipomoea carnea* leaves or indirectly due to hypoxia resulting from anaemia as evidenced by low haemoglobin and PCV values and lowered erythrocyte count. Bogolepov (1983) opined that these changes characteristically result from hypoxia and ischaemic neuronal injury from intoxication. The chromatolysis, shrinkage and disappearance of neurons might be due to poisons, metabolic disorders or diminished blood supply and gains support from the views of Minckler (1971) and Bogolepov (1983). The chromatolysis was an indication of a reactive change as a result of injury. The astrogliosis observed here might have resulted from stimulation by local injury and oedema which was in agreement with the observations of Jubb *et al* (1985).

Most pronounced changes were observed in the cerebellum. Congestion and haemorrhages were observed in cerebellum more commonly in group I and to a lesser extent in the group II. In group I and to some extent in group II displacement of Purkinje cells into the molecular and granular layers was observed with oedema of the Purkinje cell layer. In group II total loss of Purkinje cells was observed with thinned out appearance of molecular and granular layers. These changes observed in the experimental animals could be correlated with the symptoms of ataxia, incoordination and kunckling movements.

The cerebellum, though it receives impulses from cerebral cortex and spinal cord, seems to be responsible for coordination and timing activity of the groups of muscles both in the static and postural movements as propounded by Fulton (1949), Innes and Saunders (1957), Gardner (1968), Everett (1971), William (1971) and Lahunta (1977). Cerebellum regulates these functions either directly or by acting through the spinal cord. Purkinje cells are known to be inhibitory in their function and selectively inhibit the generalized excitary influence of cerebral cortex thereby the posture and movement can be expressed in appropriate and coordinated pattern (Gardner 1968). In the present study damage caused to Purkinje cells by *I. carnea* may be responsible for loss of inhibitory effect over the cerebral cortex thereby resulting in incoordination and ataxia, that are observed during the experimental period. This gains support from the observations of Gardner (1968), Dellmann and Brown (1987) and Blood and Radostits (1989). Perhaps anaemia was responsible for extreme changes in Purkinje cell layer since these cells are highly sensitive to hypoxia. Similar observations were made by Bogolepov (1983) in connection with ischaemic injury.

Literature pertaining to cerebellar lesion in *I. carnea* poisoning could not be traced out. However, similar findings were reported in other plant toxicities like *Senecio* (Innes and Saunders 1958), *Cycad* species (Hooper 1978) and chemical poisoning like mercurial poisoning (Smith *et al* 1972) with symptoms of incoordination and ataxia as was noticed in the present study. In the spinal cord changes were observed mostly in the thoracic and lumbar portions. Lesions like congestion, haemorrahages, chromatolysis, increased perineuronal space and gliosis in experimental groups were in agreement with the findings of Tartour *et al* (1974). In group I most of the neurons were in a shadow state. In group II shrinkage was observed in the white matter with swollen axons. According to Gardner (1968) the impulses enter the spinal cord chiefly by anterior and posterior spino-cerebellar tracts and there after the cerebellum. Accordingly, the function of the part of the cerebellum directed back to spinal cord. In the present study

the changes observed in the spinal cord might be secondary to the changes in cerebellum.

The liver in the group I animals presented a mottled appearance and was slightly enlarged. The gall bladder was distended with thick greenish yellow bile. Unlike the above, the liver in experimental goats of group II revealed congestion, haemorrhages and areas of necrosis and in some animals the liver was pale and soft in consistency. The difference in the appearance and consistency of liver could be attributed to the chronic toxic changes in group II. Enlarged and haemorrhagic liver was reported in goats, sheep and cattle by Adam *et al* (1973) and Idris *et al* (1973) in *I. carnea* toxicity.

In group I hydropic changes in hepatocytes, enlargement of sinusoids and portal vessels were observed microscopically. In group II hyaline droplet degeneration and focal necrosis with polymorphs were observed in addition to congestion and haemorrhages. There was also mild proliferation of the bile ducts. These findings gain support from the observations of Adam *et al* (1973), Tartour *et al* (1974) and Tirkey *et al* (1987) in *I. carnea* intoxication. Gardiner *et al* (1965) made similar observations while discussing *I. muelleri* poisoning in sheep. The observation of engorgement of the vessels along with anaemia might have been responsible for hypoxia resulting in the above changes due to prolonged intoxication in chronic cases as opined by Richardson (1981) in connction with cypermethrin toxicity in goats.

The kidneys were congested in both the experimental groups. On section the medullary region was severely congested and filled with gelatinous fluid which can be related to emaciation exhibited by experimental goats. The cortico-medullary region was dark red with petechiae which was in agreement with Adam *et al* (1973) in goats and sheep in *I. carnea* toxicity. Only pronounced hyperaemia of kidneys was observed in goats fed with *I. asarifolia* by Dobereiner *et al* (1960). The lesions might be due to toxic effects of the plant on the blood

vessels. Microscopically congestion, degenerative changes of tubular epithelium and the presence of albumin casts were observed. Increase in the cellularity of the glomeruli was noticed. These lesions were in concurrence with the observation of Tirkey *et al* (1987) in *I. carnea* toxicity and Gardiner et al (1965) in *I. muelleri* intoxication. The toxic irritant substance brought to the kidney might have exerted direct effect on the tubular epithelium or it might have been due to congestion and anaemia resulting in hypoxia as suggested by Richardson (1981).

Severe engorgement of coronary vessels was seen in both the groups. Pronounced flabbiness of the heart, gelatinization of coronary fat and hydropericardium was uniformly observed in experimental group II. The lesions observed were in close agreement with Adam *et al* (1973). The gelatinization of coronary fat observed grossly, in group II could be related to emaciation and decreased body weights.

In heart microscopic lesions of diffuse haemorrhages, loss of striations, of myocardial fibres, and mild leucocytic infiltrations in both the groups might be due to toxic effects of *I. carnea* on blood vessels and were in accordance with the findings of Tirkey *et al* (1987).

Congestion and oedema of the lungs were observed in both experimental groups. These findings were in concurrence with the findings of Adam *et al* (1973). Microscopically the alveolar capillaries were engorged with focal haemorhages. Some bronchioles and alveoli contained pink stained homogeneous material, while some other alveoli were emphysematous in group I. In addition to the above changes, congestion and diffuse haemorrhages were observed in group II. The microscopic lesions of lungs were not reported by earlier workers. The changes could be attributed to hypoxia or due to direct action of the toxic principle on the blood vessels causing increased vascular permeability or rupture of the vessels resulting in haemoorhages and oedema.

Congestion and desquamation of intestinal mucosa observed in both the experimental groups was in accordance with the findings of Adam *et al* (1973) and perhaps these changes might have caused diarrhoea clinically.

Mild enlargement of spleen was observed in both the groups of animals. The pulp was deep reddish and soft. Soft spleen was observed by Adam *et al* (1973) in *I. carnea* toxicity. Splenic enlargement was observed by Gardiner *et al* (1965) in *I. muelleri* intoxication. Observations like engorged splenic sinusoids, thickened trabeculae and atrophy of white pulp gain support from the reports of Gardiner *et al* (1965) in sheep in *I. muelleri* intoxification.

Enlargement and oedema of the mesenteric and superficial lymph nodes were observed in both groups and it was more pronounced in group II. Histologically mild depletion in the germinal center was observed. The lesions in lymph nodes were not reported by earlier workers.

I. carnea appeared to affect chiefly the CNS that is brain and spinal cord, especially Purkinje cell layer of cerebellum. The toxic lesions appeared to be more pronounced in group II because of prolonged feeding probably due to cumulative effect as compared to group I.

CHAPTER 6

SUMMARY

CHAPTER VI

6. SUMMARY

Eighteen non-descript goats of four to five months age were employed in this study. The animals were divided into three groups, consisting of six animals in each group. Goats of experimental group I were fed with fresh leave of *I. carnea* at the rate of 50 g/kg body weight for 10 weeks, whereas group II were fed with 5 g/kg body weight for 16 weeks. Green fodder and water were provided *ad libitum* to all animals.

Animals were weighed at weekly interval and clinical symptoms were recorded. Blood was collected for haematological and biochemical estimations during the experimental period at weekly intervals. Animals which died during the experiment were necropsied and the rest wre sacrificed at the end of the experimental period along with the controls.

Animals of group I and II manifested the symptoms of toxicity such as dullness, anorexia and depression. Rise of body temperature was observed in both groups. In group II animals, symptoms of alopecia, emaciation and rough hair coat were observed.

Nervous symptoms were observed in both the groups but more pronounced in group II. In group I awkward posture, walking on toes and aimless walking were observed and the animals were in a state of somnolence. Paralysis of the hind limbs and swinging and shaking of the head were observed in group II after 54-60 days. The condition became severe after 90-110 days. These symptoms were correlated with the brain lesions, especially in cerebellum.

Haematological values like RBC count, Hb, PCV and WBC count were significantly ($P < 0.01$) low in both groups. Eryhrocytic sedementation rate was increased significantly ($P < 0.01$) in group II.

Biochemical estimations revealed increase in the values of serum AST and SAP in both the groups. The total serum proteins and albumins were significantly low in experimental groups. Congestion and haemorrhages of all organs were noticed in both the groups. It was severe in group I, whereas in group II the lesions were mild and more chronic in nature as evidenced by connective tissue proliferation in various organs.

Severe congestion of the meningeal blood vessels were observed in both the groups. Neuronal degeneration, satellitosis and neuronophagia were observed in brain. Prominent lesions were seen in the cerebellum. In group I degenerative changes of the Purkinje cells were observed. Whereas in group II total loss of Purkinje cells with thinning of the molecular and granular layers was noticed. In spinal cord congestion and haemorrhages were observed in the group I and demyelinative changes in the group II.

The liver in group I revealed congestion and haemorrhages whereas in group II mild bile duct proliferation with mild fibrosis was seen.

In group I the kidney revealed congestion and haemorrhages whereas changes such as shrinkage of glomeruli and fibrosis around the Bowman's capsule were noticed in group II.

The spleen revealed severe congestion in both the groups in addition the connective tissue in the walls of the sinuses reticulum of the pulp and fibrous trabeculae was thickened in group II.

In lymph nodes severe congestion was observed in both the groups. Mild lymphoid depletion was seen in group II.

Ipomoea carnea appeared to affect chiefly the CNS that is brain and spinal cord especially Purkinje cell layer of cerebellum. The toxic lesions appeared to be more pronounced in group II because of prolonged feeding probably due to cumulative effect as compared to group I.

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