ARTHROSCOPIC PARTIAL SYNOVECTOMY AND JOINT LAVAGE IN THE TREATMENT OF SEPTIC ARTHRITIS IN BOVINES

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CHENNAI – 600 007

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Thesis submitted in partial fulfilment of the requirements for the degree of

Doctor of Philosophy

in

VETERINARY SURGERY AND RADIOLOGY

to the

TAMILNADU VETERINARY AND ANIMAL SCIENCES UNIVERSITY

Chennai- 600 051

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CERTIFICATE

This is to certify that the thesis entitled **'ARTHROSCOPIC PARTIAL SYNOVECTOMY AND JOINT LAVAGE IN THE TREATMENT OF SEPTIC ARTHRITIS IN BOVINES'** submitted in partial fulfillment of the requirements for the award of the degree of **DOCTOR OF PHILOSOPHY** in **VETERINARY SURGERY AND RADIOLOGY** to the Tamilnadu Veterinary and Animal Sciences University, Chennai is a record of bonafide research work carried out by **A.RAMANATHAN,** under my supervision and guidance and that no part of this thesis has been submitted for the award of any other degree, diploma, fellowship or similar titles or prizes and that the work has not been published in part or in full in any scientific or popular journal or magazines.

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ACKNOWLEDGEMENTS

I wish to record my boundless pleasure and sincere expression of heartfelt gratitude, satisfaction and pride to the very polite, dynamic surgeon and gifted Chairman of the Advisory Committee **Dr. R. Suresh Kumar, Ph.D,** Professor and Head, Department of Veterinary Surgery and Radiology, Madras Veterinary College, Chennai, for his expert guidance, constant encouragement, creative suggestions, laudable counseling, tireless efforts, valuable guidance and wholehearted co-operation during the period of research work rendered in spite of his busy schedule.

I am enormously thankful to **Dr. S. Ayyappan, Ph.D.,** Associate Professor, Department of Veterinary Surgery and Radiology, Madras Veterinary College, Chennai and Member of the Advisory Committee for his timely suggestion, excellent photography, constant encouragement, mental support and sharing his experience in arthroscopic surgery.

I extend my heart-felt indebtedness to **Dr. C. Balachandran, Ph.D.** Professor and Head, Department of Veterinary Pathology, Madras Veterinary College, Chennai and Member of the Advisory Committee for the help rendered in the examination of blood, serum and synovial fluid samples and synovial membrane biopsy specimens. I am indebted for his constant inspiration, untiring help, constructive ideas, and immense affection.

It is great pleasure for me to express my sincere thanks and deep sense of gratitude to the Advisory committee member, **Dr. Geetha Ramesh Ph.D.,** Professor and Head, Department of Veterinary Anatomy and Histology, Madras Veterinary College, Chennai, for his valuable suggestions, guidance, constructive advice and keen interest shown in the progress of the work.

I am extremely thankful and grateful to **Dr. B. Justin William, Ph.D.,** Associate Professor, Veterinary University Training and Research Centre, Nagercoil, who inspired me to take up the research work in bovine septic arthritis and for his love showered on me during the study period. He was very supportive and shared his innovative ideas. His unparalleled criticisms moulded my work to its full shape.

I express my sincere thanks to **Dr. L. Nagarajan, Ph.D.,** Professor and Head, Department of Clinics, Veterinary College and Research Institute,

Namakkal, for his valuable suggestion, guidance and the technical support rendered during the period of study.

I am extremely thankful to **Dr. R. Jayaprakash, Ph.D., Dr. Ravi Sundar George, Ph.D., Dr. K. Ramanujam, Ph.D., and Dr. C.Ramani, Ph.D.,** Associate Professors, Department of Veterinary Surgery and Radiology, and **Dr. R. Ganesh, Ph.D.** and **Dr. Capt. G.D. Rao**, Associate Professors Department of clinics, Madras Veterinary College, Chennai, for the advice and encouragement during the study.

I sincerely thank **Dr. Mohamed Shafiuzama, Ph.D.**, and **Dr. Mala Shammi,** Assistant Professors (SS) and **Dr. Arun Prasad** Assistant Professor, Madras Veterinary College, Chennai, for their continuous support, encouragement and excellent photography during the study.

I extend my gratitude to**, Dr. T.N. Ganesh, Ph.D.,** Resident Veterinary Officer, Madras Veterinary College, for his encouragement and support.

I consider myself fortunate to have, **Dr. H. Pushkin Raj, M.V.Sc.,** Senior Research Fellow, Department of Clinics, Madras Veterinary College as a friend and for his untiring help in carrying out every part of the research work and preparation of thesis.

I kindly remember the help and support given by **Dr. Samar Halder,** Assistant professor, West Bengal University of Animal and Fishery Sciences in conducting the arthroscopic surgery.

I am thankful to my colleague **Dr. N. Dhanalakshmi,** M.V.Sc, Assistant Professor, for her unconditional help.

I thank my junior colleagues **Dr. Karthikeyan, Dr. Suresh, Dr. Rajasekar, Dr. Shiju Simon, Dr. Saravanan, Dr. Dhanya, Dr. Smitha, Dr. Akila, Dr. Susila, Dr. Ajith, Dr. Sankar, Dr. Kokila, Dr. Sellamani, Dr. Soja, Dr. Yasotha** who lent their support and help wholeheartedly whenever approached.

I express my indebtedness and gratitude to the **Vice-Chancellor** and the **Registrar, Gandhigram Rural University, Gandhigram** and the **Dean**, Faculty of Agriculture and Animal Husbandry, Gandhigram Rural Institute for granting necessary study leave to take up my Ph.D., programme.

I am highly indebted to my parents for their blessings, encouragement and support during the period of study. I appreciate with love my wife **R. Vijayalakshmi,** who handled all the troubles and inconveniences single handedly with patience during the entire period of work. My dear daughter **R.Sivaranjani** deserves my special thanks for providing me with many inspiring and refreshing moments.

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ABSTRACT

ARTHROSCOPIC PARTIAL SYNOVECTOMY AND JOINT LAVAGE IN THE TREATMENT OF SEPTIC ARTHRITIS IN BOVINES

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Year	**:**	2007

Thirty six clinical cases of bovines with septic arthritis were randomly divided into three groups of 12 animals each and treated by conventional lavage (group I), arthroscopic fibrin debridement and lavage (group II) and arthroscopic debridement, partial synovectomy and lavage (group III). The incidence of septic arthritis was more frequent in crossbred, heifer calves as a sequela to omphalophlebitis. A mean lameness score of 3.58, 3.67 and 3.83 observed on day 1 decreased to 1.83, 1.17 and 1.00 on the 10^{th} day in group I, II and III respectively.

The purulent or turbid synovial fluid observed on day 1 became clear and viscous on day 14 in 23.81, 90.48 and 100 per cent samples of group I, II and III respectively. Significantly increased synovial fluid volume and total protein content recorded on day 1 returned to its normal value on day 21 in group I and on day 14 in both group II and III. The raised synovial fluid alkaline phosphatase activity, serum and synovial fluid glucose difference, total nucleated cell count noted on day 1 returned to their base value on days 28, 21 and 14 in group I, II and III respectively.

Synovial fluid culture was positive in 58.73 per cent of joints and the bacteria isolated were *Streptococcus spp*., *Escherichia coli*, *Pseudomonas spp*., *Staphylococcus spp.* and *Salmonella spp*. A combination of amikacin and penicillin or ampicillin provided broad-spectrum coverage against majority of the bacterial isolates.

Soft tissue swelling, increased joint space, intra-articular gas shadow and subchondral osteolysis were the salient radiographic findings. Hyperaemia, petechial haemorrhage, degeneration/necrosis in synovial membrane, thickening and clubbing of villi, articular cartilage erosion, and fibrin deposits or free floating fibrin clots were the major arthroscopic findings. Fibrin deposits and bacterial colonies in synovial membrane and articular cartilage were the major histological changes.

The outcome was sound, acceptable and unacceptable respectively in 66.67, 14.28 and 19.05 percent joints of group I animals whereas it was sound in 95.24 per cent joints and acceptable in 4.76 percent joints of both groups II and III. The mean time taken for resolution of joint infection was 18.11, 13.33 and 11.13 days in group I, II and III respectively.

It is concluded that arthroscopy permits thorough evaluation, appropriate debridement and effective lavage of septic joints with minimal tissue trauma and offers consistently good results in the management of septic arthritis in bovines than conventional lavage. Partial synovectomy is recommended during arthroscopic debridement of septic joints to eliminate colonizing bacteria and as it helps to shorten the recovery period. Needle lavage is recommended for cases that are presented within 5 to 6 days after infection and arthroscopy for cases presented 7 days or more after onset of clinical signs and those which were refractory to conventional treatment.

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INTRODUCTION

Bovine septic arthritis is a common and devastating clinical problem that accounts for 67.14 percent of all lameness cases in cattle (Singh *et al*., 1989). It is frequently encountered in young calves as neonatal polyarthritis and in older cattle as monoarthritis. It is a crippling disease that results in severe lameness and significant economic loss to the farmer due to reduced milk production, poor draught and reproductive performance, treatment costs, culling and considerable morbidity and mortality in young calves.

Bovine septic arthritis is characterized by accumulation of fibrin due to increased permeability of the synovial membrane. Subsequent deposition of fibrin over the synovial membrane and articular cartilage results in the formation of pannus. The fibrin deposits cover devitalized tissues, interfere with intra-synovial nutrition, aggravate synovial ischaemia, promote synovial adhesion and act as a nidus for bacterial multiplication and a barrier for diffusion of antimicrobial drugs.

Despite the development of new and more potent antimicrobial agents, treatment of septic arthritis represents a challenge to veterinarians, because of the difficulty in eradicating the bacteria that colonized the synovial membrane and trapped in fibrin clot, rapid depletion of proteoglycans from cartilage matrix and subsequent collagen breakdown and worsening of the articular destruction by by-products of infection like fibrin and neutrophil (Bertone *et al.*, 1987a).

The principles of treatment of septic arthritis are elimination of microorganisms, drainage of inflammatory exudate rich in fibrin particles, destructive enzymes and radicals and debridement of infected, severely inflammed and devitalized tissues (Wright *et al*., 2003). Different treatment protocols with variable rates of resolution have been reported.

Parenteral antibiotic therapy could bring about resolution of joint infection in cases diagnosed early as there is increased blood flow and hence transport of antibiotics (Jackson, 1999). However, bovine septic arthritis cases are invariably presented at a later stage with established infection and intra-synovial fibrino-cellular conglomerate (Weaver, 1981). Therefore in addition to antibiotic therapy, drainage and lavage of the infected joint is mandatory for resolution of established infection (Bertone, 1996) as presence of purulent effusion retards the efficacy of many antibiotics by decreasing the metabolic activity of bacteria (McIlwraith,

1987). Joint lavage also removes intra-articular bacteria, tissue debris, inflammatory exudate containing fibrin, neutrophils, proteolytic enzymes and various mediators of inflammation that cause degradation of proteoglycans (Schneider *et al*., 1992b). However, needle lavage is rarely effective when there is pocketing of thick purulent materials and deposition of fibrin in the recesses of the affected joint (Martens *et al*., 1986; Van Huffel *et al*., 1989).

Arthroscopy is considered to provide several advantages over conventional lavage and arthrotomy in the treatment of septic arthritis including improved visualization, identification of infected or devitalized tissues, fibrin deposits as well as access to a larger area of the synovial surfaces for debridement and synovectomy (Wright *et al*., 2003).

Synovectomy is indicated for the treatment of septic arthritis to eliminate the colonizing bacteria and to protect the articular structures from the destructive effects of neutrophils and their enzymes and the free radicals released by the inflammed synovium (McIlwraith, 1983).

Arthroscopic management of septic arthritis has been reported in humans (Jarret *et al*., 1981; Ivey and Clark, 1985; Ohl *et al*., 1991; Jones *et al*., 1994) as well as in horses (McIlwraith, 1983; Bertone *et al.* 1992; Wright *et al*., 2003) and has been associated with excellent results in terms of return to normal joint function.

Literature on the use of arthroscopic surgery in the treatment of bovine septic arthritis is scanty. Hence it requires a systematic research in bovines. The present study was therefore undertaken with following objectives

1. To survey the incidence of septic arthritis in bovines
2. To evaluate the affected joint by clinical, radiographic and arthroscopic examinations
3. To evaluate the synovial fluid characteristics of bovines with septic arthritis
4. To compare the clinical efficacy of arthroscopic partial synovectomy and joint lavage with conventional through-and-through joint lavage in the management of septic arthritis

2. REVIEW OF LITERATURE

The available literature on the subject is recited under the following heads.

2.1 Structure and function of typical synovial joint

An understanding of diarthrodial joint structure and function provides the underpinnings of the veterinarian's ability to prevent, diagnose, treat and prognosticate on joint disease (Todhunter, 1996).

Synovial or diarthrodial joints are closed spaces with a mesenchymal synovial lining that produces and maintains a selective physical, cellular and biochemical environment (Dyce *et al.*, 1996). It consisted of the articular surfaces of at least two bones covered with a thin layer of hyaline cartilage, joint capsule and ligaments, which surround and stabilize the joint (Frandson *et al.*, 2003).

The joint capsule originates from the periosteum at the margins of articular surface and encloses the joint cavity. It consists of an outer tough fibrous layer, the membrana fibrosa and an inner synovial layer, the membrana synovialis. The fibrous layer varies in thickness according to the mechanical demands made upon it and in the majority of the cases it contains the joint ligament (Nickel *et al.*, 1986).

The synovial membrane covers all the inner surfaces of the joint except the surface of the articular cartilage. It is a very thin membrane, barely visible to the naked eye and is richly supplied by blood and lymph vessels and nerves. It contains two mesenchymal cell layers, the subintima and intima. The subintima is adjacent to the fibrous layer and is principally composed of fibrous, areolar or adipose tissue depending on the function of and location within the joint. In joints of high pressure or low motion such as the pastern, the subintima contains relatively high concentrations of fibrous connective tissue and the intima assumes a flattened surface devoid of villi. This is typically seen in areas overlying the tendons and ligaments. In areas of low pressure or high motion, such as the fetlock and carpus, the subintima contains more areolar tissue and has a more folded shape with numerous villi. The intima is a thin (1-4 cells thick), incomplete lining of synoviocytes immediately adjacent to the synovial fluid (Johansson and Rejno, 1976; Henderson and Pettipher, 1985). Dislodged and indurated synovial villi may be found free in the septic joint cavity where they are called free bodies or "joint

mice". Depending on the circumstances and their position, they can give rise to severe pain (Nickel *et al.*, 1986).

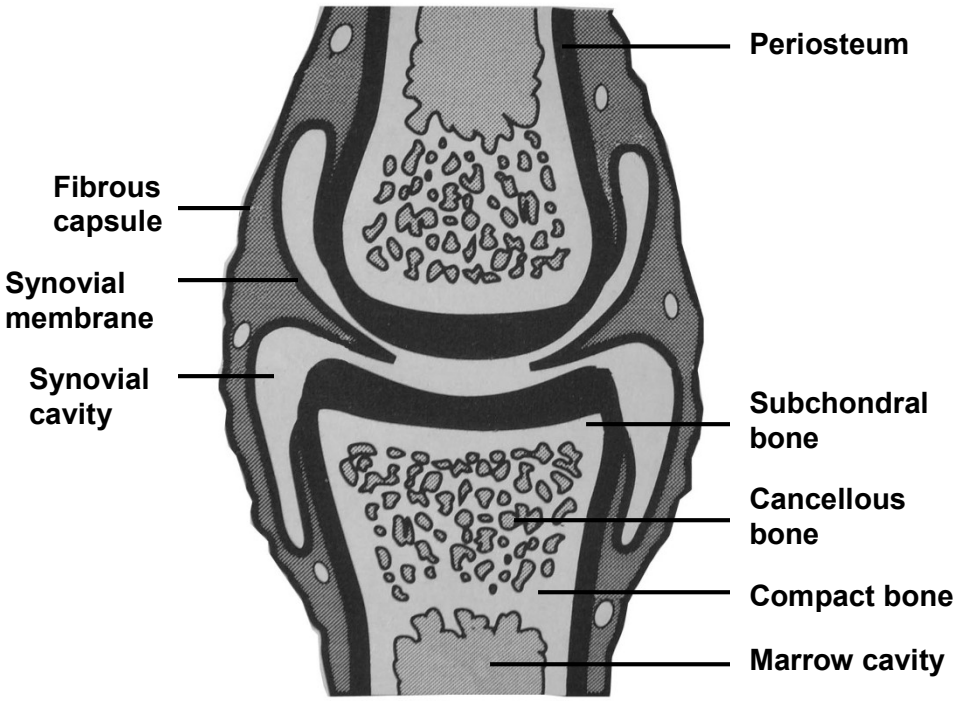

Fig. 1 Structure of a typical synovial joint

The synovial lining consists of two morphologically and functionally distinct cell populations both of bone marrow origin. They are designated as 'A' cells that are more numerous and more superficial and act as macrophage and play a role in immunological and inflammatory response; 'B' cells (fibroblast-like secretary cells, rich in rough endoplasmic reticulum), that produce hyaluronic acid; and 'intermediate' cells, which represent an undifferentiated synoviocytes capable of differentiating to either type under the appropriate stimulus (Greisen et al., 1982).

The function of the synovial membrane is to provide a selective barrier for the ultrafiltration of components from the plasma and thereby determine the synovial fluid composition. The areolar or adipose tissue of the subintima is greatly vascularised to bring blood close to the interior of the joint via a capillary plexus. Blood vessels extend to within 5 to 10 μm of the intimal surface (Todhunter, 1996). Lack of both basement membrane and junctional complexes between the intimal synoviocytes allows normal diffusion of water and small solutes from the capillary fenestrations through the synovial interstitium into the joint to form the synovial fluid. The synovial fluid functions to lubricate and protect the joint and supplies nutrients to the articular cartilage (Doige and Weisbrode, 1995).

The articular cartilage is only a few millimeters thick and is white to blue white in colour. It is attached to the bone by a zone of calcified cartilage. On concave articular surfaces it is thickest at the periphery and centrally on convex surfaces. In ruminants the articular surface may have pit like depressions devoid of cartilage called synovial fossae and are usually bilaterally symmetrical. Articular cartilage has no blood or lymph vessels. The oxygen and nutritive requirements are met by diffusion from three sources: synovial fluid within the joint cavity, vessels in the tissues at the periphery of the cartilage and to a lesser extent from subchondral vessels. Diffusion is assisted by the porosity of the cartilage matrix, which soakes up and releases fluid as the cartilage is alternately unloaded and compressed during movement of the joints (Dyce *et al.*, 2002). Due to its avascular nature, articular cartilage has very limited capacity for repair. At skeletal maturity the deeper regions of the articular cartilage mineralize and remain, as they are not removed by endochondral ossification. The junction between the unmineralized articular cartilage and the deeper mineralized articular cartilage is called the tidemark. The mineralized cartilage serves to anchor articular cartilage to

subchondral bone and limits the diffusion of substances between bone and cartilage (Liebich and Konig, 2004).

Articular cartilage is 70 to 80 per cent water by weight. It is a viscoelastic, hydrated fiber-reinforced gel that contains chondrocytes, type II collagen fibres and proteoglycans aggregates. Collagen fibres provide tensile strength and are arranged in arcades so that the tops of the arcades are parallel to the articular surface and the sides are perpendicular to the surface and parallel with radial layer of chondrocytes. The fibres pass from the underlying bone to the surface, where they bend to lie closely together. Since splitting of cartilage, common in joint diseases, tends to follow the fibre course, superficial lesions lead to tangential flaking, whereas those that extend more deeply create more or less vertical cracks (Dyce et al., 2002). Proteoglycan consists of one or more glycosaminoglycan chains covalently attached to a protein core. By binding water, proteoglycans provide stiffness to resist compression and impede the outflow of water when cartilage is under load (Todhunter, 1996).

The adult hyaline articular cartilage is divided into four distinct histological and biochemical layers; the superficial or tangential layer, the intermediate or transition layer, the radiate or deep layer and the mineralized cartilage layer (Palmer and Bertone, 1994). Functionally the superficial zone of articular cartilage resists shearing forces, the middle zone functions in shock absorption and the base serves to attach articular cartilage to bone. In scanning electron micrographs, the surface of articular cartilage is not smooth but rather has numerous depressions that might serve as reservoirs for synovial fluid (Doige and Weisbrode, 1995).

Articular cartilage functions to minimise friction created by movement, to transmit forces to underlying bone and to maximize the contact area of the joint under load (Liebich and Konig, 2004).

Lubrication of articular cartilage is accompanied by the complementary action of weeping (squeez-film) and boundary lubrication. In weeping lubrication, the loaded articular surface is supplied with pressurized fluid that carries most of the load. Only a small part is carried by cartilage-to-cartilage surface contact, which is lubricated by a glycoprotein-facilitated boundary system. Boundary lubrication implies that a substance sticks to the surface and minimizes contact. Joint stiffness

is often due to inability of periarticular soft tissues to lengthen, rather than to intra-articular friction (Banks, 1993).

2.2 Definition

Arthritis is inflammation of intra-articular structures and could affect any type of joint - fibrous, cartilaginous or synovial (Angus, 1991). The term arthritis implies inflammation of intra-articular structures while the term synovitis is restricted to inflammation of synovium. Arthritis is characterised by the presence of inflammatory cells in the synovium as well as in synovial fluid and the nature of the inflammatory process is often reflected in the volume and character of the joint fluid (Doige and Weisbrode, 1995).

Septic arthritis in cattle implies infection of the joint by pathogenic bacteria (Van Pelt, 1972a). Contamination by bacterial, viral, mycotic or mycoplasmal agents and accumulation of pus in a joint is called as septic arthritis (Weaver, 1981). Septic arthritis is the inoculation of the synovial membrane or synovial fluid with bacteria that incite an inflammatory reaction, which allows the establishment of viable organisms (Bertone, 1996). Septic, infectious or suppurative arthritis is an inflammatory disease confined to the joints as a result of sequestrated bacteria, fungi or viral microorganisms (Orsini, 2002). However bone infection in cattle originated only from bacteria, not fungi. Septic arthritis is the inflammation of the synovial membrane and articular surface as a result of infection (Radostits et al., 2003).

Polyarthritis is the concurrent inflammation of multiple articular joints in an animal (McKean, 1993). It is simply an inflammatory condition involving more than **one joint (Jacques *et al.*, 2002).**

2.3 Classification

Van Pelt et al. (1966) reported that septic arthritis might be primary by direct penetration of a joint, secondary by extension from area adjacent to joint or tertiary from septicemia or metastasis from area remote from a joint.

Firth (1983) classified lesions of septic arthritis and osteomyelitis into

'S' type: Synovitis without the presence of osteomyelitis

‘E’ type: Synovitis with bone infection in the adjacent epiphysis

‘P’ type: Bone infection adjacent to the physis

‘T’ type: Infection of the small tarsal bone

Weaver (1997) classified septic arthritis as primary if it resulted from direct penetration of the joint by a foreign body. Involvement of a joint by extension of cellulitis and necrosis from a decubital lesion was also classified as primary. Arthritis that resulted from the spread of pathogens from an adjacent localized focus was classified as secondary and that which followed systemic haematogenous spread as tertiary.

Verschooten et al. (2000) classified bone and joint infection into four types according to the anatomic localization: type 1 was haematogenous metaphyseal and/or epiphyseal osteomyelitis close to the growth plate (the growth plate mostly secondarily involved), type 2 was haematogenous primary subchondral osteomyelitis often accompanied by septic arthritis and type 3 was haematogenous septic arthritis with secondary subchondral osteomyelitis implicating that infection in the subchondral bone originated from the joint. Type 4 was outlined as osteomyelitis, when the bone lesions could not be classified into one of the previous groups.

2.4 Incidence of septic arthritis

Van Pelt (1968) after a study of 23 joint affections in cattle stated that osteoarthritis was the most frequently encountered condition with more frequent involvement of tibio-tarsal joint (78%).

Baggot and Russel (1977) reported that 88 per cent of the lesions causing lameness in cattle were in feet and 12 per cent were in the upper limb. Among the upper limb lesions, affections of the joint and ligaments accounted for 47 per cent. Among the joint affections, arthritis accounted for 27 per cent of the cases.

Weaver (1981) stated that septic arthritis was frequently encountered in young calves as “joint-ill” or “neonatal polyarthritis” and dairy calves were more commonly affected than beef calves but it was less commonly found in older cattle and usually as monoarthritis. Tarsal, stifle and carpal joints were most commonly affected in calves whereas the most common location of septic arthritis in adult cattle was the distal interphalangeal joint and occasionally the carpal or tarsal joint.

Russel *et al.* (1982) analysed the causes of lameness in dairy cattle and observed that 88.3 per cent of lesions causing lameness were in feet and the remaining 11.7 per cent in the upper limbs. Among the limb lesions the involvement of joints and ligaments were 47 per cent.

Merkens *et al.* (1984) reviewed the medical records of 123 cattle with septic arthritis of the distal intertarsal and tarsometatarsal joints and reported that the incidence was neither age-dependent nor with any breed predilection. Various breeds of cattle ranging from 1 to 12 years of age were represented in that study, but the incidence was highest in the age category of 2-7 years (the period of maximum milk yield).

Wehr *et al.* (1984) recorded septic arthritis of carpal and tarsal joints in 25 to 35 per cent of the calves housed in calf rearing premises.

Ducharme *et al.* (1985) reported that 24 per cent of the stifle disorders in cattle were caused by septic arthritis.

Mclenman (1988) surveyed the incidence of lameness in dairy cattle and observed that 83.2 per cent of lesions leading to lameness were in feet and 16.8 per cent of the lesions were in the limbs. In the limb lesions, joints and ligaments were involved in 72.2 per cent cases.

Singh *et al.* (1989) carried out a radiographic survey of bone and joint affections (excluding fracture and dislocations) in 104 cattle and recorded joint affections in 67.31 per cent cases. Joint affections were most commonly recorded in male (90.14%) than females (9.86%) but both limbs were equally affected in both the sexes. Arthritis accounted for 67.14 per cent of all joint affections.

Van Huffel *et al.* (1989) stated that infectious arthritis of the radio-carpal joint was a common cause of fore limb lameness in cattle.

Schneider *et al.* (1992a) reviewed the medical records of 192 horses affected with septic arthritis and reported that tarsocrural joint was most commonly affected (34%) followed by the fetlock (20%), carpus (18%) and stifle (9%) joints.

Baragi and Levin (1993) reported that arthritis accounted for 13.8 per cent of all lameness cases in cattle.

Desrochers *et al.* (1995) in a clinical study of 12 cases with septic arthritis of the distal interphalangeal joint reported that the affected cattle were from 3

months to 6 years old (mean 3.2 years) and body weight ranged from 75 to 1045 kg (mean 587kg). Lateral digit was affected in eight cattle (4 forelimbs and 4 hind limbs) and the medial digit was affected in four cattle (4 forelimbs).

Butson *et al*. (1996) treated intra-synovial infection in 12 horses and 10 cattle with gentamicin-impregnated polymethylmethacrylate beads and reported that the incidence was more frequent in females (80%) than males (10%).

Kofler (1996) recorded higher incidence of septic arthritis (84%) than traumatic arthritis. Carpal (34.61%) and tarsocrural joints (30.77%) were most commonly affected by septic arthritis followed by metatarsophalangeal (19.23%), metacarpophalangeal (7.69%), elbow (3.85%) and stifle (3.85%) joints. Males were most commonly affected (71.43%) than females (28.57%). Five different breeds were represented in that study viz. Simmental (71.44%), Holstein-Friesian (9.52%), Brown Swiss (9.52%), Charolais (4.76%) and Angush (4.76%).

Jackson (1999) stated that the joints most commonly affected by septic arthritis in calves were the carpus, hock and stifle.

St.-Jean (1999) reported that joint-ill was frequently encountered in calves born in unclean environment and in calves that were deprived of colostrums during the first few hours of life.

Rohde et al. (2000) reviewed the medical records of 130 cases of cattle with infectious / non-infectious arthritis and reported that septic arthritis was more frequently observed in the stifle (34.92%) followed by metacarpo-phalangeal joint (22.22%), tarsus (20.63%), carpus (13.49%), elbow (3.97%), shoulder (3.17%) and inter-phalangeal joint (1.60%). The incidence was more in calves below one year of age (70.49%) than adults (29.51%). Similarly dairy cattle were most commonly affected (64.62%) than beef cattle (35.38%). The dairy breeds included Holstein Friesian (98.80%) and Brown Swiss (1.20%).00 The beef breeds represented were Aberdeen Angus (26.09%), Hereford (10.87%), Simmental (8.69%), Limousin (10.87%), Charolais (4.35%) others (13.04%) and mixed breeds (26.09%). The incidence was more frequent in females (68.46%) than males (31.54%).

Verschooten *et al.* (2000) reviewed the clinical files and radiographs of 445 cattle with bone infection in the appendicular skeleton with or without septic

arthritis and reported that bone and joint infection was frequent in cattle less than three months of age (44.4%). Infection was evenly distributed between three and 12 months of age, increased between 12 and 24 months of age and decreased again in animals older than two years of age. Apparent differences existed between the age classes and the type of bone infection. Type 1 infection occurred mostly in bovines between one and two years of age. Types 2 and 3 occurred primarily in cattle between 0 and three months of age. Type 4 infection occurred mainly in the very young (< 3 months) or in animals of 12 to 24 months of age and older.

2.5 Etiology

2.5.1 Predisposing factors

Partial or complete failure of passive transfer of immunoglobulins from dam's colostrum (Van Pelt, 1969), delayed receipt of colostrum (Platt, 1977) and decreased acidity in the stomach of newborn (Rooney, 1962) were some of the factors that increased the incidence of septic arthritis.

Gustafson *et al.* (1989) reported that corticosteroids administered locally into the joint suppressed the immune status of the joint and thus potentiated the ability of a sub-infective dose of bacteria to establish infection.

The principal potentiating factors for establishing joint infection were immunological compromise particularly in young animals (Firth, 1983; Martens *et al.*, 1986), the presence of foreign materials (Reginato *et al.*, 1990) and / or devitalized tissues and the nature and number of contaminating organisms (Bertone, 1996).

Weaver (1997) recorded higher incidence of joint-ill in calves born in unhygienic environment, where immediate postnatal disinfection of the umbilicus was not practiced and where calves were immune compromised owing to inadequate intake of colostrum in the first few hours of life. The author also observed that contamination of abdominal skin of the umbilical region occurred more frequently in small weak calves that remained recumbent for some hours after birth than calves that were ambulatory within few minutes.

Berry (1998) reported that the risk factors for the development of haematogenous neonatal joint infection included poor farm management, failure of passive transfer and presence of unclean environment at the time of parturition.

Rohde *et al.* (2000) reported that the large and high motion joints were more commonly affected by infectious arthritis than smaller low motion joints. The authors opined that larger joints were more susceptible to inflammatory reaction because of higher mechanical activity and larger synovial membrane surface area.

Stashak (2002) reported that hypogammaglobulinemia, from either genetic defect or failure of passive transfer, systemic sepsis and trauma were the risk factors for bone or joint infection.

2.5.2 Exciting causes

Van Pelt and Langham (1966) reported that the portals of entry that provided a source of infection leading to the haematogenous conveyance of microorganisms to the joint cavity in calves were omphalophlebitis, intrauterine infection of the fetus, gastrointestinal infection, osteomyelitis, infection of the periarticular decubital ulcer and infection of wound (e.g. castration and docking).

Van Pelt *et al.* (1966) opined that direct penetration of fetlock joint and subsequent infection due to pyogenic organisms was the main cause of infectious arthritis in adult cattle.

Van Pelt (1973) noted a tendency of selective localization of pyogenic bacteria in diarthrodial joint especially the tarsal joint as a sequela to metritis and mastitis in adult cattle and omphalophlebitis in calves.

Alexander (1978) reported that septic arthritis developed following penetrating joint injury.

Bailey (1985) stated that septic arthritis might be associated with enteritis, pneumonia, uterine infection or umbilical infection with bacteraemia or septicemia.

McIlwraith (1987) reported that iatrogenic form of septic arthritis might result from arthrocentesis or following intra-articular steroid injection.

Schneider *et al.* (1992a) reviewed the medical records of 192 horses affected with septic arthritis and reported that common causes of joint infections were haematogenous localization (32%), articular wounds (24%), inoculation during intra-articular injection (22%), postsurgical infection (13%), idiopathic causes (6%) and others (3%).

Rebhun (1995) reported that septic arthritis in young calves originated from umbilical infections or septicemia whereas older calves (over 3 weeks) and heifers developed septic arthritis following exogenous wounds, periarticular cellulitis, punctures or endogenous circulation of pathogens from the intestinal or respiratory tract. Adult cattle developed septic arthritis secondary to endogenous diseases such as endocarditis, septic mastitis, pneumonia, lung abscess, liver abscess, chronic foot infections and from exogenous infections secondary to traumatic wounds, decubital sores and periarticular cellulitis or abscessation.

Joint infection might result from the introduction of microorganisms through open wounds or self-sealing punctures, via haematogenous spread, local extension of perisynovial infection or iatrogenically (Bertone, 1996).

Orsini (1996) opined that infectious arthritis and osteomyelitis were frequently sequelae to bacteraemia and septicemia, trauma, local extension from adjacent infected tissues or they might be iatrogenic.

Weaver (1997) stated that septic arthritis in adult cattle occurred usually due to direct penetration of a joint by a wood, metal stake, barbed wire, corrugated iron or haematogenous spread from suppurative mastitis, chronic abscessation, septic metritis, septic endocarditis or myocarditis and occasionally due to nonsterile procedure such as arthrocentesis.

Berry (1998) reported that neonatal septic arthritis was usually associated with haematogenous spread of bacteria from an infected umbilicus, pneumonia or enteritis.

In neonates cartilage is vascular (Whalen et al., 1998) and trauma to the developing cartilage with associated haemorrhage and exposure of the bacterial binding sites might be the inciting cause for the location of infection in the joints (Santschi, 2004).

Verschooten *et al.* (2000) reviewed the clinical files and radiographs of 445 cattle with bone infection in the appendicular skeleton with or without septic arthritis and reported that the etiological factors associated with bone/joint infection was determined only in 23 per cent of the cattle. Trauma (28%), congenital articular rigidity (25%) and umbilical infection (21%) were most commonly identified

followed by tail necrosis (9%), respiratory disorders (8%), diarrhoea (4%) and abscess (4%).

Valla and House (2002) reported that joint infection in neonatal calves usually occurred by haematogenous spread and the primary source of infection were enteritis, pneumonia and inflammed umbilical structures. The authors further stated that failure of passive transfer of immunoglobulins increased the risk of sepsis.

Desrochers (2004) stated that although direct trauma to the joint was the principal cause of septic arthritis in adult cattle, neglected periarticular wound infection might extend into the joint at a later time. Distal joints being less protected by soft tissues were more susceptible to infection from external trauma.

2.6 Bacteriology of septic arthritis

Firth *et al*. (1980) reported that negative culture result from purulent synovial fluid might be due to poor collection, storage and laboratory techniques, prior administration of antibiotics or partial success of the immune system in combating the joint infection.

Weaver (1981) reported that the most common organisms cultured from septic arthritis in cattle were *Actinomyces pyogenes*, *Escherichia coli*, *Staphylococcus aureus*, *Streptococcus spp.* and *Salmonella spp*.

Orsini (1984) reported that the microorganisms frequently implicated in septic arthritis and osteomyelitis were *Actinobacillus spp*., *Streptococcus spp.*, *Salmonella spp*. and *E. coli* and the less common organisms included *S. aureus*, *Enterobacter cloacae*, *Klebsiella spp*., *Pseudomonas spp.* and *Bacteroides spp*.

Martens *et al.* (1986) stated that examination of synovial fluid smears stained with Gram's stain might yield presumptive results more rapidly than would bacteriologic culture of the fluid. Bacterial agents commonly associated with septic arthritis / osteomyelitis in the horse were *Salmonella spp., E.coli* and *Actinobacillus equuli*. *Corynebacterium equi*, *Streptococcus spp.*, *S. aureus*, *Klebsiella spp.* and *Bacteroides spp.* have also been isolated from affected joints.

Montgomery *et al.* (1989) asserted that a positive bacterial culture of synovial fluid was not a necessity for the diagnosis of bacterial infective arthritis in

dogs as synovial fluid from experimentally infected joints had also produced negative results.

Moore *et al*. (1992) reported that isolation of a bacterial organism from synovial fluid did not indicate it as the cause of infection bacteria as it could also be secondry contamination.

Common agents causing infective arthritis in cattle were *Streptococcus spp.*, *Staphylococcus spp.*, *E. coli*, *Proteus spp.*, *Salmonella spp.* and *Corynebacterium spp.* (Jubb *et al.,* 1993)

St-Jean (1993) reported that *Streptococcus spp.* and *E. coli* were the most commonly isolated organisms from the synovial fluid of young calves affected with septic arthritis, followed by *A. pyogenes*, *Salmonella spp.*, *Mycoplasma bovis* and *Hemophilus somnus*.

Kofler (1996) performed arthrocentesis in thirty joints of cattle with arthritis and reported that no samples could be aspirated from five joints and that out of the 25 samples of synovial fluid bacteria were isolated only from 16 joints. The author further reported that several samples had mixed infection and the bacteria isolated were *A. pyogenes*, hemolytic *Streptococci* and *E. coli*.

Streptococcus spp. was the most common bacterial isolate from neonatal bovine polyarthritis followed by *E. coli*, *A. pyogenes*, and *Staphylococcus spp.*, whereas in adult cattle it was *A. pyogenes* followed by *Streptococci* and *Staphylococci* (Weaver, 1997).

Jackson (1999) reported that *A. pyogenes* and *E. coli* were the most common causal agents of septic arthritis in calves and the less common organisms included *Salmonella*, *Staphylococci* and *Streptococci*.

Steel *et al.* (1999) studied 93 cases of foals with septic arthritis and reported that failure to observe organisms in synovial fluid smear and / or failure to isolate organisms on culture of synovial fluid did not exclude a bacterial cause.

Valla and House (2002) reported that the pathogens most commonly isolated from septic joints of neonatal calves were enteric organisms including *E.coli* and *Salmonella spp.. Streptococcus spp., Staphylococcus spp.,* and *Arcanobacterium pyogenes* were the less common isolates. The authors further stated that *A. pyogenes* was more frequently isolated in older calves.

The most common organisms cultured from the synovial fluid of septic arthritic cattle were *S. aureus*, *E.coli*, *Pseudomonas aeruginosa* that accounted for more than half of the isolates obtained (Radostits *et al.,* 2003).

Gupta *et al*. (2003) reported that between 12 and 43 percent of human cases of clinically infected joints arthritis did not yield positive results on culture of synovial fluid.

Desrochers (2004) reported that the most common bacterial organisms isolated from septic arthritis in adult cattle were *Arcanobacterium pyogenes*, *E. coli* and other environmental bacteria. The author further opined that Mycoplasma and *H. somnus* should be considered as possible causes when the incidence of septic joints increased in a herd without umbilical involvement.

2.7 Pathogenesis

A clear understanding of the pathogenesis of the arthritis will ensure that affected animals are treated at the earliest possible stage, so that lameness did not become irreversible (Angus, 1991).

Van Pelt and Langham (1968) studied the synovial fluid changes produced by infectious arthritis in cattle and reported that synovial inflammation increased the vascular permeability and allowed influx of large molecular protein into synovial space and thus resulted in higher total protein concentration in synovial fluid.

Fournier *et al*. (1969) reported that the increase in synovial fluid total protein content following joint infection increased the volume of fluid by osmotic pressure and acted as a barrier to fluid resorption.

Johnson *et al.* (1970) induced septic arthritis by intra-articular injection of *Staphylococcus aureus* into the rabbit knee and observed severe lameness, permanent osteo-arthritis and higher mortality in untreated control group.

Lazarus *et al.* (1981) reported that in the initial clinical stages of acute inflammation latent forms of the non-specific mediators of inflammation such as kinin, histamine, complement and components of the blood coagulation and fibrinolytic systems, including plasminogen and trypsin entered the joint cavity due to mechanical disruption of the blood-synovial barrier. Polymorphonuclear leukocytes and monocytes which infiltrated the joint in response to the non-specific mediators were responsible for the release of stored latent and active mediators and

for the production of oxygen derived free radicals. Stimulation of macrophages, synoviocytes and chondrocytes by leukocytes derived products resulted in the upregulation and modulation of mediators such as the cytokines, eicosanoids and matrix enzymes. A balance of these mediators with the presence of naturally occurring inhibitors of these mediators then determined the extent, duration and reversibility of the cartilage and synovial membrane response. Permanent damage to the joint surface could be prevented by rapid therapeutic intervention to completely halt the inflammatory reaction.

Copious outpouring of exudate rich in fibrin and blood clotting mechanisms and shedding of polymorphonuclear (PMN) leukocytes into the synovial fluid to produce purulence followed joint infection. Lysosomal and other enzymes were released from PMN cells following phagocytosis of bacteria and their subsequent breakdown. These enzymes attacked and broken down the cartilaginous matrix and left the collagen fibrils without support. The fibrils were later fragmented mechanically by the wear and tear of the joint motion. Replacement of the normal synovial fluid with effusate, suppuration and fibrin interfered with nutrient and cell waste transport mechanisms and made the cartilage more vulnerable to injury. Fibrin deposition further impaired the entrance of nutritients into the cartilage from synovial fluid and impeded the release of metabolites from the cartilage. Fibrin itself chemotactically attracted leukocytes to the joint. Phagocytosis of this fibrin and other particulate matter by the leukocytes resulted in leukocytes degeneration and release of more lysosomal enzymes. When the healing reaction was initiated, pannus composed of granulation tissue and myriads of blood vessels was formed. Spreading of the pannus over the articular surface caused further lysis by its penetration and undermining of the cartilage plate (Brown and Newton, 1985).

Coles (1986) stated that release of prostaglandin E_2 from macrophage or synovial fibroblast following joint infection promoted fever, pain and inflammation.

Martens *et al.* (1986) reported that prostaglandin and superoxide radicals that are released from inflammed synovial tissues caused erythema, edema and pain.

Bertone *et al.* (1987a) reported that fibrin particles accumulated in the synovial membrane surface and intra-articular space of septic joints impeded nutritional exchange for articular cartilage, aggravated synovial ischemia, provided medium for protected growth of bacteria and also promoted synovial adhesion.

Tulamo *et al.* (1989) reported that joint infection produced rapid decline in articular cartilage proteoglycans content due to degradation and / or decreased synthesis. The initial loss of compressive stiffness due to degradation or decreased synthesis of proteoglycans perpetuated the loss of collagen tensile strength due to amplification of the biomechanical vector forces and direct destruction of collagen by collagenase; however grossly visible articular cartilage degradation was not apparent in most clinical cases of infectious arthritis. The authors also demonstrated reduction in the molecular size of synovial hyaluronan and reduction in viscosity and boundary lubrication normally provided by that molecule.

Angus (1991) reported that the fibrous joint capsules were tough but flexible, had poor blood supply and consequently did not readily become inflammed, nor were they readily penetrated by bacteria unless punctured during traumatic injury. By contrast synovial membranes were highly vascular and sensitive to inflammatory stimuli. Unlike vascular endothelium, the inner lining of synovium was not continuous and the constituent cells lie in a fibrillar matrix in intimate contact with a rich vascular and lymphatic plexus. The membrane is thus highly permeable. This explained the ease with which blood-borne infection gained access into the joint cavity. In inflammatory states the villi became swollen with serous exudate and inflammatory cells, which together with increased tension in the joint capsule caused by accumulation of exudate in the joint cavity, lead to pain and lameness. The synovial villi became more exaggerated after a few days because of an influx of lymphoid cells and antibody-secreting plasma cells.

The inflammatory reaction of the joint was characterized by hypertrophy and hyperplasia of cells lining the synovia and a marked immune response that resulted in the accumulation of fibrin, neutrophils and exudate containing the mediators of inflammation that finally lead to irreversible damage to the articular cartilage (Schneider *et al.*, 1992b).

Hardy *et al.* (1993) conducted *in vitro* studies on equine synovial membrane to evaluate the effect of bacterial infection on hyaluronate synthesis and reported that 10 g of normal equine synovial membrane was capable of neutralizing 100 colony-forming units (cfu) of pathogenic *Staphylococcus aureus* and the infection developed if the number of bacteria overwhelmed the synovium, the organism was more virulent or the synovium was compromised.

The acute inflammatory response seen in most cases of infectious arthritis was the result of host's recognizing the organism as foreign and initiating a rapid influx of inflammatory cells, mostly neutrophils, to eliminate the infection (Bertone and McIlwraith, 1987). The neutrophil is a rapidly mobile cell that could kill bacteria both intracellularly and with extracellular enzymes that could be released or are released upon death of the neutrophils. Destructive enzymes included lysosyme, elastase, cathepsin G, gelatinase and collagenase (Palmer and Bertone, 1994).

Bertone (1996) reported that synovitis caused larger molecules like fibrinogen from plasma to enter synovial fluid, which might be deposited over the synovial membrane and articular cartilage resulting in the formation of pannus. The pannus interfered with intra-synovial nutrition and acted as a nidus for bacterial multiplication and a barrier for antibacterial drugs.

Caron (1996) reported that pain and lameness associated with septic arthritis were due to stimulation of low thresh-hold type II mechanoreceptors located within the joint capsule. The receptors were inactive when joints were immobile and got activated when the joint underwent movement or experienced tension due to stretching of the joint capsule by effusion.

Hardy *et al.* (1996) studied the effect of increased intra-articular pressure on equine synovial membrane by coloured microspheres and reported that joint effusion due to infectious arthritis significantly reduced the blood flow to synovial membrane, fibrous layer of joint capsule and physis. The authors observed a positive correlation between degree of joint effusion and severity of joint pain. The authors also reported that ischemia to subchondral bone and articular soft tissues further damaged the joint and decreased its ability to fight infection.

Weaver (1997) reported that fibrin clots were the major gross feature of the inflammatory exudate of septic arthritis in cattle. These fibrin clots deposited on both the cartilage and synovial villi disrupted their function and contributed to permanent damage and maintenance of the inflammatory cycle. The author further stated that removal of such fibrin was an important step in therapy of joint inflammation.

Berry (1998) reported that following bacteraemia, septic emboli were lodged initially in the metaphyseal capillary loops at the physis and in the developing subchondral bone of the articular surfaces of long and cuboidal bones. The factors that contributed to bacterial colonization in these regions included i) high capillary density, ii) neovascularisation at the sites undergoing endochondral ossification and iii) rapid changes in the blood flow from arterial to sluggish flow within the sinusoids.

Madigan and House (2002) reported that following bacteraemia, a nidus of infection developed in a junction of cartilage and bone. The low pressure / slow flow and reduced oxygen pressure of the blood supply at cartilage-bone junctions predisposed to establishment of infection in these areas.

McIlwraith (2002) reported that established joint infection frequently resulted in the formation of an intrasynovial fibrinocellular conglomerate (pannus) which might cover foreign material and devitalized tissues thus interfering with their nutrition from synovial fluid and delaying their healing process.

Orsini (2002) reported that fibrin clots were the principal component of the inflammatory exudate in bovine septic arthritis. Fibrin deposited in the synovium contributed substantially to permanent joint damage and maintained a vicious cycle of inflammation. Intra-articular fibrin disrupted normal cartilage function by adhering to articular cartilage and altering the flow of nutrients to cartilage and metabolic by-products from cartilage. Leucocytes were chemotactically attracted to the fibrin where they got self-destructed and released enzymes into the synovial fluid. Joint damage could be minimized if fibrin deposition was reduced or fibrin accumulation was efficiently removed.

Wright *et al.* (2003) reported that pannus acted as nidus for bacterial multiplication and was rich in inflammatory cells, degradative enzymes and radicals and hence removal of fibrin deposit was essential to prevent further damage to articular cartilage and complete elimination of infection.

Desrochers (2004) enumerated the factors responsible for articular damage in septic arthritis as follows:

1. Bacteria directly damaged cartilage as well as synovial membrane and fluid.
2. Elastase, cathepsin, gelatinase and collagenase enzymes from neutrophils destroyed cartilage as well as bacteria.
3. Free radicals from neutrophils and synoviocytes of inflammed synovial membrane degraded the articular cartilage.
4. Matrix metallo proteinase released from chondrocytes in response to synovitis decreased the proteoglycan production and thus decreased mechanical strength of articular cartilage.

May (2005) stated that synovitis that started within 24 hours of contamination promoted the extravasation of polymorphonuclear leukocyte into the joint space and the proteolytic enzymes released by them during phagocytosis of bacteria contributed to the breakdown of articular cartilage. The author also reported that the proteolytic activity was also associated with chondrocytes and leucocytes in the pannus.

2.8 Clinical signs

Simmons and Johnston (1963) reported that the common physico-clinical signs of infectious arthritis were lameness, pain and enlarged and tender joints.

Van Pelt and Langham (1966) observed increased synovial effusion, variable degrees of hyper-arthrothermia and hyperarthresthesia in calves with septic polyarthritis. The authors reported that arthralgia was manifested by various degrees of claudication, a reluctance to ambulate and lateral recumbency in calves with severe systemic infection.

Van Pelt et al. (1966) reported that calves affected with infectious arthritis were reluctant to move and bear weight and stood keeping the affected limb partially flexed. The authors also observed persistent arthrogryposis and hyper-arthrothermia in severely affected joints.

Van Pelt (1970) reported that enlargement of joints in calves with septic arthritis was due to distension of joint capsule, peri-articular edema and fibrosis.

Van Pelt (1972b) observed visible distension of the tibiotarsal joint capsule (and to a lesser extent of the proximal intertarsal joint capsule), periarticular edema, limited range of joint motion, partial flexion of the limb when standing and

lameness of the affected hind limb on ambulation and increased cutaneous temperature in comparison to contralateral and ipsilateral joints in six Holstein-Friesian cattle with septic tarsitis.

Pratap et al. (1977) inoculated Staphylococcuc aureus, Corynebacterium pyogenes and E.coli cultures into the knee and hock joints of two buffalo calves each and reported that symptoms of arthropathy were apparent within 48 to 72 hours of inoculation. The authors observed a non-significant increase in rectal temperature, pulse and respiratory rates up to 7th post-inoculation day.

Ryan et al. (1983) observed fever, joint swelling and lameness following intra-articular inoculation of 1-2 × 109 Mycoplasma bovis into the carpal and tarsal joints of six calves. Five of the six calves had lameness of such severity that they become recumbent, while the sixth calf was never recumbent and had full ambulatory function by six weeks post inoculation.

Merkens et al. (1984) analysed the medical records of 123 cattle with septic arthritis of the distal intertarsal and tarsometatarsal joints and reported that majority of the cattle showed severe non-weight bearing lameness, swelling on the dorsomedial aspect of the hock over the distal intertarsal and tarsometatarsal joints, elevated skin temperature over the affected area, pain on application of pressure at the dorsomedial aspect of the distal part of the hock and extensive swelling of the subcutaneous tissue in most cases. Swelling of the tibiotarsal joint was not observed.

Ndikuwera et al. (1989) observed non-weight bearing lameness, markedly swollen stifle joints, severe gluteal muscle wasting, hypersensitivity and pain on manipulation and fibrotic and thickened joint capsule in five heifers with idiopathic septic gonitis. The authors also observed stretching of the skin over the joint due to subcutaneous edema and recorded significantly high skin temperature over the affected joint in comparison to the unaffected joints.

Rebhun (1995) reported that signs of septic arthritis included marked lameness with distension of the affected joint capsule, warmth over the joint and painful response when the joint was manipulated, flexed or extended.

Bertone (1996) recorded classic cardinal signs of inflammation centered around the joint viz. heat, swelling (edema and joint effusion), pain on palpation and flexion, erythema of surrounding skin and severe lameness in horses with septic arthritis. The author further noted that the lameness was frequently non-weight bearing.

Weaver (1997) reported that calves with septic arthritis exhibited sudden onset of severe lameness, swelling of major weight bearing joints due to effusion and periarticular edema, joint heat on palpation and pain on forced flexion and extension due to stimulation of nerve endings in the fibrous layer of joint capsule. The author further stated that calves with acute polyarthritis also exhibited general systemic signs like fever, lassitude, reduced appetite, weight loss and increased periods of recumbency.

Riley and Farrow (1998) observed marked in calf rectal temperature, pulse rate and respiratory rate in a calf with arthritis of the carpus and osteomyelitis of the carpal and metacarpal bones that was refractory to parenteral antibiotic treatment..

Madigan and House (2002) reported that clinical signs of septic arthritis in neonatal calves varied extremely from sudden onset of lameness in one leg in an apparent healthy neonate with or without joint distension to sudden onset of severe lameness with obvious systemic signs of illness or evidence of multiple joint distension, pain and edema. Lameness was often blamed by the owner on the dam's stepping on the newborn. The authors further stated that any neonate less than 45 days of age with sudden onset of lameness should be considered infected until proven otherwise.

Orsini (2002) reported that localizing signs in septic arthritis were lameness, heat, pain, swelling and redness involving the periarticular soft tissues accompanied by a synovial effusion. The author further stated that the lameness progressed to non weight bearing in the advanced stage of the disease.

Stashak (2002) reported that clinical signs of infective arthritis in horses included severe lameness, swelling, effusion with thickening, edema and pain on manipulation. The authors also noted that periarticular swelling was noticed in few traumatic entities, which was typical of sepsis.

The common clinical findings in septic arthritis included lameness, joint heat, swelling and pain. The degree of distension varied depending on the type of infection. In some cases the pyogenic bacteria caused the greatest degree of swelling that resulted in rupture of the joint capsule. Fever, inappetence to anorexia, endotoxemia, loss of body weight and discomfort were observed in animals with only one severely affected joint or when several joints were less severely affected. Physical impairment of joint movement was observed in chronic cases because of fibrous thickening of the joint capsule, periarticular ossification and rarely ankylosis of the joints. Crepitus was detected in joints in which much erosion has occurred (Radostits et al., 2003).

2.8.1 Grading of lameness

Assessment of the severity of the lameness was helpful to classify lameness and monitor responses to treatment (Anderson and Desrochers, 2004).

The various grading systems described to categorize the degree of lameness severity in dairy cattle are given below:

Scoring system	Scores / grades	Observations
Manson and Leaver (1988)	1.0	Minimal abduction/adduction, no unevenness of gait, no tenderness
	1.5	Slight abduction/adduction, no unevenness or tenderness
	2.0	Abduction/adduction present, uneven gait, perhaps tender
	2.5	Abduction/adduction present, uneven gait, tenderness of feet
	3.0	Slight lameness, not affecting behaviour
	3.5	Obvious lameness, some difficulty in turning, not affecting behaviour
	4.0	Obvious lameness, difficulty in turning, behaviour pattern affected
	4.5	Difficulty in rising, difficulty in walking, behaviour pattern affected
	5.0	Extreme difficulty in rising, difficulty in walking
Tranter and Morris	0	No abnormality of gait
	1	Lameness hardly noticeable
	2	Slightly lame
	3	Markedly lame
	4	Affected limb not weight bearing

Scoring system	Scores / grades	Observations
Wells *et al.* (1991)	0	None: Gait abnormality not visible at walk; not reluctant to walk
	1	Mild: Mild variation from normal gait; includes intermittent mild gait asymmetry of mild bilateral restriction in free movement.
	2	Moderate: Moderate and consistent gait asymmetry or symmetric gait abnormality but able to walk
	3	Severe: Marked gait asymmetry or severe symmetric abnormality
	4	Non-ambulatory: Recumbent
Greenough *et al.* (1997)	1	Normal gait – not lame
	2	Slight abnormal gait: stiff uneven tender gait
	3	Slight lameness: moderate and consistent lameness
	4	Obvious lameness: obvious lameness affecting behaviour, still weight bearing
	5	Severe lameness: non-weight bearing
Sprecher *et al.* (1997)	1	Normal: stands and walks normally, flat back to pelvis
	2	Mildly lame: stand with flat back top line; arches back during ambulation; slightly abnormal gait
	3	Moderately lame: stands and walks with arched back top line; shortened phase of stride
	4	Lame: Arched back top line when standing and walking; obvious diminished weight bearing in one or more limb(s).
	5	Severely lame: constantly arched back, difficulty in walking
Whay (2002)	1	Sound
	2	Imperfect locomotion
	3	Mild lameness
	4	Moderate lameness
	5	Severe lameness
	6	As lame as possible while upright
	0	Normal gait
	1	Mild: walks easily, readily; bears full weight on foot and limb but has an observable gait alteration; stands on all four limbs; line of backbone normal.

Scoring system	Scores / grades	Observations
Anderson and Desrochers (2004)	2	Moderate: reluctant to walk and bear weight but does use the limb to ambulate; short weight bearing phase of stride; rest the affected limb when standing; increased period of recumbency; arching of back bone.
	3	Severe: reluctant to stand; refuses to walk without stimulus; non-weight bearing on affected limb; 'hoops' over the limb rather than bear weight; does not use limb when standing and lies down most of the time; backbone arched with caudo-ventral tip to pelvis.
	4	Catastrophe: recumbent; unable to rise; euthanasia often indicated.

2.9 Haemogram

Van Pelt et al. (1966) reported that mean proportion of segmented neutrophil was significantly increased while the mean proportion of lymphocyte was significantly less in the blood of arthritic calves. The authors also observed a significant reduction in the mean hemoglobin value.

Kohli (1968) conducted clinical and experimental investigations on surgical infections and sepsis in cattle and reported that leucocytosis with shift to left was commonly observed in systemic infection with febrile inflammatory reaction. The author further stated that these changes were not due to direct depression of the bone marrow activity by bacterial infection but were the result of indirect effect through the nervous system.

Ryan et al. (1983) recorded a significantly elevated white blood cell and neutrophil counts following experimentally induced septic arthritis with Mycoplasma bovis in six calves. Erythrocyte counts, packed cell volumes and haemoglobin concentrations were at the lower end of the normal range.

Valli (1985) reported that in acute inflammatory conditions the body produced increased fibrinogen as well as proteins that converted serum iron into ferritin. The process continuous, and as long as the inflammatory reaction persisted, serum iron was maintained at low level, even though the body iron stores might be

normal or increased. Under these circumstances iron might be a limiting factor for erythropoiesis.

Rodak (1995) stated that the inflammatory reaction caused by severe localized infection reduced availability of iron in the monocyte/macrophage system and thus resulted in reduced total erythrocyte count, hemoglobin content and packed cell volume.

Weiser (2004) reported that leukocytosis with shift to left was commonly observed in acute septic focal inflammation with increased blood flow and swelling.

2.10 Synovial fluid analysis

2.10.1 Properties of normal synovial fluid

Krishnamurthy and Tyagi (1977) studied the composition of synovial fluid from the normal stifle joints of 70 bullocks, 17 cows and 25 she buffaloes and observed that the synovial fluid was colourless and clear in all the animals. The mean total protein content (g/100 ml) was 2.0 ± 0.18, 2.2 ± 0.17 and 2.0 ± 0.18 and the mean glucose content (mg/100 ml) was 122.4 ± 4.24, 123 ± 9.59 and 129 ± 5.21 respectively in bullocks, cows and buffaloes.

Koch (1979) reported that normal equine fluid had 800 WBC / µl (8% PMN) whereas septic synovial fluid had 50,000 WBC / µl (80% PMN).

Perman (1980) reported that the primary cell types seen on a normal synovial fluid smear were mononuclears (65 to 90%) with predomonence of monocytes and macrophages while the lymphocytes and synovial lining cells were less common. Polymorphonuclear cells were rarely seen in synovial fluid (≤10%) and signified blood contamination when found.

Normal synovial fluid was a clear, colourless to straw coloured and did not clot as it was devoid of fibrinogen and other plasma clotting factors. Instead it became gel like on standing but returned to liquid state on gentle shaking, a property called thixotrophy (Weaver, 1981).

2.10.2 Synovial fluid changes in septic arthritis

Van Pelt (1971) observed a marked increase in synovial fluid alkaline phosphatase activity in horses with monoarticular idiopathic septic arthritis and reported that it might be due to depolymerization of mucin by bacterial enzyme.

Van Pelt (1972a) analysed synovial fluid from six Holstein-Friesian dairy cattle with septic arthritis and reported that the fluid was pale yellow to amber, turbid to opaque with flocculent material and mucin precipitate quality varied from fair to very poor. The mean total leucocyte count (1000/cumm) was 38.67 ± 17.17 and per cent neutrophils was 85 ± 10.1.

Miller *et al*. (1974) reported that synovial fluid from septic joints showed poor quality mucin clot due to lysosomal enzymes released from degenerating neutrophils whereas traumatic or degenerative osteoarthropathies often showed good mucin clot.

Van Pelt (1974) stated that the increase in total protein content of synovial fluid following septic arthritis could be attributed to influx of larger molecular proteins mainly globulins (viz. alpha2 and gamma) from plasma into the synovial space due to increased vascular permeability. Reduced quantity of synovial fluid hyaluronic acid content or depolymerization or both, that occurred following joint infection also facilitated passage and retention of proteins of increasingly higher molecular weight.

Cohen et al. (1975) considered a serum synovial fluid glucose difference of 15 mg/dl in humans to indicate joint sepsis.

Krey and Bailen (1977) reported that increase in serum and synovial fluid glucose difference had been a diagnostic aid in infectious arthritis in humans

Anderson and Liberg (1980) studied the blood serum and synovial fluid protein composition in bovine laminitis and arthritis and observed a

marked increase in globulin content leading to depression of albumin : globulin ratio.

McIlwraith (1980) stated that marked synovitis following severe joint infection reduced the production and polymerization of the hyaluronic acid moiety in synovial fluid and resulted in poor to very poor mucin clot quality.

Perman (1980) reported that neutrophils were the predominant cell type seen on septic synovial fluid smear. In addition, synovial lining cells (synoviocytes), chondrocytes, pieces of cartilage, osteoblasts and osteoclasts might be seen in the fluid from joints in which there was exposed bone.

Orsini (1984) reported that a WBC count of 30,000 to 1,00,000 per μl, a polymorph proportion more than 75 per cent and a glucose level of less than 25mg per 100ml of synovial fluid were definitive indication of septic arthritis in large animals.

Goldenberg and Cohen (1985) opined that any purulent joint effusion should be considered septic unless proved otherwise.

Lipowitz (1985) reported that the synovial fluid glucose level in dog was equal to or is slightly lower than that of blood. Therefore for diagnostic purpose the serum glucose level should simultaneously be estimated and compared with synovial fluid glucose level.

Martens *et al.* (1986) reported that synovial fluid from an infected joint was turbid, amber to bloody and had low viscosity.

Bennet and Taylor (1988) reported that persistence of abnormal synovial fluid characteristics (following treatment) such as high total nucleated cell count (>25, 000 cells/mm^3) and more than 80 per cent neutrophils indicated persistent joint inflammation, continued infection and therefore failure of treatment.

Chawla *et al.* (1989a) induced septic arthritis by injecting ruminal fluid (group A) and *E. coli* (group B) in four calves each to study changes in biochemical constituents of synovial fluid and reported that the synovial fluid became turbid, yellow and opaque with varying amounts of flocculent materials following septic

arthritis. The total protein content of the synovial fluid increased significantly on the 3^{d} post-induction day and remained significantly high upto 45th post-induction day. The mean ± S.E. values of total protein (g / L) were 25.42 ± 3.05, 26.85 ± 1.93 and 34.22 ± 2.37 in group A and 21.24 ± 2.37, 26.73 ± 4.81 and 22.76 ± 2.18 in group B calves respectively on 3rd , 7th and 45th post-induction days.

Ndikuwera *et al.* (1989) analysed synovial fluid from five Holstein-Friesian heifers with septic gonitis and reported that the synovial fluid appeared pale, cloudy and turbid in all the cases and it rapidly clotted within the syringe in three cases. The total protein content (g/litre) of the synovial fluid recorded in the five cases were 34, 70, 71, 46 and 71. No organisms were isolated from the fluids but cytological analysis showed large number of neutrophils and biochemical analysis revealed increased activities of aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase and lactate dehydrogenase that were suggestive of septic arthritis.

Tulamo *et al.* (1989) studied sequential changes synovial fluid parameters following experimental joint infection in horses and reported that accumulation of cells and fibrin imparted the turbidity and opacity to synovial fluid.

Madison *et al.* (1991) opined that inflammed joints with purulent synovial fluid must be presumed to be infected and treated as such, even if bacteria could not be isolated.

Howard (1993) considered a total nucleated cell count exceeding 30×10^{9} cells per litre of synovial fluid to indicate sepsis.

Moulvi *et al.* (1993) stated that normal synovial fluid was colourless or straw coloured, clear and free from flocculent material whereas pale yellow or dark yellow and turbid samples with flocculation were usually seen in septic arthritis.

St.-Jean (1993) reported that synovial fluid changes in septic arthritis included an increased volume of fluid, a yellow to brown discolouration, presence of flocculent materials with lack of viscosity, protein content greater than 2 g / 100 ml and white blood cell count greater than 30,000 cells per µl with 90 % or more neutrophils.

Singh and Tayal (1993) reported that the percent polymorphonuclear cell count was significantly increased and the mononuclear cell count was concomitantly decreased in septic arthritic cattle.

Bennet and May (1995) reported that the synovial fluid of infected joints in dog often contained degenerate neutrophils, which differentiated it from other inflammatory arthropathies.

Kumar and Singh (1995) studied the synovial fluid changes in buffalo calves with *Staphylococcus aureus* induced septic arthritis of the radio-carpal joint and reported that increase in permeability of the synovial membrane due to dilatation of blood vessels allowed larger molecules from the circulation to enter the joint cavity and thus resulted in significant increase in volume, total protein and total leucocyte count and a significant decrease in pH, relative viscosity and mucin precipitate quality. The authors also observed a significant decrease in glucose level and increase in alkaline phosphatase level. The mean ± S.E. values of pH, total leucocyte count (1000/cumm), alkaline phosphatase (IU/L), glucose (mg/dL) and total protein (g/dL) on the 7^{th} day after induction were 5.61 ± 0.81, 28.64 ± 3.98, 482.00 ± 85.73, 10.14 ± 1.77 and 5.04 ± 1.25 respectively.

Bertone (1996) reported that clotting of synovial fluid immediately after collection indicated its contamination with blood or an increase in non-mucin protein due to abnormal vascular permeability or degeneration of synovial membrane.

Crabil *et al.* (1996) reported that negative culture results of purulent synovial fluid from inflammed joint might be due to prior administration of antimicrobial agents or the insensitivity of the culture medium or because of temporary absence of bacteria or their presence in insufficient number.

Trotter and McIlwraith (1996) reported that the quality of the synovial fluid mucin clot varied with severity of synovitis. Severe the inflammation, more the effusion and the worst the mucin clot quality.

Greenough *et al*. (1997) considered synovial fluid with total nucleated cell count exceeding 50×10^9 cells per litre or a total protein concentration exceeding 40 g per litre to be suggestive of sepsis and fluid with total nucleated cell count exceeding 100×10^9 cells per litre to be septic.

Jackson (1999) reported that synovial fluid containing more than 60 g of protein per litre, more than 10,000 cells per µl and polymorph proportion greater than 90 per cent indicated septic arthritis.

Rohde *et al.* (2000) analysed synovial fluid from 130 cattle admitted for lameness and concluded that total nucleated cell count greater than 25,000 cells per µl or polymorphonuclear cell count greater than 20,000 cells per µl, a neutrophil

proportion greater than 80 per cent and total protein content greater than 45 g per litre indicated septic arthritis.

Orsini (2002) reported that joint fluid cytology indicating inflammation i.e. a nucleated cell count in excess of 10,000 cells / µl with more than 80 per cent neutrophils and a total protein content more than 40 g/L suggested infection without positive culture or histopathologic findings to substantiate bacterial invasion.

The author reported the characteristics of synovial fluid from normal and septic joints as follows:

Characteristic	Normal	Septic
1. Colour	Clear	Yellow / green
2. Clarity	Transparent	Turbid
3. Fluid volume	Low	Increased
4. Viscosity	High	Low
5. WBC / µl	< 200	30,000 – 1, 00,000
6. PMN (%)	< 25	> 75
7. Glucose	Equal to blood	< 25 mg /dl
8. Grams staining	No bacteria seen	Bacteria observed frequently

Francoz *et al.* (2005) induced septic arthritis in the right tarsus of seven healthy Holstein bull calves by inoculating 10 colony-forming units of viable *Escherichia coli* to evaluate the effect of joint lavage and antibiotic treatment on cytologic and bacteriologic variables of synovial fluid and stated that clinical signs of septic arthritis appeared on day 2 and persisted until day 9 in all the calves. Bacterial cultures from all calves were positive for *E. coli* on day 2, and remained positive until day 3 for 1 calf and until day 4 for five calves. In addition, PCR results were positive for all calves, with six positive through day 3 and 1 positive through day 4, after which a positive result was again obtained on day 24. Synovial fluid neutrophil counts and white blood cell counts were significantly increased on days 2-4; however, synovial total protein concentrations were significantly increased ($P<0.05$) throughout the experiment in comparison to day 1. Results of all bacterial cultures were negative on day 8, although clinico-pathologic signs of inflammation persisted until day 20.

May (2005) reported that the gross changes seen in the synovial fluid in association with bacterial infective arthritis were increased volume, turbid or blood tinged fluid, poor viscosity and clotting on exposure to air due to high fibrinogen content. The cytological changes typically seen in synovial fluid smears were markedly increased numbers of white blood cells, mostly neutrophils (more than 5 to 10 neutrophils per high power field) and toxic neutrophils with pyknotic nuclei or ruptured or degenerated neutrophils.

2.11 Intra-articular changes following septic arthritis

McIlwraith and Fessler (1978) evaluated the changes in synovial membrane following experimentally induced synovitis in the mid-carpal joints of 12 ponies by arthroscopy and reported that slender, polyp-like and filamentous villi were commonly observed in the dorsomedial and dorsolateral areas of the normal mid-carpal joints whereas membranous, fan like and cauliflower like villi with hyperemia and petechiation were seen in joints with synovitis. The authors also observed abnormal development of small hyperemic villi in the medial cul-de-sac, which was normally smooth, white, and without villi. Chronic fibrotic changes were also observed; villi tended to become thicker and denser as the disease progressed. The proportion of filamentous villi also was definitely decreased in joints with synovitis. Two other unusual findings in the inflamed joints that were seen infrequently were fusion of the villi into bridges across parts of the joint and presence of fibrinous strands across the joint.

McIlwraith (1983) reported that articular cartilage degenerated following septic arthritis because of compromised nutrition attributable to incorporation of synovial fluid in fibrin clots.

Bertone et al. (1987b) reported that release of cellular enzymes, plasmin and prostaglandins following joint sepsis led to depletion of proteoglycans (< 5 days) from the cartilage matrix, alteration in cartilage pliability and subsequent collagen breakdown from increased vulnerability to mechanical forces.

Ndikuwera et al. (1989) observed extrusion of cauliflower-like fibrinous growth following arthrotomy in five heifers with septic gonitis. The other gross

changes recorded were massive deposition of fibrin all over the articular surface, roughened condylar surface and pockets of pus around the joint and thickened joint capsule.

2.12 Radiographic changes in septic arthritis

Weaver (1981) reported that radiographic findings might be negative in early stages of septic arthritis but in later stages soft tissue swelling, distension of joint capsule, widening (or) narrowing of joint space and still in later stages subchondral bone destruction, exostosis, intracapsular and extracapsular periosteitis and osteomyelitic changes were observed. But radiography did not allow localization and exact determination of the character of the soft tissue swelling.

Martens *et al.* (1986) stated that septic arthritis/osteomyelitis of haematogenous origin was characterized radiographically by various degrees of osteolysis involving the metaphysis, physis and/or ephysis; areas of reactive cortical bone and/or soft tissue swelling.

Lekharu *et al.* (1988) conducted experimental study on buffalo calves to observe the radiographic changes in infectious arthritis and reported that moderate to intense soft tissue swelling, increased joint space and osteolytic changes were observed on 15^{th} day of infection. However, the intensity of these changes was more on 30^{th} and 45^{th} day of infection.

Butler *et al.* (1993) reported that radiographic features of joint infection included

- Periarticular soft tissue swelling
- Joint capsule distension with or without apparent widening of the joint space
- Irregularity of outline of the subchondral bone
- Lucent zones in the subchondral bone with or without sclerosis
- Periarticular osteophyte formation due to secondary joint disease and
- Partial collapse of the subchondral bone.

Desrochers *et al.* (1995) reviewed the radiographs of 12 cattle with septic arthritis of distal interphalangeal joint and reported that soft tissue swelling around the coronary band, widening of the distal interphalangeal

joint space, subchondral bone lysis and osteomyelitis of the middle and distal phalanges, periosteal reaction and new bone formation on the middle and distal phalanges were the common radiographic features. The authors concluded that severity of radiographic findings varied depending on chronicity of the condition.

Kofler (1996) examined the radiographs of 25 cattle with traumatic and septic arthritis and observed soft tissue swelling of the affected joint region in all cases and in 30 per cent of the cases it was the only finding. The author noted increased intra-articular space in 36.7 per cent of the cases and subchondral osteolysis, new periosteal bone proliferation and osteomyelitis or relative displacement of the articular bones in 33.3 per cent of the cases.

Desrochers *et al.* (1997) studied the anatomic communications among the antebrachiocarpal, middle carpal, and carpometacarpal joints in cattle, using intra-articular latex, positive-contrast arthrography, and fluoroscopy and reported that there is no communication between radio-carpal and mid-carpal compartments in 96 per cent of the bovine carpal joints and in the remaining four percent of the cases, the radio-carpal compartment communicated with the mid-carpal compartment between the ulnar and intermediate carpal bone.

Weaver (1997) recorded only soft tissue swelling on radiographic examination of calves with septic arthritis in its early stages (up to seven days after infection). Some days later the changes included widening of the joint space and lack of definition of joint surface / blurring of normal bone outline due to subchondral osteolysis and occasionally gas formation within the joint space. Intracapsular and extracapsular periosteal reaction, subchondral sclerosis, exostosis formation, osteomyelitis and bone destruction were noticed in small limb bones (carpals and tarsals) in advanced stages of septic arthritis.

Hirsbrunner and Steiner (1998) observed soft tissue swelling on the cranio-medial aspect of the carpus and discrete zones of irregular radiolucency on the medio-distal aspect of the radius and on the os carpi radialis on radiographic examination of the carpal region of a Holstein Friesian cow with septic arthritis of 15 days duration. The authors also observed severe soft tissue swelling on the lateral aspect of left carpus and irregular bone architecture of the styloid process of

the ulna and proximal aspect of os carpi ulnare in another heifer with septic carpitis of 5 days duration.

Madigan and House (2002) reported that the radiographic features of septic arthritis included soft tissue swelling, widening or collapse of the joint space, osteoporosis and osteosclerosis. Repeat radiographs taken at seven days interval were valuable for assessing the degree of damage. But the degree of damage was often not detectable until 10 to 14 days after the occurrence of infection.

Desrochers (2004) opined that in calves with septic arthritis radiographic lesions had revealed a tendency to be more lytic, with less new bone formation in comparison to older animals.

May (2005) stated that radiological features of bacterial infective arthritis varied with the type and duration of infection and soft tissue swelling in and around the joint was the only evidence in early stages of infection. Marked periarticular periosteal bone reaction was observed one to two weeks after the onset of infection. Mineralisation of the periarticular soft tissue, subchondral bone erosion, irregular areas of subchondral bone sclerosis and reduction in width of the joint space were the common radiographic features in chronic cases. Occasionally subluxation was observed due to ligament damage in more chronic cases.

2.13 Ultrasonography

Munroe and Cauvin (1994) used ultrasonography for the diagnosis of septic arthritis in bovines and reported that normal synovial fluid was anechoic and appeared black on the sonogram whereas cloudy appearance was associated with presence of pus.

Kofler (1996) carried out ultrasonographic examination of 30 joints of cattle with septic or traumatic arthritis and concluded that diagnostic ultrasonography outlined the accurate location of soft tissue swelling, extent and consistence of joint effusion and involvement of concurrent periarticular synovial cavities and other soft tissue structures.

2.14 Pathology

Van Pelt and Langham (1966) studied the pathological changes in calves with non specific polyarthritis due to systemic infection and observed mild to severe hyperaemia of the synovial membrane with varying degrees of villous hyperplasia and hypertrophy and degenerative changes in the articular cartilage as evidenced by loss of their smooth hyaline appearance and by minor erosion. The

authors also observed pannus formation at the perichondral margin of the articular cartilages in some of the joints examined.

Bertone *et al.* (1987a) observed neutrophilic and lymphocytic-plasmacytic infiltration, vascular dilatation, surface fibrin accumulation, edema, villous hyper trophy and granulation tissue in the synovium of equines with experimentally induced infectious arthritis.

Angus (1991) observed swollen joints containing up to 20 ml of grayish or greenish pus, congested and velvety synovia and erosion or ulceration in articular cartilages at necropsy in lambs that died of suppurative polyarthritis.

2.15 Histological changes in synovial membrane

Van Pelt (1972a) reported that the inflammatory reaction in the joint was characterized by hypertrophy and hyperplasia of synovial lining cells, round cell infiltration of the synovium in the vicinity of blood vessels, pannus formation and degradation of articular cartilage.

Johansson and Rejno (1976) studied the features of normal and acutely inflamed synovial membrane, under light and electron microscopy and observed increased vascularisation, oedema, inflammatory cell infiltration, increase in synoviocyte number and villous hypertrophy in inflammed synovial membrane as compared with normal synovial membrane. The authors also observed electron microscopic changes like increased rough endoplasmic reticulum cisternae, an increase in the number of lysosomes and gradual dilatation of the cisternae to form vacuoles which were suggestive of increased protein synthesis and phagocytic activity.

Ndikuwera *et al.* (1989) observed mature collagen and granulation tissue, multi branched synovial villi with large epithelial cells, florid inflammatory reaction characterized by the presence of micro abscess, aggregates of neutrophils and mononuclear cells in synovial membrane sections from five heifers with septic gonitis. Formation of lymphoid follicles was also observed in several places.

Anderson *et al.* (1993) studied the effects of intra-articular chlorhexidine diacetate lavage on the stifle in healthy dogs. The authors collected synovial membrane biopsy specimens following lavage and fixed the specimens in neutral-buffered 10 per cent formalin for no longer than three weeks. After embedding the specimens in paraffin, 5 μm sections were prepared and stained with H & E.

2.16 Complications of septic arthritis

Angus (1991) reported that a complication of acute septic arthritis was the development of granulation tissue (pannus) across the articular surface. The lytic action of the pannus on the collagen together with collagenase from neutrophils caused widespread erosion of the articular surfaces. Finally organisation of the inflammed tissues together with development of bony spicules (osteophytes) in the joints greatly diminished mobility and resulted in permanent stiffness.

Rebhun (1995) reported that calves with polyarthritis remained recumbent for long time and developed secondary flexor tendon contracture.

Desrochers (2004) recorded post treatment complications like ankylosis of the joint, muscle atrophy and tendon deformities in calves with chronic septic arthritis even after control of infection.

2.17 Prognosis

Verschooten *et al.* (1974) treated 81 cases of cattle with septic arthritis in cattle by two methods and reported a 72 per cent success rate in cases treated by surgical debridement and systemic antibiotic therapy and 43 per cent recovery rate with systemic and intra-articular antimicrobials treatment.

Merkens et al. (1984) analysed the medical records of 123 cattle with septic arthritis of the distal intertarsal and tarsometatarsal joints and reported that majority of the cases (76 out of 123) had a grave prognosis and were immediately sent for slaughter for economic reasons. The remaining 47 cases treated with antibiotics (23), radiation (14) and arthrotomy (10) had a recovery rate of 48, 79 and 70 per cent respectively.

Rebhun (1995) reported that prognosis of septic arthritis should always be guarded but it was better for acute than chronic cases, better for single joints than multiple ones, better when primary etiology was corrected and better when pathogen was identified by culture.

Madigan and House (2002) reported that duration, extent of bone involvement and degree of damage affected the prognosis. Infection of multiple joints, delay in onset of treatment and presence of concurrent bony lesions on radiograph were associated with poor prognosis.

Radostits et al. (2003) reported that the prognosis in case of advanced septic arthritis was poor. Neglected animal might die or had to be destroyed because of open joints or pressure sores. Chronic arthritis and subsequent ankylosis greatly impeded locomotion and interfered with usefulness of the animal.

May (2005) reported that the prognosis for bacterial infective arthritis should always be guarded. Factors that should be taken into account in prognostication included

- Prompt diagnosis and appropriate treatment was critical to a good prognosis
- Chronic infection, rapidly destructive infections or any infection with severe joint destruction all merited a guarded to poor prognosis.
- Systemic infections such as bacterial endocarditis carried a very poor prognosis when there was joint involvement.
- A guarded prognosis was justified in immature animals because of the additional risk of secondary physeal damage.

2.18 Treatment

Brown and Newton (1985) stated that the fundamental goals in the treatment of septic arthritis were

- To clean the joints in order to avoid articular cartilage destruction and adhesion formation.
- To decompress the joint in the immature patient to avoid vascular embarrassment to the epiphysis.
- To administer adequate doses of antibiotics to eradicate the joint infection and prevent secondary spread.

The goals of therapy in acute septic arthritic cases were to prevent irreversible cartilage damage and extension of the infectious process to adjacent tissues. In chronic cases in which severe and irreversible tissue damage had already occurred, the aims were to stop further damage and facilitate pain-free and functional usage of the affected limb (Trent and Plumb, 1991).

Madigan and House (2002) reported that the aims of treatment of septic arthritis were to

- Remove the infectious agent
- Protect and minimize cartilage damage
- Minimize secondary osteoarthrosis

Wright *et al.* (2003) reported that the objective of treating synovial sepsis included removal of foreign material, debridement of contaminated/infected and devitalized tissues, elimination of micro-organisms, removal of destructive enzymes

and radicals, promotion of tissue healing and restoration of normal synovial environment.

2.18.1 Parenteral antibiotics

Schmid (1966) reported that joint infection with Corynebacteria often responded rapidly to parenteral antibiotic treatment alone, whereas the Streptococcal, Staphylococcal and less frequently encountered Proteus spp. and Pseudomonas spp. infections might resist parenteral antibiotic therapy completely.

Oddgeirson (1981) conducted a pharmacokinetic study to assess the efficacy of antibacterial drugs administered through intra-muscular, intra-venous and intra-articular injection in arthritic cattle and reported that therapeutic effective concentration in joint was achieved in 10 minutes after administration of two grams of ampicillin intravenously and joint infection was cured by intra-articular or intravenous injection.

Hau et al. (1983) reported that bacteria trapped in the fibrin clots were partially protected from effects of systemically administered antibiotics. Hence systemic antibacterial treatment alone was often insufficient to eliminate joint infections, particularly in the sub-acute or chronic stages.

McIlwraith and Turner (1987) reported that the presence of purulent effusion retarded the action of many antibiotics by decreasing the metabolic rate of bacteria. The activity of aminoglycosides in particular was significantly reduced due to lowering of the pH value of the synovial fluid due to septic effusion.

Chawla et al. (1989b) conducted experimental studies on calves to compare the various treatment schedules for septic arthritis and concluded that antibiotics given by systemic route along with local intra venous route resulted in earlier resolution of infection.

Lloyd et al. (1990) reported that the therapeutic gentamicin concentration greater than minimum inhibitory concentration (MIC) for susceptible organisms had been achieved in synovial fluid for two to six hours after single IV or IM administration. The authors further stated that the gentamicin concentration in synovial fluid is dependent on the entry of the drug into the synovial fluid after systemic administration which may be retarded by thickening of synovial membrane in chronic septic arthritis cases.

Rosin (1990) reported that while selecting antibiotics for treating joint infection, the most frequently isolated organisms and their susceptibility in that locality should be taken into consideration.

Hegewald and Murphy (1991) recommended combination of synergistically acting antibiotics such as amikacin and penicillin in the treatment of septic arthritis.

Schneider *et al.* (1992a) treated 192 horses affected with septic arthritis and reported that amikacin was highly effective against *Staphylococci*, *Enterobacteriaceae* and *Pseudomonas*; gentamicin against *Staphylococcus*, *Salmonella*, *Pseudomonas* and *Actinobacillus*; penicillin and ampicillin were very effective against β-haemolytic *Streptococci* and cephalothin was very effective against most gram-positive organisms particularly *Staphylococcus*. The authors concluded that the most effective combination of antimicrobials for the treatment of infectious arthritis before culture results were cephalothin and amikacin or gentamicin plus penicillin.

Weaver (1997) recommended procaine penicillin G 22,000 I.U. per kg i.m. bid; ampicillin trihydrate 20 mg per kg i.m. bid; ceftiofur 1 mg per kg i.m, oid; oxytetracycline hydrochloride 11 mg per kg i.v. bid or trimethoprim / sulpha 30 mg per kg i.v. bid for the treatment of septic arthritis in calves. The author further reported that antibiotic therapy should be continued for 10 to 30 days even if an initial improvement was noted.

Jackson (1999) reported that parenteral antibiotic therapy could bring about complete resolution of joint infection and a return to normal function of the joint in cases diagnosed early as there was increased blood flow and hence the transport of antibiotics.

Radostits *et al.* (2003) reported that failure of parenteral antimicrobial therapy to resolve joint infection could be due to

1. Inadequate concentrations of antimicrobials achieved in the joint cavity.
2. Presence of excessive amount of exudate and fibrin in the joint making the infectious agent inaccessible to the antimicrobials.
3. Drug-resistant infection.
4. The development of rheumatoid like arthritis that was chronic and progressive.

2.18.2 Intra-articular antibiotics

Pratap *et al.* (1975) induced septic arthritis with potent cultures *of S. aureus*, *C. pyogenes* and *E. coli* in four buffalo calves each to study the efficacy of antibiotic-steroid intra-articular therapy and reported that calves with *S. aureus* or *E. coli* joint infection responded readily to intra-articular therapy with hydrocortisone (25 mg) and oxytetracycline (200 mg) whereas those with *C. pyogenes* infection did not respond.

Lloyd *et al.* (1988) estimated the concentration of gentamicin in plasma and synovial fluid periodically for 24 hours after intra venous injection at the rate of 2.2 mg per kg body weight and intra-articular administration of 150 mg into the carpal joint in 9 horses each and reported that the peak mean synovial fluid concentration after intra-articular administration was significantly ($p<0.05$) higher than (1,828 μg/ml) than that after intravenous administration (2.53 μg/ml). The mean half-life of gentamicin in the synovial fluid after intra-articular administration (259 minutes) was 2-8 times longer than that in the plasma after intravenous administration (92 minutes). The authors further reported that the mean synovial fluid concentration of gentamicin remained well above the minimal inhibitory concentration of gentamicin for many common bacterial pathogens for 24 hours after intra-articular administration. Thus systemic administration of multiple doses per day was not required for the selective treatment of a septic joint. The authors concluded that intra-articular administration had the advantages of increased treatment interval, reduced total dosage and cost of the drug used and lessened risk of developing potentially deleterious side effects associated with systemic administration.

Schneider *et al.* (1992a) reported that while treating septic arthritis it was important to remove the pus or fibrin clot from the joint cavity as they might interfere with antimicrobial agents and might have detrimental effect on the integrity of the surface of the joint.

Schneider *et al.* (1992b) treated 26 horses with septic arthritis / tenosynovitis and reported that open drainage with joint lavage and intra-articular infusion of amikacin 1g (22 horses) or gentamicin 500 mg (two horses) or cefazolin

500 mg (two horses) was successful in resolving the joint infection in 25 of 26 horses.

Schneider (1998) reported that the synovial fluid drug concentration after intra-articular administration of antibiotics is 10 to 100 times the levels that could be achieved with systemic administration. The author further stated that amikacin (250 – 500 mg), cefazolin (250 – 500 mg) and ceftiofur (150 mg) had been injected into the infected joint without any observed ill effects.

Lescun *et al.* (2002) reported that the advantages of intra-articular administration of antimicrobials for the treatment of septic arthritis were high concentration of the drug at the site of infection, the ability to use cost-prohibitive drugs in small doses and a reduction in systemic adverse effects.

2.18.3 Joint lavage

Joint lavage removed loose intra-articular tissue debris and inflammatory mediators generated by the synovial lining. Removal of degradative enzymes from the joint in early stages of septic arthritis, allowed chondrocytes to increase their biosynthetic activity. Another mechanism by which lavage relieved the symptoms and increased the resiliency and stiffness of articular cartilage was through changing the ionic environment within the synovial fluid (Daniel *et al*., 1976)

Bertone *et al.* (1986) evaluated the effects of various concentrations of povidone-iodine solutions on the normal tarso-crural joint of horses and reported that 0.1 per cent povidone-iodine solution was rapidly bactericidal and did not induce more synovitis or articular cartilage damage than that induced by balanced electrolyte solution.

Martens *et al.* (1986) reported that distension irrigation and through-and-through needle lavage were rarely effective when the inflammatory process was advanced, when the debris was too large or too viscous to be aspirated or when the fibrin clots had organized (usually 7 to 10 days after onset of synovial distension).

Prades *et al.* (1986) evaluated various lavage solutions for arthroscopic surgery and concluded that lactated Ringer's solution and other polyionic solutions were the least toxic solutions to the joints.

Bertone *et al.* (1987a) compared various treatments for experimentally induced equine infectious arthritis and concluded that joint drainage along with

administration of antimicrobial drug was mandatory to remove inflammatory debris and combat infection.

Bertone *et al.* (1987b) conducted experimental studies on equine infectious arthritis and opined that povidone-iodine solution had no special advantage over lactated Ringer's solution as joint lavage solution.

McIlwraith and Turner (1987) reported that the ideal joint lavage solution should be bactericidal, non-toxic and non-irritating to synovial membrane, articular cartilage and periarticular structures.

Van Huffel *et al.* (1989) reported that joint lavage was rarely efficacious in the treatment of chronic septic arthritis in calves as accumulation of fibrin and pocketing of purulent material often made adequate joint drainage impossible.

Adair *et al.* (1991) conducted experimental joint lavage in clinically normal horses to compare the effects of DMSO (Dimethyl sulphoxide) and buffered lactated Ringer's solution on intra-articular structures with that of arthrocentesis alone and reported that joint lavage induced significantly greater inflammatory reaction than arthrocentesis alone. But there was no significant difference in intra-articular inflammatory change induced by buffered lactated Ringer's solution, 10, 20 and 30 per cent DMSO. The authors concluded that as DMSO had antimicrobial, hyaluronate protective and free radical scavenging properties it might be advantageous for the treatment of septic and non-septic joint diseases.

Wilson *et al.* (1994) studied the effects of 0.05 per cent chlorhexidine lavage on the tarso-crural joints of clinically normal horses and reported that chlorhexidine lavage caused marked joint effusion, pitting edema surrounding the joint, synovial ulceration, inflammation and abundant fibrin accumulation. The authors concluded that joint lavage with 0.05 per cent chlorhexidine diacetate, the lowest known bactericidal concentration was not recommended for equine joints.

Schneider (1998) advocated joint lavage with balanced electrolyte solution for the horses with septic arthritis and reported that lavage was a form of drainage and removed the inflammatory exudate containing lysosomal enzymes, neutral metalloproteinases, collagenase and hyaluronidase that could degrade the articular cartilage. However joint lavage through needles had limitations and could not effectively remove the

coagulum of fibrin and white blood cells accumulated in a chronic or non-responsive joint.

Jackson (1999) reported that joint lavage was a simple and effective treatment for septic arthritis in calves that had failed to respond to parenteral antibiotic therapy as it provided 80 per cent success rate with normal and pain free joint function.

Valla and House (2002) reported that accumulation of fibrin in the joint space of cattle often made it difficult to lavage septic joints effectively using needles.

2.18.4 Arthrotomy

Ndikuwera *et al.* (1989) treated septic gonitis in five Holstein-Friesian heifers by arthrotomy, debridement and intra-articular administration of 100 mg of methyl prednisolone acetate and 6 ml of streptomycin (330 mg/ml) and 8 ml of procaine penicillin (300 mg/ml) for one month. There was no clinical improvement in three heifers and the osteo-arthritic changes became more evident radiographically. Severity of the lameness was considerably reduced in other two cases and the joint function was good enough to allow the heifers to breed.

Bertone *et al.* (1992) observed a more severe and prolonged anaemia (a steady decrease in packed cell volume from a mean of 34 per cent before surgery to 25 per cent on day 13) in horses that underwent arthrotomy to remove fibrin from the tarsocrural joint with induced infectious arthritis and attributed this anaemia to considerable haemorrhage that occurred after arthrotomy through an inflammed joint capsule.

2.18.5 Arthroscopy

McIlwraith (1984) noted hyperaemia and petechiation of the villi, development of small hyperaemic villi in abnormal locations, new forms of villi, fusion of villi with fibrinoid strands and hypertrophy and hyperplasia of the villi during arthroscopic examination of joints of horses with synovitis.

Arthroscopy facilitated inspection of intra-articular soft tissue and cartilage that were not apparent on radiography and the visibility of most structures were

better than with arthrotomy (Miller and Presnell, 1985). Tissues were traumatised less with arthroscopy than with arthrotomy thus resulting in shortened recovery times (Person, 1989).

Arthroscopic surgery provided better visualization of the joint as well as access to larger portion of the synovial surface for inspection, removal of fibrin and synovectomy (McIlwraith, 1990).

Munroe and Cauvin (1994) performed arthroscopic debridement and lavage in two cases of septic arthritis in calves and reported that arthroscopy allowed excellent visualization of most parts of the affected joints, permitted a more selective and thorough debridement than would have been possible via arthrotomy and it also eliminated all debris and fibrin deposits by a thorough lavage under pressure.

Schneider (1998) reported that arthroscopic lavage of septic joints offered the advantages of evaluation of the cartilage surfaces, more complete removal of fibrin and curettage of areas of osteomyelitis.

O'Brien (1999) classified articular surface pathology during arthroscopic examination under the following headings.

1. Articular cartilage erosion – It was again noted on following subheadings
    i. Based on degree of erosion
        - Partial thickness
        - Full thickness with exposure of the subchondral bone
    ii. Based on distribution of lesion
        - Focal
        - Diffuse
2. Cartilage fibrillation
3. Eburnation

Wright *et al.* (2003) advocated closure of all arthroscope and instrument portals and bandaging following endoscopic surgery to treat contaminated and infected synovial cavities in 121 horses and reported that endoscopic surgery permitted effective debridement of infected tissue and physical cleansing of the synovial environment to a clean state and thus permitted closure of the portals which minimized the risk of further and/or secondary contamination or infection.

The authors further stated that application of bandage was a positive contributor to the control of infection and also minimized pain associated with synovial infection.

2.18.6 Synovectomy

McIlwraith (1983) indicated synovectomy for the treatment of septic arthritis in large animals to eliminate the colonizing bacteria and to protect the articular structures from the destructive effects of neutrophils and their enzymes and the free radicals released by the inflammed synovium.

Synovectomy was recommended in the treatment of septic arthritis as synovium released potent inflammatory mediators such as prostaglandins (Torholm *et al.*, 1983); harboured bacteria and sequestered inflammatory cells (Bertone and McIlwraith, 1987); and was the source of immunologic component of the inflammation (Riegels-Nielson and Jensen, 1984 and Riegels-Nielson *et al*., 1991).

Arnold and Kalunian (1989) reported that removal of synovium improved the joint function, reduced the joint pain and retarded destruction of the articular cartilage in severe inflammatory conditions such as rheumatoid arthritis in human being.

Riegels-Nielsen *et al.* (1991) performed arthroscopic synovectomy in the knee joints of rabbits with experimentally induced septic arthritis and reported that arthroscopic surgery for the management of septic arthritis provided superior visualization and surgical debridement of the joint, copious lavage facilitated by manipulation, inspection of the articular cartilage surfaces for damage and also allowed selective synovectomy to remove severely damaged or contaminated tissues. The authors also reported that early synovectomy also attenuated the destructive changes in articular cartilage.

Bertone *et al.* (1992) induced infectious arthritis by inoculating *Staphylococcus aureus* (3.4 to 3.9 cfu) into the tarso-crural joints of 8 horses to evaluate two methods of treatment viz. arthrotomy versus arthroscopy and partial synovectomy and reported that both treatments eliminated infection in all but a single joint. However, contamination with other organisms (*Streptococcus spp*. and *Enterobacter spp*.) developed significantly ($p<0.05$) more often in joints treated by arthrotomy.

Palmer and Bertone (1994) recommended synovectomy in the treatment of septic arthritis to remove inflammed, hypertrophic synovium and consequently to decrease the production and stimulation of enzymes degradative to cartilage.

Theoret *et al.* (1996) studied the reparative ability and function of synovium after arthroscopic synovectomy of the dorsal compartment of the equine antebrachiocarpal joint and reported that removal of synovial villi had no adverse effect on the long term composition of the synovial fluid including hyaluronan concentration or on the health of the articular cartilage or did not limit joint motion and did not result in lameness.

Hirsbrunner and Steiner (1998) treated chronic septic arthritis of the radio-carpal joint in two cattle that were refractory to parenteral antibiotics by arthroscopic debridement, partial synovectomy and lavage followed by implantation of collagen sponges impregnated with 250 mg of gentamicin and reported that the infection was eliminated from both the cattle and they recovered without residual lameness.

2.18.7 Anti-inflammatory drugs

Anti-inflammatory drugs should be used in the treatment of septic arthritis because chronically inflammed synovial tissue and its associated by-products could perpetuate cartilage destruction, even in the absence of sepsis (Martens, 1982).

2.18.7.1 Corticosteroids

Corticosteroids have also been shown to decrease the migration of neutrophils into the inflammatory site by decreasing capillary and membrane permeability and by inhibiting chemotaxis and adherence of neutrophils to vascular endothelium (Lees and Higgins, 1984).

Angus (1991) contraindicated intra-articular and parenteral corticosteroids in the treatment of neonatal septic polyarthritis. Intra-articular corticosteroids are contraindicated as it would exacerbate any erosion of the articular cartilage. Affected neonates might well have been poorly supplied with colostrum. Thus parenteral use of corticosteroids, which could have an immunosuppressant effect, is not recommended.

Intra-articular administration of corticosteroids into the inflammed joints inhibited the production of eicosanoids and platelet activating factor via the blockage of the membrane associate enzyme phospholipase A_2, virtually eliminating the pain (via the blockage of PGE_2) and decreasing the joint swelling (via stabilization of capillary endothelial cell membranes thus preventing edema formation) (May, 1992). In addition to endothelial membrane stabilization, the cell membranes of chondrocytes, synoviocytes, neutrophils and macrophages and their mitochondria and lysosomal membranes were stabilized and maintained. This stabilisation prevented the rupture of these cells and lysosomes and thus reduced the concentration of potentially degradative enzymes into the synovial fluid and articular cartilage matrix (Chunekamrai *et al*., 1989).

However, the deleterious effects of intra-articular corticosteroids such as depression of proteoglycans synthesis by chondrocytes and altered collagen fibril formation due to increase in the number of cross-linkages between type II fibrils have been confirmed in both *in vivo* and *in vitro* studies. These impaired chondrocytes metabolism were evident for long periods even after single administration (Trotter *et al*., 1991; Shoemaker *et al*., 1992).

2.18.7.2 Non steroidal anti-inflammatory drugs

Systemically administered non-steroidal anti-inflammatory drugs such as flunixin meglumine and phenylbutazone relieved pain and diminished the adverse effects of inflammation (Auer and Fackelman, 1981).

Palmer and Bertone (1994) reported that use of non-steroidal anti-inflammatory drugs in the management of infectious arthritis suppressed PGE_2 production and thus significantly reduced pain, joint inflammation and systemic signs associated with arthritis.

Weaver (1997) recommended analgesic in addition to antibiotics for the management of neonatal polyarthritis not only to reduce pain but also to encourage the calf to stand and nurse, to increase the frequency of feeding and to avoid additional problems of recumbency.

3. MATERIALS AND METHODS

3.1 Source of clinical material

Bovines presented to the Large Animal Surgery Unit of Madras Veterinary College Teaching Hospital, Chennai- 600 007, with the history and clinical signs of septic arthritis were subjected to detailed clinical, radiographic and laboratory examinations of the joint fluid samples from the affected joint and the cases diagnosed as septic arthritis were included in the study.

3.2 Criteria for inclusion in the study

A case with swollen, warm and painful joint was considered as septic arthritis if the analysis of the synovial fluid from the affected joint suggested bacterial infective arthritis with one or both of the following criteria,

1. A highly cellular appearance, observed subjectively in direct smear examination, with a predominantly polymorph population of cells (or) total nucleated cell count of more than 25,000 cells / mm^3 and/or more than 80 per cent polymorphs.
2. Culture of bacteria from the synovial fluid of affected joint (Rohde *et al.*, 2000 and Clements *et al.*, 2005)

3.3 Design of the study

Bovines with septic arthritis were randomly divided into three groups irrespective of their age, breed or joints affected and treated as follows.

Group	No. of animals	No. of joints	Treatment
I	12	Carpal – 15 Tarsal – 4 Elbow – 1 Fetlock - 1	Needle lavage with lactated Ringer's solution
II	12	Carpal – 15 Tarsal – 4 Elbow – 1	Arthroscopic fibrin debridement and lavage with lactated Ringer's

		Fetlock - 1	solution
III	12	Carpal – 15 Tarsal – 4 Elbow – 1 Fetlock - 1	Arthroscopic fibrin debridement, partial synovectomy and lavage with lactated Ringer's solution

3.4 Through and through needle lavage

3.4.1 Instruments

Sterile 1.5 to 3 inch long 14 or 16 gauge needles were used depending upon the size of the joint and nature of the joint fluid. A sphygmomanometer rubber bulb was attached to a needle inserted into the sachet above the fluid column. The fluid sachet was pressurized by pumping air into the sachet. A 50 ml or 30 ml syringe was used for initial lavage and subsequently the fluid was delivered through a sterile infusion set.

3.4.2 Portals

The portals used for inlet and outlet needles for various joints are given below. But whenever the maximal distension of the joint was away from the recommended site the ingress / inlet needle was inserted at the centre of maximum distension.

3.4.2.1 Carpal joint

With the joint partially flexed, a sterile inlet needle was inserted into the dorso-lateral joint capsule lateral to the tendon of extensor carpi radialis and medial to the combined tendon of two bellies of the common digital extensor muscle. To enter the radio-carpal compartment, the needle was inserted between the distal portion of the radius and the proximal dorsal edge of the intermediate carpal bone. The mid-carpal compartment was entered between the distal portion of the intermediate carpal bone and axial dorsal articular facet of the fourth carpal bone. The outlet needle was placed medial to the extensor carpi radialis tendon. The needle was inserted between the distal dorsal rim of the radius and proximal dorsal articular rim of the radial carpal bone for radio-carpal compartment. In the mid-carpal joint, the outlet needle was placed between the distal portion of the radial carpal bone and dorsal articular surface of the third carpal bone.

3.4.2.2 Elbow joint

The inlet needle was inserted into the depression between the lateral epicondyle of the humerus and the lateral tuberosity of the radius at the anterior edge of the lateral collateral ligament. The needle was directed obliquely in a caudo-medial direction to a depth of 2 to 3 cm. The outlet needle was inserted into the depression between the medial epicondyle of the humerus and the medial tuberosity of the radius just behind the medial collateral ligament.

3.4.2.3 Metacarpo-phalangeal joint

The inlet needle was inserted into the lateral out pouch of the metacarpo-phalangeal joint, medial to the lateral digital extensor tendon while the outlet needle was placed into the medial out pouch medial to the medial digital extensor tendon.

3.4.2.4 Tarsal joint

The inlet needle was inserted into the dorso-medial out pouch medial to the extensor tendon and 2 cm ventral to the medial malleolus of the tibia. The easily palpable medial malleolus was also used to assess the level of penetration. The outlet needle was placed in the centre of the distended plantar lateral pouch.

3.4.3 Anaesthesia and Control

Calves were fasted overnight and the adult cattle were prepared by withholding feed for 18- 24 hours and water for 12 hours prior to sedation. All the animals were sedated by administering xylazine hydrochloride[1] at the rate of 0.1 mg/kg body weight intramuscularly. The animals were secured in lateral recumbency with the limb to be examined above. The site was prepared by shaving, scrubbing with soap and water and rinsing with 70 per cent surgical spirit. One ml of two per cent lignocaine hydrochloride[2] was infiltrated subcutaneously at the site using 2 cm 21 gauge needle. An additional 1 ml of the solution was infiltrated into the deeper layers upto the fibrous joint capsule. A 20 gauge 1.5 inch long needle was introduced into the joint till few drops of joint fluid was seen at the hub of the needle. After draining the accumulated synovial effusion intra-articular analgesia was induced by infusing 4 to 10 ml of two per cent lignocaine hydrochloride

[1] Xylaxin, Indian Immunologicals, Hyderabad.
[2] Lignocaine Hydrochloride, Mount Mettur Pharmaceuticals Ltd., Gummidipundi.

solution into the joint depending upon the size of the joint and the needle was withdrawn. A minimum of 10 minutes was allowed for the analgesia to set in.

3.4.4 Technique

A sterile ingress needle was introduced into the joint through ingress portal for different joints as described above. The needle was advanced into the joint by taking care to avoid direct contact wherever possible with bones until some spontaneous flow of fluid occurred from the joint. A second needle of the same size was introduced through the egress portal in the same way as mentioned above. Lactated Ringer's solution[3] was the fluid used for the joint lavage. The fluid was warmed to 37º C before lavage. Initial lavage was performed using 50 ml or 30 ml syringe (Plate 1) so as to create turbulence and dislodge fibrin particles adhered to the synovial membrane. Lavage using 50 ml syringe was continued until the egress fluid was some what clear. The fluid was then delivered by attaching the infusion set from the pressurised fluid sachet to the inlet needle (Plate 1). The outlet portal is allowed to drain freely or a second infusion set was connected for drainage to a collecting system. Whenever the joint was open, the egress fluid was allowed to exit through the wound (Plate 2). The depth of the needle was adjusted to ensure good flow of fluid. The outflow tract was frequently occluded so as to distend the joint. Later the inlet and outlet portals were exchanged so as to reverse the direction of fluid and to ensure thorough cleaning. One to two litres of warm lactated Ringer's solution was used for lavage until the egress fluid becomes clear. At the end of lavage, the fluid was manually expressed from the joint. Once the residual fluid was expressed the limb was bandaged. The lavage was repeated on every third day in animals that had persistent effusion or lameness and continued until clinical improvement was detected.

3.5 Arthroscopy

3.5.1 Instrumentation

3.5.1.1 Telescope (arthroscope)

Standard wide angle 30º forward-oblique Hopkins II rod lens 4 mm telescope (Karl Storz Endoscopy India Pvt. Ltd., New Delhi), of 18 cm length (Plate 3k) containing incorporated fibre optic light transmission mechanisms were used in this study.

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[3] RL, Parenteral Drugs (India) Ltd., Indore.

3.5.1.2 Arthroscopic sheath

Arthroscope sheath was used to maintain the ingress portal within the joint, to protect the arthroscope and to direct the lavage fluid into the joint. The arthroscope was inserted and self locked into a 5.5 mm outside diameter sheath/cannula with a working length of 12 cm (Plate 3l). The sheath had one luer-lock adaptor and one stopcock mounted at the side that acted as ingress portal for the fluid system. The space between the sheath and arthroscope served as the passage for fluid flow into the joint.

3.5.1.3 Trocar/obturator

Sharp trocar and blunt obturator (Plate 3m&n) were used for introduction of the sheath into the joint before insertion of the arthroscope. The sharp trocar and arthroscopic sheath combination was used to pass through the tough fibrous part of the joint capsule and blunt obturator was used for insertion through the synovial membrane and for positioning the sheath within the joint.

3.5.1.4 Fibreoptic light cable

A 4.8 mm diameter, 250 cm long fibreoptic cable (Plate 3j) having connectors at either end with extreme heat resistance was used in the present study for transmission of light from the light source to the arthroscope without transfer of heat into the joint. Sterilization was done by immersing it in activated dialdehyde two per cent solution.

3.5.1.5 Camera

The endovision TELECAM-DX video camera with a focal length of 30 mm (Plate 3i) was attached to the eyepiece of the arthroscope and connected with camera control unit (CCU) through a 180 cm long cable. This lightweight camera with fine tuning adjustment, capture and print button facilitated the surgeon not only to capture the important images during operation but also to give command for print by himself. The camera was covered with sterile plastic sheet after attaching it with eyepiece of the arthroscope to maintain sterility.

3.5.1.6 Camera control unit

Camera control unit (Plate 3c) was connected with the video recorder (Plate 3b) and video colour printer (Plate 3d) by two separate connecting cables. CCU had

a control button to reset camera system's chromiance that conformed to the colour temperature of the light source what is known as 'white balancing'.

3.5.1.7 Light source

3.5.1.7.1 Cold light fountain XENON 300

In the present study, cold light fountain XENON 300 (Plate 3e) was used as a light source in 12 cases.

3.5.1.7.2 Halogen light

Halogen miniature light source 150 Watt-481C (Plate 3g) was used in another 12 cases.

3.5.1.8 Irrigating system

Joint distension and continuous irrigation of the joint was required for inspection of the joint, to increase the working room and to decrease bleeding within the joint. In the present study, Arthropump (Plate 3f), a motorized roller pump (HAMOU Endomat) with facilities to control both fluid flow rate and intra-articular pressure was used to pump irrigation fluid into the joint.

3.5.1.9 Egress cannula

An egress irrigation cannula of 7 cm length and 3.2 mm or 5 mm outer diameter (Plate 3o&p) with accompanying locking trocar with an angulated point for insertion through the joint capsule was used to flush the joint in order to clear away blood, fibrin and tissue debris. A long flexible egress tube was attached to the cannula to transmit the egress fluid to a bucket on the floor rather than spilling fluid over the surgical site (Plate 3e).

3.5.1.10 Grasping instruments

Grasping forceps (Plate 3s) with 16 cm working length and 5 × 11 mm jaws was used for retrieving and removing attached or detached fibrin clots, fragments and debris. Arthroforceps-III (Plate 3r), a biopsy forceps, 3.5 mm in diameter and having a working length of 13 cm or Ferris/Smith cup rongeur (Plate 3t) with 14 cm working length and 5 × 8 mm spoon was used for partial synovectomy.

3.5.1.11 Sterilization of instruments

Activated dialdehyde 2 per cent solution (Cidex ®[4]) was used for cold sterilization of instruments. The arthroscope and surgical instruments were soaked for a minimum of 10 minutes (Yovich and McIlwraith, 1986).

3.5.2 Arthroscopic procedure

3.5.2.1 Sedation, presurgical preparation and control

All the animals were sedated, restrained and prepared for arthroscopic intervention as described for through and through needle lavage. The animals were placed in lateral recumbency with limb to be treated on the upper side and secured separately while the other three limbs were strapped together.

3.5.2.2 Intra-articular analgesia and joint distension

The joint was distended (Plate 4A) before insertion of the arthroscopic sheath in to joint space to increase the working length and prevent damage to the articular cartilage when the trocar penetrated the joint capsule. In order to distend the joint, an 18 gauge, 1.5 inch sterile disposable needle was introduced into the joint through instrument portal (Table 1) and the accumulated synovial effusion was drained out. A 20 or 30 ml syringe filled with an equal amount of 2 per cent lignocaine hydrochloride and lactated Ringer's solution was attached to the needle and injected into the joint until resistance was felt and the joint was tightly distended to decrease the bleeding within the joint. Throughout the study, the joint distension was maintained by adjusting the flow rate and flow pressure of the irrigating fluid.

3.5.2.3 Portals

The portals used for arthroscopic examination of different joints are given in Table 1.

3.5.3 General arthroscopic procedure

After second surgical preparation of the site as described for arthrocentesis, the limb was suitably draped. A 6 to 10 mm stab incision was made through the skin and underlying tissue to the appropriate depth at the site of insertion of the

[4] Johnson & Johnson India Ltd., Mumbai.

arthroscope with a No.15 or No.11 blade (Plate 4B). The trocar was placed within the arthroscopic sheath and this combination was inserted through the fibrous joint capsule with a gentle twisting motion. The sheath was pointed perpendicularly to the skin surface, avoiding any tendency to angle toward the ultimate position of the scope. This position was important to obviate opening a subcutaneous dissection plane. When a "give" was felt associated with penetration of the fibrous joint capsule, the sharp trocar was replaced with the blunt obturator to complete the entry and positioning of the sheath within the joint. Advancement of the sheath within the joint was achieved most safely by the use of blunt conical obturator, because the articular cartilage was at risk for damage when the sheath containing the arthroscope was advanced. Once the arthroscopic sheath was in place, the blunt obturator was replaced with the arthroscope, and the fibre optic light cable and the ingress fluid system were attached to the arthroscope and the sleeve respectively. The endocamera was attached to the eyepiece of the arthroscope and connected to the camera control unit. The monitor was positioned in such a way that the arthroscope pointed towards the monitor and allowed the surgeon to view the monitor with ease and facilitated the surgeon's orientation of intra-articular structures. The arthroscope was white balanced for proper colour contrast using gauze sponges. The ingress fluid line was cleared of air bubbles to avoid entry of the latter into the joint. Video camera was then attached. Egress cannula was introduced through the instrument portal (Table 1) in a similar fashion. Any cloudiness or haemorrhage within the joint was cleared by opening the egress cannula and pumping fluid through the ingress system. Once the view was clear, the stopcock on the egress cannula was closed during visualization; otherwise, villi waving within the flowing fluid could obstruct the view. Simply by rotating the arthroscope (without changing the position of the arthroscope), the visual field of view was greatly increased. The skin incisions were closed with one or two simple interrupted sutures.

3.5.3.1 Carpal joint

The inter-carpal compartment was examined first followed by radio-carpal joint. Skin incision for the arthroscopic portal was made before joint distension to

avoid damage to the tendon sheaths and misplacement of the portal of entry point which might in turn decrease the manoeuvrability of arthroscope inside the joint (McIlwraith, 1990). The arthroscope was introduced through the lateral portal and the medial portal was used for insertion of instruments (Plate 4C). Following preliminary lavage, the medial angle of the joint formed by the distal radial carpal and third carpal bones were examined. After slight withdrawal of the Arthroscope, the viewing field was rotated proximally to examine the radial carpal then distally to examine the third carpal bone. By gradually withdrawing the scope and by directing the viewing field in palmar direction the articulations of the radial carpal, intermediate carpal and third carpal bones were examined. Then the arthroscope and instrument portals were exchanged. The arthroscope was inserted as far lateral into the joint as possible. The arthroscope was manipulated in a similar fashion as through the lateral portal and the lateral joint angle formed by the ulnar and fourth carpal bones, articulations of the ulnar, fourth and lateral part of the third carpal bones were examined.

The radio-carpal joint was examined in a similar fashion as that of mid-carpal joint. The arthroscope was first introduced through the lateral portal and medial joint angle formed by the distal radius and radial-carpal bone was examined. By gradually withdrawing and orienting the scope in a sagittal plane the dorsal surface of the distal radius and proximal part of the intermediate carpal bones were examined. Then by exchanging the arthroscope to the medial portal the lateral part of the distal radius and proximal part of the ulnar carpal bone were examined. The synovial surface of the medial and lateral pouch and dorsal recess of each compartment was carefully examined to identify fibrin deposits and pannus if any.

3.5.3.2 Elbow joint

The arthroscope was introduced just cranial to the cranial edge of the lateral condyle of the humerus and 2 cm proximal to the articulation of the humerus and radius. The arthroscope was then advanced in a caudad-proximad direction to enter the cranio-lateral aspect of the joint. The lateral condyle, the intercondylar area and axial surface of the medial condyle of the humerus and cranial aspect of the proximal surface of the radius were examined. Then the arthroscope was introduced

through the lateral synovial pouch in the olecranon fossa and caudal aspect of the humeral condyles, olecranon fossa and the cranial aspect of the olecranon, anconeal process and proximal caudal radius were examined.

3.5.3.3 Metacarpo-phalangeal joint

The arthroscopic examination of the metacarpo-phalangeal joint was carried out by first introducing the arthroscope through the lateral portal and egress cannula through the medial portal (Table). To begin with the arthroscope was introduced as far medially as possible and the synovial membrane of the medial pouch was examined. Then with slight retraction and orienting the arthroscope across the joint the synovial membrane flap at dorsal pouch proximal to the articular cartilage of the distal metacarpus was examined. Then by rotating the lens in distal direction the medial condyle, its mid-sagittal ridge and intercondylar groove of the distal metacarpus were examined. The tip of the arthroscope was then moved distally to examine the proximal dorsal articular edge of the first phalanx of the third digit. The synovial membrane of the dorsal joint capsule was also evaluated during this manoeuvre. The arthroscope and instrument portal were then exchanged and the arthroscope was inserted through the medial portal as far laterally as possible. The synovial membrane of the lateral and lateral pouch, lateral condyle, its mid-sagittal ridge and intercondylar groove of the metacarpus and proximal dorsal articular rim of the first phalanx of the fourth digit were examined in a similar fashion as described above.

3.5.3.4 Tarsal joint

The dorso-lateral pouch of the tarso-crural joint was examined by introducing the arthroscope through the centre of the dorso-medial out pouching (Table) as far laterally as possible. By rotating the field of view of arthroscope, proximal, middle and distal part of the lateral trochlear ridge and lateral malleolus of the tibia were examined. Then by slightly withdrawing the arthroscope and orienting the lens in plantar direction the intermediate ridge of the tibia and proximal portion of the trochlear groove were examined. The medial trochlear ridge was examined by further withdrawal of the arthroscope and by rotating the field of view in proximal direction. The more distal aspect of the joint was examined by flexing the hock or by moving the scope more distally. Then the arthroscope and

instrument portal were exchanged so as to examine the medial trochlear ridge and synovial membrane of the dorso-medial pouch.

The plantaro-lateral pouch of the tarso-crural joint was examined by introducing the arthroscope through the plantaro-medial out pouch and egress cannula through the plantaro-lateral out pouch. Systematic examination was performed starting with the tarso-crural joint flexed to approximately 90° and continuing until the leg was fully extended. The synovial membrane of the dorso-medial cul-de-sac was examined by directing the arthroscope dorsally. Then by withdrawing the arthroscope slightly and re-directing medially, the plantar aspect of the trochlear groove, medial trochlear ridge of the talus and caudal aspect of the distal tibia were examined. Then the arthroscope and instrumental portal were exchanged. Through the plantaro-lateral arthroscopic portal the synovial membrane of the dorso-lateral cul-de-sac and the lateral malleolus was examined by directing the arthroscope dorsally. Then the arthroscope was withdrawn and re-directed laterally to examine the plantar aspect of the trochlear groove and lateral trochlear ridge of the talus and caudal aspect of the lateral part of the distal tibia.

3.5.4 Arthroscopic findings

Arthroscopic evaluation of each joint was studied based on normal and/or pathological changes of the following structures.

3. 5.4.1 Synovial membrane and villi

Synovial membrane of each joint was examined thoroughly to note normal architecture and following changes (McIlwraith, 1984).

- Hyperaemia and petechiation of the villi
- Development of small hyperaemic villi in abnormal locations
- New forms of villi
- Fusion of villi and the presence of fibrinoid strands
- Hypertrophy and hyperplasia of the villi

3.5.4.2 Articular cartilage

In the present study, during arthroscopic evaluation, articular surface of each joint was examined thoroughly and any deviation from the normal architecture was

noted and tabulated. Arthroscopic observations on articular surface pathology were noted and classified on the following category as described by O'Brien (1999)

1. Articular cartilage erosion – It was again noted on following subheadings
    i. Based on degree of erosion
        - Partial thickness
        - Full thickness with exposure of the subchondral bone
    ii. Based on distribution of lesion
        - Focal
        - Diffuse
2. Cartilage fibrillation
3. Eburnation

3.5.5 Arthroscopic fibrin debridement and partial synovectomy

Arthroscopic fibrin debridement with and without partial synovectomy of the affected joint was carried out in animals of group II and III respectively. Following introduction of arthroscope and egress cannula through respective portals (Table 1) the joint was lavaged under high pressure (50 to 80 mm Hg) with egress cannula fully opened. The preliminary lavage was performed to remove loose debris and free floating fibrin particles and continued until the field of visualisation became clear. Egress cannula was then closed. A systematic examination of the various compartments, pouches and recesses within each compartment was carried out to identify pocketing of purulent materials, tissue debris, desquamated cartilage pieces, fibrin deposits, pannus formation and foreign material, if any. Grasping forceps was used to remove (Plate 4 D) loosely adherent materials and Ferris-Smith rongeurs for removal of firmly adherent fibrin deposits and pannus. After fibrin debridement (Plate 5 A, B, C & D) partial synovectomy was performed using rongeurs or biosy forceps. Areas of synovial membrane that were infected, severely inflammed, devitalised or necrotic were manually removed with rongeur (Plate 5 E & F). Then the joint was lavaged under high pressure until all the areas were visibly clean. The instrument and arthroscope portals were then exchanged so as to facilitate debridement and partial synovectomy on the other side. Portions of

cartilage pieces and synovial membrane were preserved for histological examination.

3.5.6 Post-operative care

All the animals were given systemic antibiotics – gentamicin[5] @ 3 mg/kg b.wt. I.V., b.i.d or amikacin sulphate[6] @ 7 – 10 mg/kg b.wt., I.V., o.i.d and procaine penicillin G[7] @ 22,000 I.U. / kg b.wt. I.M., o.i.d or ampicillin[8] @ 20 mg/kg b.wt. I.V., b.i.d as the initial antibiotic treatment. Gentamicin (40 to 240 mg) or amikacin (100 to 500 mg) was administered intra-articularly for first three days. Antibiotic treatment was changed if antimicrobial susceptibility results indicated that the organism was resistant or if the response to treatment was not detected. Duration of antibiotic treatment was determined by the response of the individual animal. Administration continued until there was consistent improvement in lameness and the animal became fully weight bearing, the swelling was resolved and the synovial fluid became clear and viscous. All the animals also received phenylbutazone or meloxicam for the first four days to decrease the inflammation and to improve use of the joint in the immediate post-operative period.

3.6 Patient evaluation

3.6.1 Anamnesis

A detailed history of each case was obtained and the following information was collected.

1. Duration of the condition before presentation.
2. Incidence of previous illness / injury.
3. In case of neonatal calves, details about colostrum intake, calving environment, and time taken by the calf to stand after birth were collected.
4. Previous treatment, if any?

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[5] Genster, Sterling Laboratory, Uppal, Ranga Reddy district.
[6] Amikacin sulphate, Mount Mettur Pharmaceuticals Ltd., Gummidipundi.
[7] Fortified procaine penicillin, sarabhai Zydus, Ahmedabad.
[8] Dynacil, Hindustan Antibiotics Ltd., Pune.

3.6.2 Signalment

Details of each case like breed, age, sex and joint/joints affected were recorded.

3.6.3 Clinical examination

Each case was monitored subjectively every day for joint heat, joint swelling / effusion, evidence of joint pain and gait at the walk and objectively evaluated for pulse rate, respiratory rate and rectal temperature.

3.6.3.1 Observation

Each case was observed carefully to note joint swelling / effusion, pointing of the limb and deformity, if any.

3.6.3.2 Palpation

The affected joint was palpated completely both during weight bearing and with the limb flexed to assess joint heat, pain and effusion. The unaffected contra lateral joint was also palpated to appreciate increased joint heat. Pain was detected when a painful withdrawal response was noticed on joint palpation or joint flexion.

3.6.3.3 Joint effusion

Joint effusion visualized or detected during observation and palpation was graded as 0: no distension, 1: mild distension, 2: moderate distension and 3: severe distension.

3.6.3.4 Degree of lameness

Lameness was graded on the day of presentation and at 3 days interval following treatment as per the method described by Anderson and Desrochers (2004) with little modification and graded as follows.

Grade 1: Normal gait

Grade 2: Mild: walk easily, readily; bear full weight on the limb but has an observable gait alteration; stand on all four limbs.

Grade 3: Moderate: reluctant to walk and bear weight but does use the limb to ambulate; short weight bearing phase of stride; rest the affected limb when standing; increased period of recumbency.

Grade 4: Severe: reluctant to stand; refuses to walk without stimulus; non- weight bearing on affected limb; 'hoops' over the limb rather than bear weight; does not use limb when standing and lies down most of the time.

3.6.4 Radiological evaluation

Cranio-caudal / dorso-palmar and lateral views of the affected joints were taken before and at the end of treatment using a Siemens 800 mA, 3 phase, 6 pulse x-ray generator.

3.6.5 Haematological evaluation

Blood samples were collected on the day of presentation and on 7^{th}, 14^{th}, 21^{st} and 28^{th} days after treatment for the estimation of the following hematological parameters as per the standard procedures (Jain, 1986).

1. Haemoglobin content estimated by Sahli's acid haematin method
2. Total erythrocyte count
3. Packed cell volume
4. Total leukocyte count
5. Differential leukocyte count

3.6.6 Serum glucose

Blood samples were collected for estimation of serum glucose level on the day of presentation and on 7th, 14th, 21st and 28th days after treatment and were done in a semiautoanalyser♣.

3.6.7 Synovial fluid analysis

3.6.7.1 Collection of Synovial fluid

Synovial fluid samples were collected on the day of presentation and on 7th, 14th, 21st and 28th days after treatment. The affected joint was clipped, shaved and prepared for aseptic surgery using povidone iodine scrub and surgical spirit rinse. Arthrocentesis was usually performed through the arthroscope portals described in Table 1 or at the site of maximum distension of the affected joint (Plate 6). Whenever wound was present with clinical signs cellulitis and trauma arthrocentesis was performed away from sites of possible periarticular infection. The animals were sedated by administering xylazine at the rate of 0.1 mg / kg body weight I.M. Synovial fluid samples were collected under strict aseptic precautions, not only to prevent infection of the joint at the time of arthrocentesis, but also to avoid subsequent secondary bacterial contamination of the sample. The synovial fluid specimens were collected in a 5 ml syringe through a sterile 18-gauge disposable needle and placed into vacutainer tubes containing ethylene diamine tetra acetic acid (EDTA) in order to improve the cytological details of synovial fluid leucocytes.

3.6.7.2 Physical examination

3.6.7.2.1 Appearance

Colour observed at sample collection was characterized as colourless, haemorrhagic or yellowish. Appearance of the sample on visual examination was categorised as clear, turbid or purulent. Presence of flocculent material, if any, was also noted.

♣ BTS – 320 photometer, Biosed, Castlenuovo di Porto – Roma.

3.6.7.2.2 Volume

The total volume of joint fluid that was aspirated from the affected joint was recorded.

3.6.7.2.3 Mucin clot test

Mucin clot test was performed either on a glass slide or in a test tube. One drop of synovial fluid was mixed on a slide with three drops of two per cent acetic acid solution or 0.5 ml of synovial fluid was slowly added to 2 ml of two per cent acetic acid solution in a test tube taking care that the sample did not come in contact with the wall of the test tube. The tube was gently swirled and allowed to stand for one hour at room temperature and the clot was evaluated and graded as follows

Good - when there was a tight ropy clump in a clear solution,
Fair - when there was soft clot in a slightly cloudy pale yellow or pale amber solution,
Poor - when there were small, friable masses in a turbid and pale yellow or pale amber solution and
Very poor - when there were only a few flecks of precipitate in a very turbid pale yellow to dark yellow or pale amber to dark amber solution (Van Pelt, 1974).

3.6.7.3 Biochemical tests

The synovial fluid samples were subjected to biochemical analysis to estimate the glucose, total protein, albumin, globulin and alkaline phosphatase levels. Biochemical estimations were done in a semiautoanalyser.

3.6.7.4 Synovial fluid cytology

3.6.7.4.1 Synovial fluid total leukocyte count

For total leukocyte count, the synovial fluid was diluted with isotonic saline solution in a 1:2 dilution and mixed thoroughly. The filled haemocytometer was placed in a wet chamber for 10 minutes to allow the cells to settle and the cells were counted with a light microscope.

3.6.7.4.2 Synovial fluid differential leukocyte count

Differential counts were made from Leishman stained direct smears immediately after sample collection. For making direct smears a small drop of synovial fluid was placed near one end of a clean glass slide. The drop was allowed to spread along the edge of the spreader slide, which was advanced slowly at approximately a 25˚ angle, depending on the viscosity of the fluid. For thinner fluid, the angle and speed of the spreader slide were increased. Air-dried smears were stained with Leishman stain and examined under light microscope. Cells were classified into two categories viz. mononuclear (monocyte-macrophage and lymphocyte) and polymorphonuclear cells. The number of cells per field at 400X or 1000X magnification (used if cell number was too high at 400X) was recorded. Ten fields were examined in the body of each smear and the mean cell count was recorded. The mean percentage (based on ten fields and a minimum of 200 cells) of polymorphonuclear (PMN) and mononuclear cells was calculated. The synovial fluid was considered normal if no more than two cells per field at 400X magnification were noted and its differential included less than 12 per cent PMN cells and septic if more than 5 to 10 cells with more than 80 per cent PMN cells (Fernandez *et al.*, 1983).

3.6.7.5 Synovial fluid Bacteriology

3.6.7.5.1 Grams staining

Synovial fluid smears were stained with Gram's stain and examined under light microscope to identify the presence of bacteria, if any, as per the standard procedure (Coles, 1986).

3.6.7.5.2 Bacterial isolation and identification

Synovial fluid samples were subjected to bacterial isolation by direct plating on blood agar and nutrient agar media. After incubation at 37ºC for 24 hours suspected colonies of the bacteria were lifted on blood agar slants and identified by microscopic, cultural and biochemical characteristics (Cruickshank, 1960).

3.6.7.5.3 Antibiotic sensitivity test

The antibiotic sensitivity test was conducted by Bauer method (Bauer *et al.*, 1966). Synovial fluid samples were inoculated into nutrient broth and incubated at 37ºC for 24 hours. A sterile swab dipped in broth culture was evenly smeared on five per cent bovine blood agar plate. Then antibiotic discs were placed on the inoculated surface. The different antibiotics used for the purpose were streptomycin, gentamicin, ampicillin, amoxycillin, oxytetracyclin, penicillin-G and ciprofloxacin. After incubation at 37ºC for 24 hours the zone of growth inhibition was recorded as sensitive and lack of growth inhibition as resistant. The antibiotic to which the organisms were most sensitive was selected for the treatment.

3.6.8 Histological processing

During arthroscopic examination in group III cattle, biopsy materials from the diseased synovial membrane and articular cartilage were collected for histopathological examination. The specimens were fixed in neutral-buffered 10 per cent formalin for no longer than three weeks. Specimens were embedded in paraffin and 5 µm sections were prepared and stained with H & E. Sections were subsequently examined by use of light microscopy (Anderson *et al.*, 1993).

3.6.9 Outcome

The response to treatment was determined by clinical examination of the animal and synovial fluid analysis. Resolution of joint infection was confirmed at follow-up examination if the swelling was resolved, the animal became fully weight bearing on the limb and the arthrocentesis of the affected joint, produced synovial fluid of normal volume, gross appearance and normal cytological characteristics as evidenced by mildly cellular appearance upon smear examination with less than 10% neutrophils or a total nucleated cell count of less than 10,000 cells / mm (Clements *et al.,* 2005). Abnormal synovial fluid characteristics after treatment as described for inclusion criteria were ascribed to persistent joint inflammation and continued infection and therefore failure of treatment (Bennet and Taylor, 1988). The outcome was categorized as

Sound – if the infection was resolved without any residual lameness or swelling.

Functional – if the infection was resolved but with residual lameness/swelling in the affected joint and the animal was

retained (for the purpose for which it is maintained) with.

Unacceptable – if the animal had to be salvaged because of poor response to treatment.

3.6.10 Statistical analysis

Statistical analysis of clinical, haematological and synovial fluid parameters were performed to find out whether there was any significant difference in these values between different groups and among days by two way analysis of variance (ANOVA) with interaction using the software SPSS (Statistical Package for Social Science) version 10.

4. RESULTS

Thirty six cattle with 63 septic joints are presented in this study. The clinical summary of each case is given in Table 3.

4.1 Incidence

4.1.1 Breeds

Four different breeds of cattle were represented in the study. The breed wise distribution of cases is shown in Table 2 and Figure 2. Exotic crossbreds (83.33%) were most commonly affected than indigenous cattle (16.67%). Of the 36 cattle with septic arthritis, 22 were crossbred Jersey (61.11%), 8 were crossbred Holstein Friesian (22.22%), 2 were Tharparkar (5.56%) and 4 were non-descript (11.11%) cattle.

Table 2. Age, gender and breed of 36 cattle with septic arthritis

	Details	Number of cases
Breed	Crossbred Jersey	22
	Crossbred Holstein Friesian	8
	Non-descript cattle	4
	Tharparkar	2
Gender	Male	6
	Female	30
Age	Up to 1 month	25
	> 1 to 6 months	7
	> 6 months to 1 year	1
	> 1 year	3

4.1.2 Gender

Of the 36 cases of septic arthritis 30 were females and 6 were males (Table 2 and Figure 3).

4.1.3 Age

Age of the affected cattle varied from 5 days to 7 years. Age wise distribution of cattle affected with septic arthritis is given in Table 2 and Figure 4. Of the 36 affected cattle 25 were less than one month old (69.44%), 7 were above

one month to 6 months of age (19.44%) and 4 were more than 6 months of age (11.12%).

4.1.4 Joints affected

The sixty three joints with septic arthritis in 36 cattle included 45 carpal (71.43%), 12 tarsal (19.05%), three elbow (4.76%) and three fetlock joints (4.76%) (Figure 5). Of the 36 cattle, 11 had one affected joint, 23 had two affected joints and two had three affected joints. Of the 45 carpal joints, radio-carpal compartment alone was affected in 19 joints (42.22%), both middle carpal and carpo-metacarpal compartments were involved in 18 joints (40.00%) and all the three compartments were involved in 8 joints (17.78%). Of the 12 tarsal joints, both tibio-tarsal and proximal intertarsal compartments were involved in 11 joints (91.67%) while the involvement of distal inter tarsal and tarso-metatarsal compartments was observed in only one joint. The mean age of cattle with monoarthritis (17.5 months) was significantly higher ($p<0.01$) than that with polyarthritis (4.04 months).

4.2 Origin of infection

Of the 36 cattle with septic arthritis 33 were of tertiary origin and three were of primary origin. Among the 33 cattle with tertiary septic arthritis, omphalophlebitis was the source of infection in 18 cases, post partum metritis, respiratory tract infection and enteritis in one case each. Source of infection could not be ascertained in the remaining 12 cases. All the three cases with primary septic arthritis were the sequel to penetrating wound due to sharp objects.

4.3 Previous treatment

Of the 36 cases reported in the present study eight were treated unsuccessfully with antibiotics and/or non-steroidal anti-inflammatory drugs by the owner or local veterinarian. The duration of previous treatment varied from 1 to 4 days. Among the eight cases that underwent previous treatment two received only antibiotics and four had both antibiotics and non-steroidal anti-inflammatory drugs and two had non-steroidal anti-inflammatory drugs only.

4.4 Duration of infection

The time between onset of clinical signs of septic arthritis and presentation for treatment of each case are given in Table 3. The mean duration was 5.69 days and it varied from 2 to 15 days.

4.5 Clinical signs

Clinical signs noticed on the day of presentation are summarized in Table 4. The affected joint was swollen and painful in all the 36 animals and recorded as being palpably warm in 29 animals. Distension of the affected joint due to synovial effusion on the day of presentation was graded as severe (Plate 7A) in 43 (35 carpal, 7 tarsal and one fetlock) joints (68.25%) and moderate in 20 joints (10 carpal, 5 tarsal, 3 elbows and one fetlock) joints (31.75%). The owners reported lethargy and depression in 15 cases. A discharging sinus/wound was noticed on the dorsal aspect of carpus (Plate 7 B) in 3 cases and lateral aspect of the hock in one case. Partial flexion of the affected joint with limited range of movement was observed in carpal joint of two animals (Plate 7C) and tarsal joint of one animal. Severe lameness (Plate 7D) was seen in 25 cases and moderate lameness in 11 cases. General systemic signs such as pyrexia in 27 cases and inappetence in 13 cases were also observed. Erythema of the skin over the affected joint was noticed in 4 cases.

Table 4. Summary of clinical findings

S.No.	Clinical findings	Number of cases
1.	Painful swollen joint(s)	36
2.	Palpably warm joint(s)	29
3.	Erythema of the overlying skin	4
4.	Discharging sinus / wound	4
5.	Limited range of movement / partial flexion of the affected joint	3
6.	Lameness	36
	Moderate	11
	Severe	25
7.	Pyrexia	27
8.	Inappetence	13

9. Lethargy and depression 15

4.6 Lameness score

Mean ± S.E. values of lameness score recorded at three days intervals from the day of presentation up to 16th day in the three groups of animals are given in Table 5. On the day of presentation, animals exhibited moderate to severe lameness with a mean score of 3.58, 3.67 and 3.83 in groups I, II and III respectively. The lameness was graded as severe in 25 cases (7, 8 and 10 cases in group I, II and III respectively) and moderate in the remaining 11 cases (5, 4 and 2 cases in group I, II and III respectively). In subsequent intervals the lameness score showed a declining trend (Figure 6) in all the three groups. However, it remained significantly higher ($p<0.05$) on days 4, 7, 10, 13 and 16 in group I when compared to their respective values of both group II and III. There was no significant difference in the mean lameness score between group II and III on all post-operative days. Majority of animals of both group II (83.33%) and III (91.67%) became sound on 10th post-operative day whereas in group I only 41.67 percent of the animals became sound on 10th post-operative day and 66.67 and 75.00 percent became sound on 13th and 16th post-operative days respectively.

4.7 Rectal temperature

Mean ± S.E. values of rectal temperature (°F) recorded in animals of group I, II and III from day 1 to 7 are given in Table 6. The mean rectal temperature on the day of presentation was significantly higher ($p<0.05$) than their respective value on day 7 in all the three groups. The mean rectal temperature on day 2 and 3 in group I was significantly higher ($p<0.05$) than their respective values in both group II and III. There was no significant difference in the mean rectal temperature between different groups on days 1, 4, 5 and 6. The rectal temperature declined gradually after initiation of treatment (Figure 7) and returned to its normal base value on day 4 in group I and on day 3 in both group II and III.

4.8 Pulse rate

Mean ± S.E. values of pulse rate per minute recorded in animals of group I, II and III from day 1 to 7 are given in Table 7. The mean pulse rate per minute on the day of presentation was significantly higher ($p<0.05$) than their respective value

on day 7 in all the three groups. The pulse rate declined gradually after initiation of treatment (Figure 8) and returned to its normal base value on day 4 in group I and on day 3 in both group II and III.

4.9 Respiratory rate

Mean ± S.E. values of respiratory rate per minute recorded in animals of group I, II and III from day 1 to 7 are given in Table 8. The mean respiratory rate per minute on the day of presentation was significantly higher than their respective value on day 7 in all the three groups. The respiratory rate declined gradually after initiation of treatment (Figure 9) and returned to its normal base value on day 4 in group I and on day 3 in both group II and III.

4.10 Haemogram

4.10.1 Haemoglobin content

Mean ± S.E. values of the haemoglobin content (g/100ml) recorded at different time intervals in the three groups of animals are given in Table 9. The haemoglobin content increased gradually from the day of presentation to day 28 in all the three groups (Figure 10). But the increase was non-significant up to day 14 in group I and up to day 7 in both group II and III. A significant increase ($p<0.05$) in haemoglobin content was recorded on day 14 and 21 in both group II and III and on day 21 and 28 in group I. The haemoglobin content reached its near normal base value on day 28 in group I and on day 21 in both group II and III animals.

4.10.2 Packed cell volume

Mean ± S.E. values of the packed cell volume recorded at different time intervals in the three groups of animals are given in Table 10. The packed cell volume also exhibited similar increase as that of haemoglobin content from the day of presentation to day 28 in all the three groups (Figure 11). The increase in packed cell volume was non-significant up to day 14 in group I and up to day 7 in both group II and III. A significant increase ($p<0.05$) in packed cell volume was recorded on day 14 and 21 in both group II and III cattle and on day 21 and 28 in group I cattle. The packed cell volume reached its near normal base value on day 28 in group I and on day 21 in both group II and III animals.

4.10.3 Total erythrocyte count

Mean ± S.E. values of the total erythrocyte count (million/mm^3) recorded at different time intervals in the three groups of cattle are given in Table 11. A gradual

increase in total erythrocyte count was observed from the day of presentation to day 28 in all the three groups (Figure 12). The increase in total erythrocyte count was non-significant up to day 14 in group I and up to day 7 in both group II and III. A significant increase ($p<0.05$) in total erythrocyte count was recorded on day 14 and 21 in both group II and III animals and on day 21 and 28 in group I animals. The total erythrocyte count reached its near normal base value on day 28 in group I and on day 21 in both group II and III animals.

4.11 Leucogram

4.11.1 Total leucocyte count

Mean ± S.E. values of the total leucocyte count ($1000/mm^3$) recorded in all the three groups at different time intervals are given in Table 12. The values were significantly higher ($p<0.05$) on the day of presentation when compared to their respective values on day 28 after treatment in all the three groups. At subsequent intervals there a gradual decline in mean total leukocyte count (Figure 13) was observed in all the three groups. The values returned to the near normal reference value on day 7 in both group II and III and on day 14 in group I.

4.11.2 Differential leucocyte count

Mean ± S.E. values of the differential leucocyte count (in percentage) recorded in all the three groups at different time intervals are given in Table 13. A significant increase ($p<0.05$) in percent neutrophil count with concomitant significant decrease ($p<0.05$) in percent lymphocyte count was noted on the day of presentation in all the three groups. In subsequent intervals a gradual decrease in percent neutrophil and a concomitant increase in percent lymphocyte were observed in all the three groups. The percent neutrophils and lymphocyte returned to their normal reference value on day 14 in both group II and III and on day 21 in group I. The mean value of percent eosinophil and monocyte fluctuated within normal reference value at different time intervals in all the three groups.

4.12 Synovial fluid findings

4.12.1 Appearance

The appearance of synovial fluid on the day of presentation and at subsequent intervals is given in Table 14. The colour of the synovial fluid on the day of presentation varied from colourless to yellow or red and majority of the samples were opaque and turbid with suspended fibrinous flocculent material (47.62%) or purulent (33.33%) on visual examination (Plate 8).

Table 14. Summary of synovial fluid findings on visual examination

Day	Appearance	Number of joints		
		Group I	Group II	Group III
1	1) Serous fluid with fibrino-flocculent material	1	2	-
	2) Watery yellow fluid with flocculent material	2	-	3
	3) Fibrino-purulent (Greyish / yellowish)	8	10	12
	4) Purulent (Greyish / yellowish)	8	7	6
	5) Uniformly diffuse haemorrhagic fluid	2	2	-
7	1) Fibrino-purulent (Greyish / yellowish)	4	-	-
	2) Turbid	12	5	2
	3) Slightly turbid	4	12	10
	4) Clear, colourless and viscous	-	4	9
	5) Clear, straw coloured and viscous	1	-	-
14	1) Fibrino-purulent (Greyish / yellowish)	4	-	-
	2) Turbid	3	2	-
	3) Slightly turbid	7	-	-
	4) Clear, colourless and viscous	5	19	17
	5) Clear, straw coloured and viscous	2	-	4
21	1) Fibrino-purulent (Greyish / yellowish)	4	-	-
	2) Turbid	2	-	-
	3) Slightly turbid	1	-	-
	4) Clear, colourless and viscous	12	21	17
	5) Clear, straw coloured and viscous	2	-	4

28	1) Fibrino-purulent (Greyish / yellowish)	4	-	-
	2) Turbid	-	-	-
	3) Slightly turbid	-	-	-
	4) Clear, colourless and viscous	15	21	17
	5) Clear, straw coloured and viscous	2	-	4

Synovial fluid samples gradually became more clear and viscous in the follow up samples in animals of all the three groups. On day 14 after initiation of treatment the synovial fluid became apparently clear and viscous in all the affected joints of group III cattle and 19 joints (90.48%) of group II cattle. However, only seven samples (33.33%) became clear and viscous on day 14 in group I and the remaining 14 samples (66.67%) appeared turbid and contained variable amounts of flocculent material. The clarity of the synovial fluid samples improved further on day 21 and all the 21 samples from group II and 14 samples (66.67%) from group I became clear and viscous. Variable amounts of flocculent material were still observed on day 28 in four samples from two animals of group I that were refractory treatment.

4.12.2 Volume

Mean ± S.E. values of the volume of synovial fluid that could be collected from each of the affected joint in all the three groups of animals at different time intervals are given in Table 15. On the day of presentation the values were significantly higher ($p<0.01$) when compared to their respective values on day 28 after treatment in all the three groups. The synovial fluid volume in group I cattle remained significantly higher ($p<0.01$) in all the post-operative days when compared to their respective values in group II and III. However, there was no significant difference in synovial fluid volume between group II and III in all the post-operative days. The synovial effusion observed on the day of presentation decreased gradually (Figure 14) after initiation of treatment in all the three groups. The synovial fluid volume

decreased to its near normal base value on day 21 in group I and on day 14 in both group II and III. In two animals of group I that were refractory to treatment the synovial fluid volume remained higher through out the period of study.

4.12.3 Mucin clot quality

The mean ± S.E. values of the mucin clot quality score of the synovial fluid samples from affected joints at different time intervals in all the three groups are given in Table 16. The mucin clot quality of the synovial fluid was graded as very poor in 55 joints and poor in 8 joints on the day of presentation (Plate 9). The mucin clot quality score was significantly lower on the day of presentation in all the three groups when compared to their respective value on day 28. The mucin clot quality remained significantly lower up to day 21 in group I when compared to their respective value of both group II and III. The mucin clot quality score improved gradually (Figure 15) following treatment in all the three groups and returned to its near normal base value on days 28, 21 and 14 in group I, II and III respectively.

4.12.4 Total protein

Mean ± S.E. values of the total protein content (g/100 ml) recorded in synovial fluid samples from the affected joints of all the three groups of animals at different time intervals are given in Table 17. Mean total protein content (g/100 ml) of 6.13, 5.65 and 6.20 recorded in synovial fluid samples on the day of presentation in group I, II and III respectively were significantly higher ($p<0.01$) when compared to their respective values on day 28 after treatment. The mean total protein content of the synovial fluid showed a declining trend after initiation of treatment in all the three groups (Figure 16). However, it remained significantly higher ($p<0.01$) on day 7, 14 and 21 in group I cattle when compared to their respective values of both

group II and III. But there was no significant difference in the synovial total protein content between group II and III on all post- treatment days. The values returned to its normal base value on day 28 in group I and on day 14 in both group II and III respectively. The mean synovial fluid total protein content on day 28 in group I cattle remained significantly higher than its respective value of both group II and III.

4.12.4.1 Albumin

Mean ± S.E. values of albumin content (g/100 ml) recorded in synovial fluid samples from the affected joints of all the three groups of animals at different time intervals are given in Table 18. There was no significant change in mean synovial fluid albumin content between different groups as well as between different days within a group (Figure 17). The albumin content fluctuated within normal reference value during the post-operative periods in all the three groups.

4.12.4.2 Globulin

Mean ± S.E. values of globulin content (g/100 ml) recorded in synovial fluid samples from the affected joints of all the three groups of animals at different time intervals are given in Table 19. On the day of presentation the globulin content was significantly higher ($p<0.01$) in the affected joints of all the three groups. The globulin content of the synovial fluid showed a declining trend after initiation of treatment in all the three groups (Figure 18). However, it remained significantly higher ($p<0.01$) up to day 21 in group I and up to day 7 in both group II and III. The globulin content returned to its normal base value on day 28 in group I and on day 14 in both group II and III.

4.12.4.3 Synovial fluid albumin:globulin ratio

Mean ± S.E. values of albumin to globulin ratio in synovial fluid samples from affected joints of all the three groups at different time intervals are given in Table 20. The mean value of albumin to globulin ratio on the day

of presentation was significantly lower ($p<0.01$) when compared to their normal base value on day 28 in animals of all the three groups. Although the ratio increased gradually (Figure 19) after initiation of treatment in all the three groups it remained significantly lower up to day 21 in group I and up to day 7 in both group II and III. The ratio returned to its normal base value on day 28 in group I and on day 14 in both group II and III.

4.12.5 Alkaline phosphatase activity

Mean ± S.E. values of alkaline phosphatase activity in synovial fluid samples from affected joints of animals of all the three groups at different time intervals are given in Table 21. The mean alkaline phosphatase activity in synovial fluid was significantly raised ($p<0.01$) on the day of presentation when compared to their normal base value on day 28 in affected joints of all the three groups. After initiation of treatment, the follow-up samples showed a gradual decrease in synovial fluid alkaline phosphatase activity (Figure 20). However, the alkaline phosphatase activity in group I remained significantly higher ($p<0.01$) up to day 21 when compared to their respective values in both group II and III. The alkaline phosphatase activity returned to their normal reference base value on days 28, 21 and 14 in group I, II and III respectively.

4.12.6 Serum-synovial fluid glucose difference

Mean ± S.E. values of serum glucose level (mg/100ml) in all the three groups of cattle on the day of presentation and at subsequent intervals are given in Table 22. There was no significant difference in serum glucose level estimated at different time intervals as well as between different groups.

Mean ± S.E. values of glucose level (mg/100ml) in synovial fluid samples from affected joints of animals of all the three groups at different time intervals are given in Table 22. The synovial fluid glucose level on the day of presentation was significantly lower when compared to their respective base value on day 28 in affected joints of all the three groups. Although the synovial fluid glucose level increased gradually (Figure 21) after initiation of treatment it remained significantly lower up to day 21, 14 and 7 in group I, II and III respectively.

Mean ± S.E. values of serum synovial fluid glucose difference in affected joints of animals of all the three groups at different time intervals are given in Table

22. The serum-synovial fluid glucose difference on the day of presentation was significantly higher ($p<0.01$) when compared to their respective base value on day 28 in all the three groups. Although serum and synovial fluid glucose difference lessened (Figure 22) at follow up samples, it remained significantly higher up to day 21, 14 and 7 in group I, II and III respectively. There was no significant variation in serum synovial fluid glucose difference between different groups on day 28. The serum synovial fluid glucose difference reached its minimum base value on day 28, 21 and 14 in group I, II and III respectively.

4.12.7 Synovial fluid total nucleated cell count (TNCC)

The mean ± S.E. values of the total nucleated cell count ($1000/mm^3$) recorded in the synovial fluid samples from affected joints at different time intervals in animals of all the three groups are given in Table 23. The mean synovial fluid total nucleated cell count of the affected joints on the day of presentation was significantly higher ($p<0.01$) in all the three groups when compared to their respective base value on day 28. On the day of presentation, however there was no significant difference in the count among the three groups. Although a gradual decrease in the count was observed (Figure 23) after initiation of treatment in all the three groups it was more rapid in group II and III. The total nucleated cell count in group I remained significantly higher on all the post-operative treatment days when compared to their respective values in both group II and III. In group II, the count was significantly higher up to day 14 when compared to its base value on day 28. The count returned to its near normal base value on days 28, 21 and 14 in group I, II and III respectively.

4.12.8 Synovial fluid differential leucocyte count

Mean ± S.E. values of the percent polymorphonuclear (PMN) and mononuclear (MN) cell count recorded in synovial fluid samples from the affected joints at different time intervals in the three groups of animals are given in Table 24. Mean synovial fluid percent PMN cell count on the day of presentation was significantly higher and the MN cell count was significantly lower in comparison to their normal base value on day 28 in animals of all the three groups. No significant difference was observed in both PMN and MN cell count between different groups

on the day of presentation. After initiation of treatment the PMN cell count started decreasing and MN cell count started increasing (Figure 24) in all the three groups but the change was more rapid in group III and II when compared to group I. The PMN cell count remained significantly higher and MN cell count significantly lower on day 7, 14 and 21 in group I when compared to their respective values in both group II and III. Similarly the percent PMN cell count remained significantly higher and MN cell count significantly lower on day 7 and 14 in group II when compared to their respective values in group III. The count returned to its base value on day 28, 21 and 14 in group I, II and III respectively.

4.12.9 Synovial fluid cytology

Synovial fluid cytology revealed that neutrophils were the predominant cell type in majority of the samples on the day of presentation. Degenerative changes in neutrophil morphology were observed in samples from 15 joints (28.57%). Other cells such as erythrocytes in 10 samples (15.87%) and synoviocytes in four samples (6.35%) were also observed.

Bacteria were observed in Gram's stained synovial fluid smear in 45 (71.43%) samples. Of the 45 samples in which bacteria were observed 29 were extracellular (64.44%) and 16 were intracellular (35.55%). Bacteria were observed in synovial fluid smear from eight samples (12.70%) with an absence of bacterial growth upon synovial fluid bacterial culture. Of the 45 samples in which organisms were seen in Gram's stained smear 22 were Gram positive (48.89%) and 23 were Gram negative (51.11%). Yeast colonies were observed in two samples.

4.12.10 Synovial fluid culture

Results of culture of synovial fluid from the affected joint were given in Table 26. The synovial fluid culture was positive in 37 joints (58.73%) and negative in 26 joints (41.27%). The bacteria isolated were *Streptococcus spp*. (23.81%), *Escherichia coli* (14.29%), *Pseudomonas spp*. (11.11%), *Staphylococcus spp.* (6.35%) and *Salmonella spp*. (3.17%) (Table 26).

Table 26. Bacteria isolated from the synovial fluid of 36 cattle with 63 septic joints

S.No.	Bacteria	Isolates	
		No. of joints	Percentage
1.	*Streptococcus spp*.	15	23.81
	Beta hemolytic	9	14.29
	Non-beta hemolytic	6	9.52
2.	*Escherichia coli*	9	14.29
3.	*Pseudomonas spp*.	7	11.11
4.	*Staphylococcus spp*.	4	6.35
	Coagulase positive	2	3.17
	Coagulase negative	2	3.17
5.	*Salmonella spp*.	2	3.17
6.	Negative	26	41.27

4.12.11 Antibiotic sensitivity

The antimicrobial susceptibility of various bacterial isolates are given in Table 27. Among the 15 isolates of *Streptococcus* species 2 were susceptible to gentamicin, 10 to penicillin, 13 to amoxicillin, 10 to ampicillin, 11 to cloxacillin and 7 to tetracycline. All the 15 isolates were resistant to amikacin as well as to kanamycin. All the nine isolates of *E. coli* were susceptible to amikacin, 5 to kanamycin, 7 to gentamicin, 1 to penicillin, 4 to amoxicillin, 2 to ampicillin as well as cloxacillin, 1 to ciprofloxacin and 4 to tetracycline. All the 7 isolates of *Pseudomonas* were susceptible to amikacin and 5 to gentamicin. All the 7 isolates were resistant to all other antibiotics. The 2 isolates of *Salmonella* were susceptible to amikacin, kanamycin, gentamicin, ampicillin, cloxacillin, ciprofloxacin and tetracycline and were resistant to penicillin and amoxicillin. All the 4 isolates of *Staphylococcus* were susceptible to amikacin, 3 were susceptible to gentamicin, amoxicillin, cloxacillin, ciprofloxacin and tetracycline, 2 were susceptible to kanamycin, and 1 was susceptible to kanamycin. In the present study, *Pseudomonas* was the most resistant organism.

Table 27. Antibiotic sensitivity of bacterial isolates (%)

Antibiotic	*E.coli*	*Pseudomonas*	*Salmonella*	*Streptococci*	*Staphylococci*
Amikacin	100.00	100.00	100.00	0	100.00
Kanamycin	55.56	0	100.00	0	50.00
Gentamicin	77.78	71.43	100.00	13.33	75.00
Penicillin	11.11	0	0	80.00	25.00
Amoxicillin	44.44	0	0	86.67	75.00
Ampicillin	22.22	0	100.00	66.67	25.00
Cloxacillin	22.22	0	100.00	73.33	75.00
Ciprofloxacin	11.11	0	100.00	80.00	75.00
Tetracycline	44.44	0	100.00	46.67	75.00

4.13 Radiographic findings

Summary of the radiographic findings in Table 28 and details of radiographic findings of the affected joints are given in Table 29 and. Soft tissue swelling was observed in all the affected joints and in 52 joints (82.54%) it was the only finding. The soft tissue swelling was described as severe in 43 joints (68.25%) and moderate in 20 joints (31.75%). Increased intra-articular space was observed in 10 joints (15.87%), intra-articular gas shadow in 6 joints (9.52%) and subchondral osteolysis and blurring of normal bone outline in 3 joints (4.76%).

Table 28. Summary of radiographic findings

S.No.	Radiographic lesion	Number of joints				Total
		Carpus	Tarsus	Elbow	Fetlock	
1.	Soft tissue swelling	45	12	3	3	63
2.	Widened joint space	10	-	-	-	10
3.	Gas shadow	5	1	-	-	6
4.	Subchondral osteolysis and blurring of normal bone outline	2	1	-	-	3

4.14 Arthroscopic findings

Details of arthroscopic findings of the affected joints are given in Table 29 and summary of the arthroscopic findings in Table 30. The synovial membrane was hyperaemic with congested vessels in all the 42 joints. Petechial hemorrhages were observed in the synovial membrane in 10 joints and devitalised / necrotic areas in 6 joints. The villi were dense and thickened in seven joints, clubbing of villi with fibrin strands were observed in four tarsal joints and atrophy of villi in one tarsal joint. Free floating fibrin clots were observed in 18 joints and fibrin deposition in 24 joints.

Table 30. Summary of arthroscopic findings

S.No.	Radiographic lesion	Number of joints				Total
		Carpus	Tarsus	Elbow	Fetlock	
1.	Hyperaemic/congested synovial membrane	30	8	2	2	42
2.	Petechial hemorrhage in the synovial membrane	8	2			10
3.	Dense thickened villi	1	6	-	-	7
4.	Devitalised / necrotic areas in synovial membrane	6	-	-	-	6
5.	Clubbing of villi	-	4	-	-	4
6.	Atrophy of villi	-	1	-	-	1
6.	Free floating fibrin clots	17	-	-	1	18
7.	Fibrin deposits	13	8	2	1	24
8.	Pannus	1	5	1	-	7
9.	Articular cartilage erosion	3	-	-	-	3
10.	Hair	-	1	1	-	2
11.	Abnormal communication	8	-	-	-	8

4.15 Histopathology

On histopathological examination of the biopsy specimens bacterial colonies were observed in 11 synovial membrane specimens (52.38%), eight fibrin deposits, one articular cartilage and two subchondral bone specimens. Fibrin deposits over the articular cartilage and synovial membrane in 10 specimens (47.61%), degeneration and necrosis of synovial membrane in five specimens (23.81%),

granulation tissue formation four specimens (19.05%) and necrosis of subchondral bone in two specimens (9.52%) were also observed (Plate 42 to 49).

4.16. Outcome

Of the 21 joints treated by through and through needle lavage the outcome was described as sound in 14 joints (66.67%), acceptable in three joints (14.28%) and unacceptable in four joints (19.05%). The mean duration of infection before lavage was 3.25 days in cases which became sound, 6.5 days in cases in which the joint infection was resolved but had residual swelling/lameness and 8.5 days in cases which were refractory to treatment. The mean number of repeat lavage required for resolution of infection was 5.65 and it varied from 2 to 10. The mean time taken for resolution of joint infection in group I animal was 18.11 days and it varied from 7 to 28 days.

The outcome was described as sound in 20 joints each and acceptable in one joint each in both group II and III. However, the mean time taken for resolution of joint infection was 13.33 days and 11.13 days in group II and III respectively.

5. DISCUSSION

5.1 Incidence

5.1.1 Breed

In the present study exotic crossbred cattle (83.33%) were most commonly affected by septic arthritis than indigenous breeds (16.67%). Among the crossbreds, the incidence was more frequent in crossbred Jersey (73.33%) than crossbred Holstein Friesian (26.67%). Among the indigenous cattle, non-descript (11.11%) were more commonly affected than Tharparkar breed (5.56%). Genetic breed predisposition to septic arthritis has so far not been reported. However, Weaver (1981) and Kofler (1996) recorded septic arthritis more frequently in dairy cattle than beef cattle. The frequent presentation of crossbred cattle in the present study is a reflection of predominance of crossbred cattle in this locality and did not reflect any breed predilection to septic arthritis.

5.1.2 Gender

Female cattle were more frequently presented (83.33%) with septic arthritis than males (16.67%). Several reports indicate high incidence of septic arthritis in females (Van Pelt, 1972a; Ducharme *et al*., 1985; Butson *et al*., 1996; Kofler, 1996 and Rohde *et al*., 2000). However Singh *et al.* (1989) recorded higher incidence of joint affections in male (90.14%) than female (9.86%). In the present study over representation of female is a reflection of the hospital population rather than a disease predilection. It may also be due to much importance given by cattle rearers of this locality to heifer calves than bull calves which are neglected and are not frequently brought to hospital for treatment.

5.1.3 Age

In the present study young calves upto 6 months of age were more frequently (88.88%) affected by septic arthritis than adults (11.12%). Septic arthritis is frequently encountered in young calves as “neonatal polyarthritis” (Weaver, 1981 and Jackson, 1999). Whereas Merkens *et al*. (1984) reported that the incidence was not age dependent. But, Wehr *et al*. (1984) recorded septic arthritis of the carpal and tarsal joints in 25 to 35 per cent of the calves housed in calf

rearing premises. Rohde *et al*. (2000) also reported that the incidence was more in calves below 1 year of age (70.49%) than adults (29.51%). Similar observation was made by Verschooten *et al.* (2000) in a study of 445 cattle with bone and joint infection in appendicular skeleton wherein calves below three months accounted for 44.40 per cent. The higher incidence of septic arthritis in neonatal calves observed in the present study could be due to unhygienic calving environment and failure to disinfect the umbilicus in the immediate postnatal period (Weaver, 1997 and St.-Jean, 1999); abnormal birth circumstances including calving more than two weeks earlier than expected dates, excessive prepartum loss of colostrum or dystocia, excessive blood loss from the umbilical stump, delayed onset of standing or sucking and general weakness that increase the susceptibility of neonates to septic arthritis (Firth, 1983).

5.1.4 Joints affected

Carpal joint (71.43%) was most commonly affected with septic arthritis in the present study followed by tarsal (19.05%), elbow (4.76%) and fetlock (4.76%) joints. Similar observations were made by Wehr *et al.* (1984), Kofler (1996), and Jackson (1999). Whereas, frequent involvement of tibio-tarsal joint (78.00%) and stifle (34.92%) was reported by Van Pelt (1968) and Rohde *et al.* (2000) respectively. Septic arthritis may involve any articulation; however, the larger and high motion joints such as the stifle, hock, carpus and fetlock are affected most frequently (Martens *et al*., 1986). Larger and high motion joints are more susceptible to inflammatory reaction because of higher mechanical activity and a larger synovial membrane surface area (Rohde *et al.* 2000).

The reason for apparent predisposition of carpal joint to infection was not clear in the present study. In foals recent trauma has been reported to predispose a joint to infection (Firth, 1983). External trauma without skin damage may induce haematogenous bone and joint infection as traumatic lesions are probably easily infected in bovines (Verschooten *et al.,* 2000). Hyperemia and increased vasculature has been proposed as mechanisms by which traumatic arthritic joints are predisposed to bacterial infection (Palmer and Bertone, 1994). In neonates cartilage is vascular (Whalen *et al.,* 1988) and trauma to the developing cartilage

with associated haemorrhage and exposure of the bacterial binding sites might be the inciting cause for the location of infection in the joints (Santschi, 2004). Carpus is more likely to sustain 'functional trauma' (e.g., slight distortion, sprain of joint) than other joints as the cattle move on the dorsal aspect of these joints and carpus bears weight against hard floor while the animal is lying down and getting up (Verschooten *et al*., 2000). This may be a contributing factor for over representation of carpal joint in the present study.

5.2 Origin of infection

Tertiary septic arthritis of haematogenous origin has been frequently encountered in young calves (Van Pelt, 1969; Weaver, 1981; Rebhun, 1995; Berry, 1998; Jackson, 1999; Verschooten *et al.*, 2000 and Valla and House, 2002). As majority of the cases (69.44%) in the present study were less than a month old the incidence of tertiary septic arthritis was more frequent (91.67%) than primary septic arthritis (8.33%). Conversely less frequent observation of primary septic arthritis could be attributed to less frequent presentation of older calves and adult cattle. Omphalophlebitis was found to be the source of infection in 54.55 per cent of the cases with tertiary septic arthritis. Tertiary septic arthritis has been reported to be common in calves born in unclean environments and where immediate postnatal disinfection of the umbilicus was not practiced (Berry, 1998). Contamination of abdominal skin of the umbilical region has been foun more likely to occur in weak neonates that remained recumbent for some hours after birth (Firth *et al.,* 1980). Of the three tubular structures comprising the umbilicus (umbilical artery, vein and urachus) infection is more likely to develop in the flaccid and thin walled vein resulting in omphalophlebitis (Weaver, 1997). The other structures have a smaller lumen and with more elastic tissue in the wall tend to retract into the abdominal cavity far back to the top of the bladder after rupture of the umbilical cord but umbilical vein remain temporarily outside

the umbilicus and close gradually there by more prone for contamination and infection.

5.3 Previous treatment

For antibiotic therapy to be successful it is necessary for the antibiotic agents to cross the synovial membrane and their passage may be retarded by the thickened membranes (Orsini, 1984 and Lloyd et al., 1990). Of the 36 cases reported in the present study, eight were treated unsuccessfully with antibiotics and/or non-steroidal anti-inflammatory drugs by the owner. Parenteral antibacterial treatment alone is often insufficient to eliminate joint infections, particularly in the sub-acute or chronic stages as bacteria trapped in the fibrin clots are partially protected from the effects of systemically administered antibiotics (Hau et al. 1983); Presence of purulent effusion retards the action of many antibiotics particularly the activity of aminoglycosides by decreasing the metabolic rate of bacteria and by lowering of the pH value of the synovial fluid (McIlwraith and Turner, 1987). The failure of the arbitrary antimicrobial and/or non-steroidal anti-inflammatory drug therapy by the owner may also be due to inadequate concentration of antimicrobials achieved in the joint cavity or due to drug-resistant infection (Radostits et al., 2003).

5.4 Duration of infection

Clinical studies have indicated a direct relationship between duration of the infection before treatment and the ultimate result (Clements et al., 2005). Early identification and treatment of infectious arthritis is essential for successful management and return to function (McIlwraith, 1983 and Rohde et al., 2000). In the present study, the duration of clinical signs of septic arthritis before presentation for treatment was ≤ two days in two animals (8.33%), three to five days in 18 animals (50%), and more than five days in 15 animals (41.67%). Frequently a traumatic cause (stepped on or kicked by the dam) has been assumed by the owner. This assumption by the owner

caused a delay in the presentation of 9 cases (25.00%) cases. Treatment with antibiotic and/or anti-inflammatory drugs by the owner was the reason for delayed presentation in 8 cases (22.22%).

Delay in onset of treatment would result in rapid proteoglycans loss from articular cartilage (Steel et al., 1999); permanent osteoarthritis, lameness and loss of the affected animal (Johnson et al., 1970 and Daniel et al., 1976). In the present study, two cases of group I cattle that were presented seven and 10 days after onset of clinical signs of joint infection did not respond to through and through needle lavage with lactated Ringer's solution and one case each in group II and III that were presented 15 days after infection had residual swelling after completion of treatment. The residual joint swelling observed in the two cases even after resolution of infection could be due to periarticular thickening and fibrosis due to delay in initiation of appropriate treatment (Radostits et al., 2003).

5.5 Clinical signs

Animals of all the three groups exhibited symptoms of moderate (reluctant to walk, short weight bearing phase of stride, resting the affected limb when standing) to severe lameness (reluctant to stand, refuses to walk without stimulus, non-weight bearing on affected limb). Similar signs of lameness have also been observed by earlier workers (Munroe and Cauvin, 1994; Hirsbrunner and Steiner, 1998; Butson *et al.,* 1996 and Jackson, 1999).

Joint effusion on the day of presentation was graded as severe in 68.25 percent of joints and moderate in 31.75 percent of joints. Joint effusion is the first indication of synovitis (Van Pelt and Langham, 1968). The distension of the joint is usually due to synovial effusion and peri-articular edema in acute cases (Weaver, 1997 and Orsini, 2002) and thickening and fibrosis of the joint capsule in chronic cases (Van Pelt, 1970 and Stashak, 2002). The degree of distension is an indication of severity of inflammatory process and

varies depending upon the type, number and virulence of the bacteria (Leitch, 1979) and the pyogenic bacteria produces the greatest degree of swelling (Radostits *et al.,* 2003). The findings of the present study also supports this view as arthrocentesis of the severely distended joints invariably revealed purulent joint fluid and pyogenic bacteria like *S. aureus*, *E. coli* and *Pseudomonas* have been isolated from such joints.

Published clinical signs more commonly described for cattle with septic arthritis are lameness, enlarged and tender joints due to increased synovial effusion, pain on manipulation of the affected joint, arthrothermia, partial flexion of the limb when standing, limited range of joint motion and general systemic signs such as fever and inappetence to anorexia (Simmons and Johnston, 1963; Van Pelt and Langham, 1968; Merkens *et al.,* 1984; Ndikuwera *et al.,* 1989; Rebhun, 1995; Weaver, 1997; Madigan and House, 2002 and Radostits *et al.,* 2003). The prominent signs recorded in the present study included warm, swollen and painful joints with moderate to severe lameness. Erythema of the skin over the affected joints and discharging sinus were observed in 4 cases each, which has not been previously reported in arthritic cattle.

5.6 Lameness score

On the day of presentation, 69.44 per cent of the cases were severely lame and 30.56 per cent of the cases were moderately lame. Joint pain and lameness in septic arthritis are due to stimulation of low thresh-hold type II mechanoreceptors located within the joint capsule that are inactive when joints are immobile and are activated when the joint undergoes movement or experiences tension due to stretching of the joint capsule by effusion (Caron, 1996).

A mean lameness score of 3.58, 3.67 and 3.83 observed on the day of presentation decreased to 2.42, 1.75 and 1.67 on the 7th day and to 1.83, 1.17 and 1.00 on the 10th day in group I, II and III respectively. Marked improvement in lameness was noticed within seven days in animals of both group II and III. Munroe and Cauvin (1994) also observed no apparent swelling or lameness on 6th day following arthroscopic debridement and partial synovectomy in two calves with septic arthritis. Similar observations were also reported by Hirsbrunner and Steiner (1998) in two cows that underwent arthroscopic debridement and partial synovectomy for septic arthritis. However, the mean lameness score remained significantly lower in both group II and III in all the post treatment days when compared to its respective value in group I which indicates that arthroscopic surgery is more effective in decompressing the septic joints than conventional through and through needle lavage.

5.7 Rectal temperature, pulse and respiratory rate

A significant increase in rectal temperature, pulse rate and respiratory rate was commonly observed in animals of all the three groups. Similar significant or non-significant increase in these clinical parameters was also recorded in calves with experimentally induced septic arthritis (Pratap et al., 1977; Chawla et al., 1989b and Kumar and Singh, 1995) and clinical cases of cattle with septic arthritis (Munroe and Cauvin, 1994; Kofler, 1996; Hirsbrunner and Steiner, 1998; Riley and Farrow, 1998 and Jackson, 1999). The increase in these parameters could be attributed to the acute inflammatory reaction induced by the colonizing bacteria and subsequent release of non-specific mediators of inflammation such as kinin, histamine and complement (Lazarus et al., 1981); prostaglandins and superoxide radicals (Martens et al., 1986). Prostaglandin E2 from macrophage or synovial fibroblast promotes fever pain and inflammation (Coles, 1986).

5.8 Haemogram

The mean total erythrocyte count, hemoglobin content and packed cell volume on the day of presentation were lower than their respective values on day 28 in all the three groups. The lower haemogram values observed in the present study might be due to reduced availability of iron in the monocyte/macrophage system caused by inflammatory response to infection (Rodak, 1995). In acute inflammatory conditions the body produces increased fibrinogen as well as proteins that convert serum iron into ferritin. The process is continuous, and as long as the inflammatory reaction persists, serum iron is maintained at low level, even though the body iron stores may be normal or increased. Under these circumstances iron may be a limiting factor for erythropoiesis (Valli, 1985). These values showed an increasing trend after initiation of treatment and reached their near normal base value on day 28 in group I and on day 21 in both group II and III. Earlier workers also reported a significant decrease ($P<0.05$) in total erythrocyte count, hemoglobin content and packed cell volume in dairy cattle with idiopathic septic arthritis (Van Pelt, 1972a) and buffalo calves with induced septic arthritis (Pratap *et al.,* 1977). The gradual increase in haemogram value recorded in the post treatment periods suggest that the treatment methods were effective in eliminating joint infection.

5.9 Leucogram

Leukocytosis with significant increase in percent neutrophil and concomitant decrease in percent lymphocyte was observed on the day of presentation in all the three groups. Cattle characteristically develop leukocytosis with reversal of the neutrophil-lymphocyte ratio in acute localised infection (Valli, 1985). The percent eosinophil and monocyte count fluctuated within normal reference range without any consistent pattern. Earlier workers also reported leukocytosis with shift to left in septic arthritic cattle (Van Pelt and Langham, 1968; Van Pelt, 1972a and Pratap et al.,

1977). Leukocytosis with shift to left was commonly observed in acute septic focal inflammation with increased blood flow and swelling (Weiser, 2004); systemic infection with febrile inflammatory reaction and these changes were not due to direct depression of the bone marrow activity by bacterial infection but were the result of indirect effect through the nervous system (Kohli, 1968). Coles (1986) reported that localised infection by pyogenic microorganisms such as Staphylococci and Streptococci produced much greater neutrophilia than systemic disease as also observed in the present study. Moreover, the extent of neutrophilia is not always related to the extent of the localised process but the pressure exerted within it is more important. Increased intra-articular pressure due to severe joint effusion in majority of the cases with active local inflammatory reaction observed in the present study also supports this view. These parameters returned to their base value on day 14 in group II and III and on day 21 in group I which indicate the relative efficacy of the three treatment techniques.

5.10 Synovial fluid findings

5.10.1 Appearance

Normal synovial fluid in cattle is clear, colourleess or pale yellow, non-clotting and free from flocculent material (Krishnamurthy and Tyagi, 1977 and Weaver, 1997) whereas that from infected joint is turbid, amber to bloody and has fibrinous flocculent material (Van Pelt, 1970). The colour of synovial fluid on the day of presentation varied from colourless to yellow or red and majority of the samples were purulent or opaque and turbid with suspended fibrinous flocculent material. Red colour indicates fresh hemorrhage and yellow or amber fluid indicates previous hemorrhage and subsequent breakdown of erythrocyte and formation of conjugated bilirubin which imparts a dark yellow or amber colour to synovial fluid (Van Pelt, 1974). Bloody synovial fluid may be due to hemorrhage from the severely pathologic synovial membrane even from minor functional trauma to the

inflammed joint or due to previous intra-articular therapy (Van Pelt and Langham, 1968). Majority of the samples were opaque and turbid with suspended fibrinous flocculent material. The turbidity and opacity of the synovial fluid observed in the present study could be attributed to accumulation of cells and fibrin (Tulamo et al., 1989); degenerated cartilaginous fibrils and cellular debris (Perman, 1980 and Weaver, 1997). The synovial fluid samples gradually became clear after initiation of treatment. All the synovial fluid samples became clear and viscous on day 14 in group III and on day 21 in group II whereas only 80.95 per cent samples became clear on day 28 in group I. The difference in time taken for the synovial fluid to become normal reflects the relative efficacy of the three treatment techniques in removing the exudate, cellular debris and fibrin deposits and resolution of inflammatory reaction and infection.

5.10.2 Volume

The mean volume of the synovial fluid on the day of presentation was significantly higher than their respective mean value on day 28 in all the three groups. After initiation of treatment the synovial effusion started decreasing gradually and the volume returned to its normal base value on day 21 in group I and on day 14 in both group II and III. Significant increase in synovial fluid volume has also been reported in calves with experimentally induced septic arthritis (Kumar and Singh, 1995) and clinical cases of cattle with septic arthritis (Van Pelt, 1972a; Ndikuwera *et al.,* 1989 and Hirsbruner and Steiner, 1998). The amount of effusion present inside the joint cavity is generally at a maximum when the signs of inflammation are at their height (Perman, 1980). The findings of the present study also support this view as majority of the animals were presented with severe signs of septic arthritis and the volume of the synovial fluid on the day of presentation was 3.27 times as that of the volume on the day 28.

After initiation of treatment, as the signs of inflammation subsided in response to the treatment, the volume of the fluid also decreased gradually and returned to their base value at elimination of the infection. Amount of inflammatory exudate varies according to the vascularity of the areas and the kind of tissue (Jubb *et al*., 1993). Severe effusion following joint infection could be attributed to lack of both basement membrane and junctional complexes between the intimal synoviocytes and high vascularity of the loose areolar tissue of the subintima in high motion joints that allow rapid influx of plasma from the capillary fenestration through the synovial interstitium into the joint (Doige and Weisbrode, 1995).

5.10.3 Mucin clot quality

Determination of mucin clot quality is an estimate of intra-synovial hyaluronic acid concentration and its polymerization (Tulamo *et al*., 1989) and is good index of presence of infection in the joint (Moulvi *et al*., 1993). The mucin clot quality is poor to very poor in case of septic arthritis (Van Pelt, 1974) as also observed in the present study. The more inflammed the joint, the worse the mucin clot (Trotter and McIlwraith, 1996). Poor to very poor mucin clot quality is characteristic of marked inflammation and is indicative of reduced production and polymerization of the hyaluronic acid moiety in synovial fluid (McIlwraith, 1980). Very poor mucin clot quality observed in majority of the infected joints (87.30%) on the day of presentation reflects the intensity of the inflammatory process on the day of presentation and might be due to synovial dilution by effusion (Weaver, 1997); decreased production and poor polymerization of hyaluronic acid by synovial lining cells or hyaluronic acid degradation by bacterial hyaluronidase. The rapid improvement in mucin clot quality in group II and III indicated an early modulation of hyaluronic acid secreting ability of

synovial membrane as it is chiefly dependent upon the content and quality of hyaluronic acid (Gingerich *et al*., 1981).

5.10.4 Synovial total protein

Total protein content of the synovial fluid was significantly higher on the day of presentation in all the three groups when compared to their respective base value on day 28. A gradual fall in these values was observed in the post treatment period. However, the mean total protein content remained significantly higher up to day 21 in group I and up to day 14 in group II. The total protein content returned to its normal base value on day 28 in group I and on day 14 in both group II and III. Similar observations were made by earlier workers in cattle with experimentally induced septic arthritis (Kumar and Singh, 1995 and Chawla *et al*., 1989a) and clinical cases of septic arthritis (Van Pelt and Langham, 1968; Van Pelt, 1970; Ndikuwera *et al*., 1989 and Rohde *et al*., 2000).

The synovial fluid total protein content on day 1 was nearly 3 times higher than its respective value on day 28 in all the three groups and it was mainly due to marked increase in globulin content. This marked increase in globulin content leading to depression of albumin : globulin ratio observed in the present study could be regarded as an evidence of severity of inflammatory process as also described by Van Pelt (1970) and Anderson and Liberg (1980). The increase in total protein content may be attributed to influx of larger molecular proteins mainly globulins (viz. alpha2 and gamma) from plasma into the synovial space due to increased vascular permeability following synovial infection. Reduced quantity of synovial fluid hyaluronic acid content or depolymerization or both, that occurred following joint infection also facilitated passage and retention of proteins of increasingly higher molecular weight (Van Pelt, 1974). The increase in synovial fluid total protein content also explains increased volume of fluid as observed in the

present study by the fact that increased concentration of protein increases synovial fluid osmotic pressure and act as a barrier to fluid resorption (Fournier et al., 1969).

5.10.5 Synovial Alkaline phosphatase activity

A significant increase in synovial fluid alkaline phosphatase activity was recorded on the day of presentation in all the three groups in comparison to their respective base value on day 28. After initiation of treatment, the raised synovial fluid alkaline phosphatase activity started decreasing in the follow-up samples and returned to their base value on days 28, 21 and 14 in group I, II and III respectively. Earlier workers also reported increase in the activity of alkaline phosphatase in synovial effusion from septic arthritic cases (Van Pelt, 1973; Ndikuwera et al., 1989 and Kumar and Singh, 1995).

In healthy joints, plasma alkaline phosphatase does not enter the synovial cavity along with plasma dialysate and it is derived primarily from articular cartilage rather than from synoviocytes (Van Pelt, 1970). In septic joints the increase in synovial fluid alkaline phosphatase activity has been attributed to the increased neutrophil count (Van Pelt and Riley, 1967; Van Pelt and Langham, 1968) and depolymerization of mucin (Van Pelt, 1971). Further healthy articular cartilage provides a better source of alkaline phosphatase than does the diseased articular cartilage (Van Pelt, 1970). Therefore, it can be assumed that the increase in alkaline phosphatase activity observed in the present study is primarily derived from neutrophils and depolymerized mucin rather than articular cartilage. Van Pelt (1971) has also observed inverse correlation between synovial fluid viscosity and alkaline phosphatase activity in septic arthritis as also observed in the present study.

5.10.6 Serum and synovial fluid glucose difference (SSGD)

Synovial fluid glucose level on the day of presentation was significantly lower in animals of all the three groups in comparison to their respective base value on day 28. Normally the synovial fluid glucose level closely parallels that of serum or is slightly lower (Van Pelt, 1971). Therefore to be more meaningful as diagnostic tool the serum glucose level should simultaneously be estimated and compared with synovial fluid glucose level (Lipowitz, 1985).

Serum and synovial fluid glucose difference has been reported to be a diagnostic aid in infectious arthritis in man (Krey and Bailen, 1977) and horse (Van Pelt, 1971). A serum synovial fluid glucose difference of 15 mg/dl in humans (Cohen *et al*., 1975) and 23.4 – 39.6 (1.3 to 2.2 m.mol/litre) in horses has been considered as diagnostic for infectious arthritis. In the present study, the mean serum synovial fluid glucose difference (mg/dl) on the day of presentation were 44.75, 39.44 and 42.65 in group I, II and III respectively and exceeded the stated diagnostic value for humans and horses. The disparity between the serum and synovial fluid glucose level increase in septic arthritis may be due to increase in the number of synovial fluid leucocytes that metabolise glucose (Perman, 1980), rapid glycolysis by bacteria (Lipowitz, 1985) and pain and synovial necrosis (Tulamo *et al*., 1989).

5.10.7 Synovial total nucleated cell count

Cytological examination of the synovial fluid sample is the single most useful test in evaluating joint infection (Rohde *et al*., 2000). Synovial fluid of septic arthritis is generally characterized by a leukocyte count in excess of 10,000 cells per µl and a polymorph proportion greater than 90 percent (Jackson, 1999 and Orsini, 2002). Count as high as 1, 00,000 cells per µl has been reported (Orsini, 1984). Extent of leukocytosis is an indication of the magnitude of synovial membrane inflammation (Tulamo *et al*., 1989). The mean total nucleated cell count (1000/cumm) on the day of presentation was 33.12, 30.89 and 338.17 in group I, II and III respectively. Synovial fluid total nucleated cell count over 30,000 cells / cumm is pathognomonic for septic arthritis (Howard, 1993; St.-Jean, 1993 and

Rohde *et al.,* 2000). The synovial fluid total nucleated cell count on the day of presentation was significantly higher in all the joints of all the three groups when compared to their respective base value on day 28. Similar increase the count has been reported by several authors (Orsini, 1984; Ndikuwera *et al.,* 1989; Howard, 1993; Kumar and Singh, 1995; Rohde *et al.,* 2000 and Francoz *et al.,* 2005).

The increase in synovial fluid total nucleated cell count in the present study is attributable primarily to shedding of polymorphonuclear cells into the synovial fluid in response to latent forms of non-specific mediators of inflammation such as kinin, histamine, complement and components of the blood coagulation and fibrinolytic systems, including plasminogen and trypsin that entered the joint due to disruption of the blood-synovial barrier following infection (Lazarus *et al.,* 1981; Brown and Newton, 1985 and Schneider *et al.,* 1992b)

The influx of polymorphonuclear cells into the synovial fluid is the first attempt of the host to eliminate organisms that were recognized as foreign and started within 24 hours of contamination (Bertone and McIlwraith, 1987 and May, 2005). Chemotactic attraction of neutrophils by fibrin particles that accumulated in the joint cavity also contribute to higher total nucleated cell count (Brown and Newton, 1985 and Orsini, 2002). After initiation of treatment, the count declined gradually and returned to its base value on day 28, 21 and 14 in group I, II and III respectively which reflects the relative efficacy of the three methods of treatment in removing inflammatory debris and fibrin particles and subsequent resolution of infection. Early return of the count to its normal base value in group III could be attributed to removal of severely inflammed synovial membrane that sequestered polymorphonuclear cells into the joint cavity (Bertone and McIlwraith, 1987); removal of fibrin deposits that chemotactically attracted PMN cells and provided protected medium for bacterial growth (Munroe and Cauvin, 1994); debulking of bacterial load and physical cleansing of the synovial environment to a clean state by visually directed thorough lavage under high pressure Wright *et al.,* 2003).

5.10.8 Synovial differential leucocyte count

Total and differential synovial fluid leukocyte count is a rapid, sensitive and accurate method to differentiate between septic and other inflammatory conditions from non-inflammatory degenerative joint conditions (Van Pelt, 1974 and Jacques

et al., 2002). Rohde *et al.* (2000) recommended cytological differentiation of cellular synovial fluid constituents in addition to evaluation of total nucleated cell count and total protein concentration as these synovial fluid variables from cattle with infectious arthritis were significantly different from cattle with non-infectious arthritis. The findings of the present study also support this view as percent polymorphonuclear cell count was significantly higher with concomitant significant decrease in percent mononuclear cell count in animals of all the three groups. Similar increase in synovial fluid percent polymorphonuclear cell count with concomitant decrease mononuclear cell count has been reported by several authors in septic arthritic cattle (Coles, 1986 and Singh and Tayal, 1993).

The increase in polymorph proportion observed in the present study might be due to vasodilation in the subintima of the synovial membrane and release of chemotactic substances that work to increase the egress of neutrophil from local marginated pool in the subintima to joint cavity. After initiation of treatment, there was a gradual decrease in synovial fluid percent polymorphonuclear (PMN) cell count with concomitant increase in mononuclear (MN) cell count in animals of all the three groups except four joints in group I cattle (that were refractory to treatment) in which the percent PMN cell count remained significantly higher throughout the period of study. The return of proportion PMN cells to MN cells to their base value on day 28, 21 and 14 in group I, II and III respectively indicate the relative efficacy of the three treatment methods.

Neutrophils were the predominant cell type observed in synovial fluid smear on the day of presentation. The primary cell type seen in normal synovial fluid were mononuclear cells (Perman, 1980), whereas infected joints have a higher percentage of neutrophils (Weaver, 1997 and Rohde *et al*., 2000). Synovial fluid of infected joints in dog often contains degenerate neutrophils which differentiate it from other inflammatory arthropathies (Bennet and May, 1995). Whereas, Van Pelt (1971) and Koch (1979) reported that degeneration of neutrophils was not common in clinical cases of infectious arthritis in the horse. However, in the present study degenerative changes in neutrophil morphology were observed in synovial fluid samples from 23.81 per cent of joints with septic arthritis. Clements *et al*. (2005) also observed

degenerative changes in neutrophil morphology in 22.58 per cent of the dogs with septic arthritis. *Staphylococcus aureus* organisms cause degeneration and lysis in 3 hours which in turn initiates the necrosis of synovial membrane and articular cartilage by liberating lysosomal enzymes (Johnson *et al*., 1970). The findings of the present study also support the above fact as degenerated neutrophils were observed in synovial fluid smear from all the four joints from which *Staphylococcus aureus* was isolated. Erythrocytes were seen in 15.87 per cent of the samples which may be due to hemorrhage from the severely pathologic synovial membrane even from minor functional trauma to the inflammed joint or due to previous intra-articular therapy (Van Pelt and Langham, 1968) or contamination of the sample with blood at the time of arthrocentesis (Van Pelt, 1974). Synoviocytes were observed in 6.35% samples which indicate degenerative changes in synovial membrane (Perman, 1980) which was confirmed on arthroscopic examination of the affected joint.

Although bacterial culture is the gold standard test presence of bacteria in synovial fluid smear is a good evidence of infection (Madison *et al*., 1991). The value of synovial fluid smear examination is illustrated in the present study as bacteria were observed in synovial fluid smear in 12.70 per cent of cases with absence of bacterial growth upon synovial fluid bacterial culture.

5.10.9 Synovial fluid culture

Culture of synovial fluid from the affected joint was positive in 58.73 percent of joints and negative in 41.27 percent joints. Culture of synovial fluid is not always successful in septic arthritis (Bennet and Taylor, 1988). Positive synovial culture results usually confirm the clinical diagnosis of infectious arthritis, but negative culture results do not rule out infectious cause (Rohde *et al*., 2000). Microbiological culture of synovial fluid has been reported to yield bacterial growth in approximately in 50 percent of joints thought clinically to be infected (Madison *et al*., 1991). Negative culture results may be due to primary localization of bacteria in synovial membrane (Johnson *et al*., 1970 and Bertone *et al.*, 1987b); intrinsic bactericidal properties of septic synovial effusion (Moulvi *et al*., 1993). Poor collection, storage and laboratory techniques, prior administration of antibiotics or

partial success of the immune system in combating the infection may also contribute to failure to isolate bacteria from synovial fluid (Firth *et al*., 1980 and Steel *et al*., 1999). In the present study negative culture results in 11 joints (42.31%) was associated with prior administration of antibiotics. Amplification of bacterial DNA by polymerase chain reaction has been described in horses and may be valuable for rapid and specific diagnosis of septic arthritis particularly in cases already treated with antibiotics or infection caused by fastidious organisms (Crabil *et al*., 1996).

The species of bacteria isolated were *Streptococcus spp*. (23.81%), *E. coli* (14.29%), *Pseudomonas spp*. (11.11%), *Staphylococcus spp*. (6.35%) and *Salmonella spp*. (3.17%) which is in agreement with previous reports (Jubb *et al.,* 1993; St-Jean, 1993, Weaver, 1997 and Radostits *et al.,* 2003).

In the present study positive culture results for synovial fluid were obtained in 58.73 percent of affected joints. Between 12 and 43 percent of reported human cases (Gupta *et al*., 2003) and 23.75 to 45 per cent of equine cases (Madison *et al*., 1991; Schneider *et al*., 1992a and Steel *et al*., 1999) of septic arthritis will not yield positive synovial fluid culture. Therefore the diagnosis must be made upon based on clinical examination, radiography and synovial fluid analysis. A negative result from a bacterial culture of synovial fluid from clinically infected cases, as confirmed by synovial membrane culture (Bennet and Taylor, 1988) or experimentally infected joints (Montgomery *et al*., 1989) supports the assertion that a positive bacterial culture of synovial fluid is not a necessity for the diagnosis of bacterial infective arthritis.

Selecting an antibiotic is an important decision in the treatment of bovines with septic arthritis. Initial antibiotic therapy is usually instituted before culture and susceptibility results are known. Therefore, knowledge of the most common bacteria associated with septic arthritis and their probable antimicrobial susceptibility pattern is necessary to select an antibiotic that will eliminate infection. The bacteria cultured in this study reflect the population of microorganisms causing septic arthritis in bovine population of this locality. Isolation of a bacterial organism does not automatically implicate it as the cause of

infection (Moore *et al*., 1992). However, all the bovines in the present study showed evidence of bacterial infection of joint. Additionally, the specimens were collected after routine aseptic preparation of the overlying skin. Therefore, the organisms reported here represent those of clinical significance.

Streptococcus spp. was the most common bacteria isolated from neonatal bovine polyarthritis (Weaver, 1997) as also observed in the present study in which *Streptococcus spp*. constituted 23.81 per cent of the total isolates. Enteric organism [*E. coli* (14.29 %) and *Salmonella spp*. (3.17%)] was the second most common isolate followed by *Pseudomonas spp.* (11.11%). Neonatal polyarthritis following coliform septicemia is common in calves born in unhygienic environment due to faecal contamination of the skin of the umbilical region (Firth, 1983 and Weaver, 1997).

5.10.10 Antibiotic sensitivity test

Selection of antibiotics for treating joint infection requires consideration of the most frequently isolated organisms and their susceptibility (Rosin, 1990). But successful treatment is also dependent upon effective drainage in addition to antibiotic susceptibility (Schneider *et al*., 1992a). The *invitro* results should be extrapolated to the *invivo* situation with caution depending on the properties of the antibiotic and characteristics of the effusion (Moore *et al*., 1992).

All the 15 isolates of *Streptococcus spp.* were resistant to amikacin and 5 were resistant to penicillin. Although individually amikacin is ineffective and penicillin is not highly effective against *Streptococci*, the combination may be effective due to synergistic effect (Hegewald and Murphy, 1991) as also observed in the present study.

All the three Gram-negative bacterial isolates were highly susceptible to both amikacin as well as gentamicin. Amikacin and gentamicin were also effective against isolates of *Staphylococci*. The results of the present study indicate that amikacin was highly effective against all the isolates except *Streptococci*. Combining this drug with penicillin G sodium or ampicillin provides broad-spectrum coverage against majority of the bacterial isolates identified in the study.

Hence initial antibiotic therapy for septic arthritis should include a combination of amikacin or gentamicin and penicillin and other antibiotics shall be kept as reserve and used for treatment if culture and susceptibility results demonstrated their efficacy.

5.11 Radiographic findings

No significant radiographic abnormality was present in 82.54 percent of joints in which only soft tissue swelling was observed. Radiographic findings may be negative in early stages of septic arthritis (Weaver, 1981). Soft tissue swelling in and around the joint was the only radiographic evidence in early stage of joint infection (Weaver, 1997 and May, 2005). Other identified lesions in the present study were widened joint space (15.87%), intra-articular gas shadow (9.527%) and subchondral osteolysis (4.76%). Similar radiographic lesions have also been observed by earlier workers in bovine septic arthritis (Lekharu *et al.*, 1988; Kofler, 1996; Weaver, 1997 and Madigan and House, 2002). Intra-articular gas shadow observed in the present study could be due to gas producing organism such as *E. coli*, *Pseudomonas*, *Streptococci* and *Staphylococci* or due entry of air into the joint through penetrating wound.

Severity of the radiographic abnormalities varies depending upon the chronicity of the condition (Desrochers *et al.*, 1995). In the present study severe radiographic lesions like subchondral osteolysis and marked periosteal reactions were observed in three chronic cases. Further, no radiographic changes were observed in the follow up radiographs in animals of group II and III. However, 2 cases in group I in which there was no response to treatment exaggerated osteolytic and periosteal reactions were observed in the follow up radiographs. These findings indicate that arthroscopic treatment was more effective in restricting the pathological process in the joint than through and through needle lavage.

5.12 Arthroscopic findings

Hyperaemia and congestion with or without petechial haemorrhages were invariably observed in the synovial membrane of all the affected joints. These changes were consistent with synovitis and concur with what was recorded in horse (McIlwraith and Fessler, 1978 and McIlwraith, 1984)). Cauliflower like or polyp

like thickened villi and clubbing of villi with fibrin strands also were noted in the present study. These abnormal type villi were not seen in normal joints. McIlwraith (1987) also recorded such abnormal villi in ponies with experimentally induced infectious arthritis. Free floating fibrin clots and/or extensive fibrin deposits over the synovial membrane and articular cartilage and pannus formation were the other major arthroscopic findings. Similar changes have also been recorded by earlier workers (Munroe and Cauvin, 1994 and Hirsbrunner and Steiner, 1998).

Another interesting finding was the presence of abnormal communication between the between the radio-carpal and mid-carpal compartments on the dorso-lateral aspect in eight joints. The synovial sac of all the three compartments of the carpal joint is complete (Nickel et al., 1986). There is no communication between radio-carpal and mid-carpal compartments in 96 per cent of the bovine carpal joints and in the remaining four percent of the cases, the radio-carpal compartment communicated with the mid-carpal compartment between the ulnar and intermediate carpal bone (Descrochers et al., 1997). In the present study, abnormal communication between the two compartments on the dorso-lateral aspect were probably the result of tearing of the damaged joint capsule and separation of the facial planes by the excessive joint fluid and necrosis caused by joint infection.

The objectives in treating septic arthritis are removal of foreign material, debridement of infected and devitalised tissue, elimination of microorganism, removal of destructive enzymes and radicals, promotion of tissue healing and restoration of a normal synovial environment (Wright et al., 2003). These objectives were most effectively met by arthroscopic surgery in the present study. Arthroscopy permitted excellent visualization of all parts of the affected joint, thorough debridement of fibrin deposits and a more selective removal of infected, devitalised and necrosed synovial membrane (partial synovectomy), than would have been possible via arthrotomy. It also permitted the elimination of free floating debris, debulking of microorganisms, destructive radicals and enzymes by mechanical cleansing of all the recesses of the joint. Debridement and excision of septic joints followed by extensive visually directed lavage under high pressure made the joint to

a clean state and allowed closure of arthroscope and instrument portals which minimized the risk of secondary contamination.

The present study prioritized physical cleansing of synovial environment over medical management of pathogen. Physical cleansing of the infected joint to a contaminated/clean state helped to reduce the period of systemic antibiotic therapy. The mean duration of systemic antibiotic therapy was 13.33 and 11.13 days in group II and III respectively. The period of systemic antibiotic therapy in the present study was shorter than other reports and recommendations in literature (Orsini, 1984; Bertone et al., 1987a and Weaver, 1997). Reduced period of systemic antibiotic therapy following arthroscopic treatment of septic arthritis has also been reported in horses (Wright et al., 2003).

5.13 Histopathological findings

Fibrin deposition (over the synovial membrane and articular cartilage) and bacterial colonies (in synovial membrane, fibrin deposits, articular cartilage and subchondral bone) were the major findings on histopathological examination. Degeneration and necrosis of synovial membrane, articular cartilage and subchondral bone as well as granulation tissue formation were also observed. Earlier workers also reported bacterial colonies (Madison et al., 1991), granulation tissue formation, degeneration synovial membrane and articular cartilage (Van Pelt., 1972b and Ndikuwera et al., 1989). Identification of bacterial colonies in biopsy specimens justifies the usefulness of debridement and partial synovectomy in the management of septic arthritis.

5.14 Outcome

The prognosis for bovine septic arthritis should always be guarded (Rebhun, 1995 and Radostits *et al*., 2003) particularly in cases with multiple joint involvement and concurrent bony lesions (Madigan and House, 2002). However, the outcome was described as sound, acceptable and unacceptable in 66.67, 14.28 and 19.05 percent joints of group I animals. The results of

present study compare favourably with earlier reports with multiple treatment protocol (Verschooten *et al*., 1974 and Merkens *et al*., 1984).

The outcome was described as sound in 95.24 per cent joints and acceptable in 4.76 percent joints in both groups II and III which concurs with the findings of Butson *et al*. (1996) in both cattle as well as in horses, Hirsbrunner and Steiner (1998) in cattle Bertone *et al*. (1992) treated by arthroscopy followed by systemic antibiotic therapy or intra-articular implantation of gentamicin impregnated polymethylmethacrylate beads or collagen sponges. The mean time taken for resolution of joint infection was comparatively less (11.13 days) in group III when compared to that of group II (13.33 days) which could be attributed to the beneficial effects of partial synovectomy. Synovectomy eliminates the colonizing bacteria and protects the articular structures from the destructive effects of neutrophils and their enzymes and the free radicals released by the inflammed synovium (McIlwraith, 1983 and Bertone and McIlwraith, 1987). Synovectomy also improves the joint function, reduces the joint pain in severe inflammatory condition by eliminating the source of potent inflammatory mediators such as prostaglandins (Torholm *et al.*, 1983 and Arnold and Kalunian, 1989). The rationale for synovectomy was also justified by histopathological examination of biopsy specimen which revealed bacterial colonies.

5.15 Conclusion

1. Arthroscopy permits thorough evaluation, appropriate debridement and effective lavage of septic joints with minimal tissue trauma.
2. Arthroscopy provides consistently good results in the management of septic arthritis in bovines than conventional lavage.
3. Time taken for resolution of joint infection is significantly lower in arthroscopic management than conventional treatment.
4. Partial synovectomy is recommended during arthroscopic debridement of septic joints to eliminate colonizing bacteria.

5. Arthroscopy is indicated in cases refractory to conventional treatment and if a case is presented seven days or more after onset of clinical signs.
6. Needle lavage is recommended for cases that are presented within 5 to 6 days after infection.
7. Combination of amikacin or gentamicin with penicillin is recommended for initial antibiotic therapy. Other antibiotics shall be kept as reserve and used for treatment if culture and susceptibility results demonstrated their efficacy.

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6. SUMMARY

The research work was carried out to find out the incidence of septic arthritis in cattle and to compare the clinical efficacy of arthroscopic debridement, partial synovectomy and conventional through and through needle lavage in the management of septic arthritis by clinical, radiographic and synovial fluid examination. Thirty six clinical cases of bovines with septic arthritis were randomly divided into three groups of 12 animals each and treated by conventional lavage (group I), arthroscopic fibrin debridement and lavage (group II) and arthroscopic debridement, partial synovectomy and lavage (group III).

In the present study, exotic crossbred cattle (83.33%) were most commonly affected by septic arthritis than indigenous breeds (16.67%). Among the crossbreds, the incidence was more frequent in crossbred Jersey (73.33%) than crossbred Holstein Friesian cattle (26.67%). Female cattle were more frequently presented (83.33%) with septic arthritis than male (16.67%) among the cases presented to the hospital. The incidence was more frequent in young calves up to 6 months of age (88.88%) than adults (11.12%). Carpal joint (71.43%) was most commonly affected by septic arthritis followed by tarsal (19.05%), elbow (4.76%) and fetlock (4.76%) joints.

The prominent signs recorded in the present study included warm, swollen and painful joints with moderate to severe lameness. Erythema of the skin over the affected joints and discharging sinus were also observed. A mean lameness score of 3.58, 3.67 and 3.83 observed on day 1 decreased to 1.83, 1.17 and 1.00 on the 10th day in group I, II and III respectively. Elevated rectal temperature, pulse rate and respiratory rate observed on the day of presentation returned to it normal base values on day 3 in both group II and III and on day 4 in group I.

The lower haemoglobin content, packed cell volume and total erythrocyte count observed on the day of presentation showed an increasing trend after initiation of treatment and reached their near normal base value on day 28 in group I and on day 21 in both group II and III. Leukocytosis with shift to left observed on the day of presentation returned to their base value on day 14 in both group II and III and on day 21 in group I.

Majority of the synovial fluid samples on the day of presentation were purulent or opaque and turbid with suspended fibrinous flocculent material. All the samples of group III and 90.48 per cent samples of group II and 23.81 percent samples of group I became clear on the 14th post-operative day. A significant increase in synovial fluid volume observed on the day of presentation started decreasing in the post-operative period and the volume returned to its normal base value on day 21 in group I and on day 14 in both group II and III.

Poor to very poor mucin clot quality of the joint fluid observed on the day of presentation improved gradually following treatment in all the three groups and became almost normal on days 28, 21 and 14 in group I, II and III respectively. The highly elevated total protein content of the synovial fluid on the day of presentation returned to its normal base value on day 28 in group I and on day 14 in both group II and III. The increase in synovial total protein content observed on the day of presentation was mainly due to marked increase in globulin content and thus significantly lowered the albumin:globulin ratio. The globulin content showed a declining trend after initiation of treatment and returned to its normal base value on day 28 in group I and on day 14 in both group II and III.

A significant increase ($p<0.01$) in synovial fluid alkaline phosphatase activity recorded on the day of presentation started decreasing in the follow-up samples and returned to their base value on days 28, 21 and 14 in group I, II and III respectively. Significantly lower glucose level recorded in synovial fluid samples on the day of presentation increased gradually after initiation of treatment and reached normal value on days 28, 21 and 14 in group I, II and III respectively. Significantly higher serum and synovial fluid glucose difference observed on the day of presentation gradually lessened at follow up samples and reached its minimum base value on day 28, 21 and 14 in group I, II and III respectively.

Significantly higher ($p<0.01$) total nucleated cell count in synovial fluid samples from the affected joints on the day of presentation returned to its near normal base value on days 28, 21 and 14 in group I, II and III respectively. Significantly higher ($p<0.01$) percent polymorphonuclear cell count with concomitant significantly lower mononuclear cell count observed on the day of presentation returned to its near normal base value on days 28, 21 and 14 in group I, II and III respectively. Degenerative changes in neutrophil morphology were

observed in 28.57 percent of samples and other cells such as erythrocytes in 15.87 per cent samples and synoviocytes in 6.35 per cent samples were also observed.

Synovial fluid culture was positive in 58.73 per cent of joints and the bacteria isolated were *Streptococcus spp*. (23.81%), *Escherichia coli* (14.29%), *Pseudomonas spp*. (11.11%), *Staphylococcus spp.* (6.35%) and *Salmonella spp*. (3.17%). *Streptococcus spp.* was resistant to amikacin whereas *Escherichia coli*, *Pseudomonas spp*., and *Salmonella spp*. were highly susceptible to both amikacin as well as gentamicin. Amikacin and gentamicin were also effective against isolates of *Staphylococci*. Combination of amikacin and penicillin or ampicillin provided broad-spectrum coverage against majority of the bacterial isolates.

Radiographic examination revealed moderate to severe soft tissue swelling in all the affected joints and in 82.54 per cent joints it was the only finding. Increased intra-articular space was observed in 15.87 per cent joints, intra-articular gas shadow in 9.52 per cent joints and subchondral osteolysis and blurring of normal bone outline in 4.76 per cent joints.

The synovial membrane was hyperaemic with congested vessels in all the affected joints. Petechial haemorrhages were observed in the synovial membrane of 23.81 per cent joints and devitalised / necrotic areas in 14.29 per cent joints. The villi were dense and thickened in 33.33 per cent joints. Clubbing of villi with fibrin strands (9.52%) and atrophy of villi (2.38%) were also observed. Free floating fibrin clots were observed in 42.86 per cent joints and fibrin deposition in 57.14 per cent joints.

Fibrin deposition (over the synovial membrane and articular cartilage) and bacterial colonies (in synovial membrane, fibrin deposits, articular cartilage and subchondral bone) were the major findings on histopathological examination. Degeneration and necrosis of synovial membrane, articular cartilage and subchondral bone as well as granulation tissue formation were also observed.

The outcome was described as sound, acceptable and unacceptable in 66.67, 14.28 and 19.05 per cent joints of group I animals. The mean duration of infection before lavage was 3.25 days in cases which became sound, 6.5 days in cases in which the joint infection was resolved but had residual swelling/lameness and 8.5 days in cases which were refractory to treatment. The mean number of repeat lavage required for resolution of infection was 5.65 and it varied from 2 to 10. The

mean time taken for resolution of joint infection in group I animal was 18.11 days and it varied from 7 to 28 days.

The outcome was described as sound in 95.24 per cent joints and acceptable in 4.76 per cent joints in both groups II and III. However, the mean time taken for resolution of joint infection was 13.33 days and 11.13 days in group II and III respectively.

It is concluded that arthroscopy permits thorough evaluation, appropriate debridement and effective lavage of septic joints with minimal tissue trauma and offers consistently good results than conventional lavage. Time taken for resolution of joint infection is significantly lower in arthroscopic management than conventional treatment. Partial synovectomy is recommended during arthroscopic debridement of septic joints to eliminate colonizing bacteria. Arthroscopy is indicated in cases refractory to conventional treatment and if a case is presented 7 days or more after onset of clinical signs. Needle lavage is recommended for cases that are presented within 5 to 6 days after infection. Combination of amikacin or gentamicin with penicillin is recommended for initial antibiotic therapy. Other antibiotics shall be kept as reserve and used for treatment if culture and susceptibility results demonstrated their efficacy.

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Table 3. Clinical data, treatment and outcome of 36 cattle with septic arthritis

S.No.	Breed	Age	Sex	Joints affected	Origin	Probable source	Duration	Previous treatment	Temp. (° F)	Pulse rate/ min.	Resp. rate/min	Joint effusion	Lameness score	Synovial fluid culture	Treatment	Outcome
1	CBJ	6m	F	LC, RC	T	I	4d	-	103.6	82	26	m, m	3	Streptococcus	NL	S
2	CBHF	20d	F	LC, RC, LT	T	OP	8d	AB, AI	102.8	116	30	s, s, s	4	Pseudomonas	AFDL	S
3	CBJ	30d	F	LC, RC,	T	OP	4d	AB	103.0	106	38	m, m	4	Negative	APSL	S
4	CBJ	15d	F	LC, RC	T	OP	5d	-	104.2	124	48	m, m	4	Negative	NL	S
5	CBJ	3m	F	RT	P	PW	7d	-	101.8	98	40	s	3	Negative	AFDL	S
6	CBJ	5y	F	LC, RC	T	PPM	3d	-	101.2	72	22	s, s	3	Staphylococcus	APSL	S
7	CBHF	8y	F	LFF	P	PW	3d	-	102.0	88	20	s	3	Staphylococcus	NL	S
8	CBJ	15d	F	LC, RC, LFF	T	OP	6d	-	104.0	118	52	s, s, s	4	Negative	AFDL	S
9	CBHF	10d	F	LC, RC	T	OP	4d	-	103.8	126	48	s, s	4	Pseudomonas	APSL	S
10	ND	20d	M	LC, RC	T	OP	10d	AB	102.6	124	74	s, s	4	Negative	NL	UA
11	ND	45d	F	RC	T	I	3d	-	102.4	102	46	s	4	Streptococcus	AFDL	S
12	CBJ	20d	M	LC, RC	T	RTI	7d	-	102.4	112	44	s, s	4	Negative	APSL	S
13	CBJ	8d	F	LC, RC	T	OP	3d	-	104.2	132	66	s, s	4	E. coli	NL	S
14	ND	4m	F	LC	T	I	8d	AB, AI	102.0	78	42	s	4	Negative	AFDL	S
15	Tharp	2m	F	LT, RT	T	OP	15d	AB, AI	101.6	96	38	m, m	4	Negative	APSL	A
16	Tharp	9d	M	LT, RT	T	OP	2d	-	103.2	124	40	m, m	3	Salmonella	NL	S
17	ND	14d	F	LC, RC	T	OP	5d	-	104.0	142	88	s, s	4	E. coli	AFDL	S
18	CBJ	7m	F	LT, RT	T	I	7d	-	100.6	98	34	s, s	3	Streptococcus	APSL	S
19	CBHF	15d	F	LC, RC	T	OP	7d	AB	102.8	122	56	s, s	4	Pseudomonas	NL	UA
20	CBJ	10d	F	LT, RT	T	OP	6d	-	102.8	128	44	s, s	4	Negative	AFDL	S
21	CBJ	11d	F	LC	T	OP	7d	-	103.0	134	52	s	4	E. coli	APSL	S
22	CBHF	30d	F	LC, RC	T	I	3d	-	102.4	118	38	m, m	4	Streptococcus	NL	S
23	CBJ	7y	F	LC	T	I	7d	-	101.0	74	18	s	3	Staphylococcus	AFDL	S

24	CBJ	10d	F	LC, RC	T	OP	5d	AB,AI	102.2	116	38	s, s	4	Negative	APSL	S
25	CBJ	30d	F	RE	T	I	3d	-	101.8	112	34	m	4	Negative	NL	S
26	CBJ,	10d	F	LC, RC	T	OP	5d	-	104.2	122	48	s, s	3	Streptococcus	AFDL	S

Contd.

S.No.	Breed	Age	Sex	Joints affected	Origin	Probable source	Duration	Previous treatment	Temp. (° F)	Pulse rate/ min.	Resp. rate/min	Joint effusion	Lameness score	Culture	Treatment	Outcome
27	CBJ	30d	M	LE	T	I	8d	-	102.8	118	36	m	4	Streptococcus	APSL	S
28	CBHF	20	F	LC, RC	T	OP	8d	AB, AI	102.6	104	44	s, s	4	Negative	NL	A
29	CBJ	8d	F	LC, RC	T	OP	2d	-	104.4	130	60	s, m	3	E. coli	AFDL	S
30	CBJ	14d	F	LC, RC	T	Enteritis	4d	-	103.2	114	42	s, s	4	E. coli	APSL	S
31	CBJ	6m	F	LT,RT	T	I	3d	-	101.2	922	32	s, m	3	Streptococcus	NL	S
32	CBJ	10d	F	LC, RC	T	I	5d	AB	103.8	126	40	s, s	4	Negative	AFDL	S
33	CBJ	11d	F	RFF	T	I	3d	-	103.2	122	38	m	4	Streptococcus	APSL	S
34	CBHF	12d	M	LC	T	I	5d	-	103.0	120	36	s	3	Negative	NL	A
35	CBJ	2m	M	LE	P	PW	15d	AB, AI	101.8	108	34	m	4	Negative	AFDL	A
36	CBJ	15d	F	LC, RC	T	OP	2d	-	102.8	126	42	m, m	4	Streptococcus	APSL	S

Signalment:

CBJ: Crossbred Jersey, **CBHF**: Crossbred Holstein Friesian, **ND**: Non Descript, **Tharp**.: Tharparkar
d: Days, **m**: Months, **y**: Years, **M**: Male, **F**: Female

Joints

LC: Left carpal joint, **RC**: Right carpal joint, **LT**: Left tarsal joint, **RT**: Right tarsal joint, **LFF**: Left fore fetlock joint, **RFF**: Right fore fetlock joint, **RE**: Right elbow joint, **LE**: Left elbow joint

Origin

T: Tertiary, **P**: Primary

Source

I: Idiopathic, **OP**: Omphalophlebitis, **PPM**: Postpartum metritis, **PW**: Penetrating wound , **RTI**: Respiratory tract infection,

Previous treatment

AB: Antibiotics, **AI**: Anti-inflammatory drugs

Joint effusion

M: Moderate, **S**: Severe

Treatment

NL: Needle Lavage, **AFDL**: Arthroscopic fibrin debridement and lavage, **APSL**: Arthroscopic partial synovectomy and lavage

Outcome

S: Sound, **A**: Acceptable and **UA**: Unacceptable

Table 5. Lameness score at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 4	Day 7	Day 10	Day 13	Day 16
I	$3.58^{a} \pm 0.15$	$3.00^{b} \pm 0.25$	$2.42^{c} \pm 0.19$	$1.83^{d} \pm 0.24$	$1.50^{e} \pm 0.23$	$1.35^{e} \pm 0.19$
II	$3.67^{a} \pm 0.14$	$2.50^{c} \pm 0.15$	$1.67^{d} \pm 0.14$	$1.08^{f} \pm 0.08$	$1.08^{f} \pm 0.08$	$1.08^{f} \pm 0..08$
III	$3.83^{a} \pm 0.11$	$2.83^{b} \pm 0.11$	$1.67^{d} \pm 0.15$	$1.08^{f} \pm 0.08$	$1.08^{f} \pm 0.08$	$1.08^{f} \pm 0.08$

Means bearing different superscripts differ significantly ($p<0.05$)

Table 6. Rectal temperature (°F) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
I	102.93^{a} ± 0.22	102.35^{b} ± 0.16	101.60^{c} ± 0.11	101.12^{d} ± 0.16	100.92^{d} ± 0.13	100.93^{d} ± 0.12	100.91^{d} ± 0.14
II	103.17^{a} ± 0.33	101.79^{c} ± 0.23	101.07^{d} ± 0.02	100.87^{d} ± 0.06	101.12^{d} ± 0.08	100.87^{d} ± 0.06	101.10^{d} ± 0.04
III	102.85^{a} ± 0.29	101.56^{c} ± 0.16	100.92^{d} ± 0.07	101.06^{d} ± 0.08	100.77^{d} ± 0.06	101.02^{d} ± 0.06	100.70^{d} ± 0.05

Means bearing different superscripts differ significantly ($p<0.05$)

Table 7. Pulse rate / minute at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
I	$114.00^{a} \pm 4.41$	$103.17^{b} \pm 4.00$	$97.17^{c} \pm 3.77$	$90.16^{d} \pm 4.05$	$87.33^{d} \pm 3.93$	$89.33^{d} \pm 3.73$	$88.50^{d} \pm 3.60$
II	$109.50^{a} \pm 5.08$	$97.83^{c} \pm 5.10$	$90.16^{d} \pm 4.10$	$89.83^{d} \pm 3.90$	$90.17^{d} \pm 4.10$	$87.33^{d} \pm 3.80$	$90.00^{d} \pm 3.90$
III	$112.17^{a} \pm 5.90$	$94.33^{c} \pm 4.29$	$85.50^{d} \pm 3.88$	$84.34^{d} \pm 3.59$	$86.35^{d} \pm 3.66$	$88.50^{d} \pm 3.36$	$85.67^{d} \pm 3.52$

Means bearing different superscripts differ significantly ($p<0.05$)

Table 8. Respiratory rate / minute at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
I	$45.00^{a} \pm 4.98$	$40.06^{b} \pm 3.14$	$34.82^{c} \pm 2.23$	$29.50^{d} \pm 1.66$	$27.58^{d} \pm 1.36$	$28.83^{d} \pm 1.49$	$27.50^{d} \pm 1.44$
II	$43.17^{a} \pm 4.54$	$36.33^{c} \pm 3.14$	$28.17^{d} \pm 2.31$	$29.33^{d} \pm 1.68$	$25.83^{d} \pm 1.32$	$28.08^{d} \pm 1.59$	$26.12^{d} \pm 1.56$
III	$41.64^{a} \pm 2.24$	$34.62^{c} \pm 1.60$	$28.75^{d} \pm 1.55$	$26.50^{d} \pm 1.44$	$27.25^{d} \pm 1.60$	$27.00^{d} \pm 1.43$	$29.20^{d} \pm 1.44$

Means bearing different superscripts differ significantly ($p<0.05$)

Table 9. Haemoglobin content (g/100ml) in blood at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$8.44^{a} \pm 0.48$	$8.56^{a} \pm 0.45$	$8.60^{a} \pm 0.41$	$8.86^{b} \pm 0.43$	$9.12^{c} \pm 0.43$
II	$8.51^{a} \pm 0.28$	$8.67^{ab} \pm 0.26$	$8.87^{b} \pm 0.25$	$9.16^{c} \pm 0.26$	$9.14^{c} \pm 0.23$
III	$8.47^{a} \pm 0.27$	$8.51^{a} \pm 0.30$	$8.79^{b} \pm 0.29$	$9.06^{c} \pm 0.27$	$9.26^{c} \pm 0.27$

Means bearing different superscripts differ significantly ($p<0.05$)

Table 10. Packed cell volume (%) in blood at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$27.84^{a} \pm 1.54$	$28.04^{a} \pm 1.43$	$28.36^{a} \pm 1.62$	$29.72^{b} \pm 1.43$	$30.80^{c} \pm 1.47$
II	$28.02^{a} \pm 1.02$	$28.34^{a} \pm 0.95$	$29.58^{b} \pm 1.00$	$30.75^{c} \pm 1.07$	$31.50^{c} \pm 0.83$
III	$27.66^{a} \pm 0.83$	$27.68^{a} \pm 0.92$	$29.17^{b} \pm 0.96$	$30.67^{c} \pm 0.89$	$31.00^{c} \pm 0.75$

Means bearing different superscripts differ significantly ($p<0.05$)

Table 11. Total erythrocyte count (million/mm^3) in blood at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$4.21^{a} \pm 0.20$	$4.24^{a} \pm 0.19$	$4.28^{a} \pm 0.19$	$4.45^{b} \pm 0.20$	$4.72^{c} \pm 0.21$
II	$4.28^{a} \pm 0.12$	$4.37^{ab} \pm 0.13$	$4.49^{b} \pm 0.12$	$4.72^{c} \pm 0.13$	$4.85^{c} \pm 0.12$
III	$4.19^{a} \pm 0.12$	$4.24^{a} \pm 0.14$	$4.45^{b} \pm 0.14$	$4.67^{c} \pm 0.15$	$4.81^{c} \pm 0.14$

Means bearing different superscripts differ significantly ($p<0.05$)

Table 12. Total leucocyte (1000/mm^3) in blood at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$11.39^{a} \pm 1.91$	$9.78^{c} \pm 1.33$	$8.12^{d} \pm 1.24$	$8.08^{d} \pm 1.12$	$8.03^{d} \pm 1.05$
II	$11.84^{a} \pm 1.88$	$7.92^{d} \pm 0.59$	$7.85^{d} \pm 0.21$	$7.81^{d} \pm 0.21$	$7.84^{d} \pm 0.16$
III	$13.05^{b} \pm 1.51$	$7.86^{d} \pm 0.69$	$7.91^{d} \pm 0.41$	$7.73^{d} \pm 0.26$	$7.58^{d} \pm 0.24$

Means bearing different superscripts differ significantly ($p<0.05$)

Table 13. Differential leucocyte count (%) in blood at different time intervals in various groups of animals (Mean ± S.E.)

Group	Cell	Day 1	Day 7	Day 14	Day 21	Day 28
I	N	$59.16^{a} \pm 3.88$	$51.75^{ab} \pm 3.47$	$44.17^{b} \pm 3.64$	$37.42^{bc} \pm 3.84$	$33.58^{c} \pm 4.08$
	L	$38.25^{a} \pm 3.46$	$44.42^{ab} \pm 3.16$	$50.67^{b} \pm 3.38$	$56.92^{bc} \pm 3.72$	$60.34^{c} \pm 4.16$
	M	00.67 ± 0.28	01.67 ± 0.36	02.00 ± 0.33	02.33 ± 0.26	02.33 ± 0.26
	E	01.92 ± 0.38	02.16 ± 0.32	03.16 ± 0.32	03.33 ± 0.14	03.75 ± 0.35
II	N	$58.25^{a} \pm 5.28$	$46.00^{b} \pm 3.32$	$31.67^{c} \pm 1.14$	$28.94^{c} \pm 0.51$	$29.67^{c} \pm 0.59$
	L	$39.08^{a} \pm 5.06$	$50.17^{b} \pm 3.14$	$63.24^{c} \pm 0.96$	$65.86^{c} \pm 0.52$	$66.92^{c} \pm 0.73$
	M	01.17 ± 0.21	01.42 ± 0.26	01.92 ± 0.23	02.58 ± 0.36	01.83 ± 0.27
	E	01.50 ± 0.31	02.41 ± 0.36	03.17 ± 0.39	02.62 ± 0.47	02.58 ± 0.36
III	N	$54.91^{a} \pm 3.46$	$40.42^{b} \pm 2.54$	$31.50^{c} \pm 0.92$	$28.83^{c} \pm 0.64$	$28.67^{c} \pm 0.52$
	L	$41.02^{a} \pm 3.77$	$54.25^{b} \pm 2.66$	$62.25^{c} \pm 1.03$	$65.17^{c} \pm 0.84$	$65.17^{c} \pm 0.55$
	M	01.57 ± 0.28	02.08 ± 0.29	02.58 ± 0.36	02.58 ± 0.36	02.67 ± 0.31
	E	02.50 ± 0.58	03.25 ± 0.25	03.67 ± 0.28	03.42 ± 0.26	03.50 ± 0.44

Means bearing different superscripts within a row differ significantly ($p<0.05$)

N – Neurtrophil L – Lymphocyte M – Monocyte E – Eosinophil

Table 15. Synovial fluid volume (ml) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$18.67^{a} \pm 1.37$	$11.14^{c} \pm 1.04$	$08.33^{d} \pm 0.99$	$06.57^{e} \pm 0.92$	$06.33^{e} \pm 0.89$
II	$17.43^{a} \pm 1.68$	$07.14^{e} \pm 0.55$	$04.31^{f} \pm 0.24$	$03.74^{f} \pm 0.21$	$03.60^{f} \pm 0.26$
III	$20.40^{b} \pm 1.61$	$06.22^{e} \pm 0.31$	$05.02^{f} \pm 0.25$	$04.69^{f} \pm 0.21$	$04.33^{f} \pm 0.21$

Means bearing different superscripts differ significantly ($p<0.01$)

Table 16. Mucin clot quality score of synovial fluid at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$1.08^{a} \pm 0.08$	$2.08^{b} \pm 0.19$	$2.67^{c} \pm 0.31$	$3.42^{e} \pm 0.26$	$3.58^{e} \pm 0.23$
II	$1.17^{a} \pm 0.11$	$2.58^{c} \pm 0.15$	$3.50^{e} \pm 0.19$	$3.92^{f} \pm 0.08$	$4.00^{f} \pm 0.00$

III	$1.08^{a} \pm 0.14$	$3.17^{d} \pm 0.17$	$3.92^{f} \pm 0.08$	$4.00^{f} \pm 0.00$	$4.00^{f} \pm 0.00$

Means bearing different superscripts differ significantly ($p<0.01$)

Table 17. Synovial fluid total protein (g/100 ml) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$6.13^{a} \pm 0.36$	$3.43^{c} \pm 0.16$	$3.16^{d} \pm 0.11$	$2.50^{e} \pm 0.07$	$2.15^{f} \pm 0.04$
II	$5.65^{b} \pm 0.29$	$3.16^{d} \pm 0.10$	$2.14^{f} \pm 0.08$	$2.10^{f} \pm 0.04$	$1.94^{f} \pm 0.03$
III	$6.20^{a} \pm 0.32$	$2.96^{d} \pm 0.15$	$2.03^{f} \pm 0.05$	$1.94^{f} \pm 0.05$	$2.08^{f} \pm 0.03$

Means bearing different superscripts differ significantly ($p<0.01$)

Table 18. Synovial fluid albumin (g/100 ml) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	1.20 ± 0.08	1.26 ± 0.09	1.28 ± 0.05	1.24 ± 0.03	1.14 ± 0.03

II	1.04 ± 0.03	1.08 ± 0.05	1.13 ± 0.04	1.14 ± 0.04	1.03 ± 0.03
III	1.18 ± 0.04	1.22 ± 0.06	1.10 ±0.03	1.06 ± 0.03	1.13 ± 0.02

Means bearing different superscripts differ significantly ($p<0.01$)

Table 19. Synovial fluid globulin (g/100 ml) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$4.93^{a} \pm 0.31$	$2.17^{c} \pm 0.10$	$1.88^{d} \pm 0.06$	$1.26^{e} \pm 0.04$	$1.01^{f} \pm 0.08$
II	$4.61^{b} \pm 0.27$	$2.08^{c} \pm 0.07$	$1.01^{f} \pm 0.05$	$0.96^{f} \pm 0.05$	$0.90^{f} \pm 0.07$
III	$5.02^{a} \pm 0.26$	$1.74^{d} \pm 0.09$	$0.93^{f} \pm 0.03$	$0.88^{f} \pm 0.03$	$0.95^{f} \pm 0.07$

Means bearing different superscripts differ significantly ($p<0.01$)

Table 20. Synovial fluid albumin: globulin ratio at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28

I	$0.24^{a} \pm 0.01$	$0.58^{b} \pm 0.04$	$0.68^{c} \pm 0.03$	$0.98^{d} \pm 0.08$	$1.13^{e} \pm 0.09$
II	$0.23^{a} \pm 0.04$	$0.52^{b} \pm 0.03$	$1.12^{e} \pm 0.05$	$1.19^{e} \pm 0.19$	$1.14^{e} \pm 0.18$
III	$0.24^{a} \pm 0.04$	$0.70^{c} \pm 0.07$	$1.18^{e} \pm 0.05$	$1.20^{e} \pm 0.03$	$1.19^{e} \pm 0.03$

Means bearing different superscripts differ significantly ($p<0.01$)

Table 21. Synovial fluid alkaline phosphatase (IU/100 ml) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$440.25^{a} \pm 3.46$	$234.42^{d} \pm 12.91$	$182.08^{e} \pm 8.69$	$156.17^{e} \pm 7.39$	$105.67^{f} \pm 6.63$
II	$609.83^{b} \pm 5.47$	$184.17^{e} \pm 12.58$	$160.42^{e} \pm 7.19$	$90.33^{f} \pm 2.54$	$94.67^{f} \pm 4.73$
III	$381.58^{c} \pm 21.83$	$157.92^{e} \pm 10.19$	$87.50^{f} \pm 3.96$	$83.25^{f} \pm 3.65$	$89.08^{f} \pm 3.00$

Means bearing different superscripts differ significantly ($p<0.01$)

Table 22. Serum-synovial fluid glucose difference (mg/100 ml) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Parameter	Day 1	Day 7	Day 14	Day 21	Day 28
	Sr. glucose	59.35 ± 0.31	59.54 ± 0.42	56.34 ± 0.49	56.08 ± 0.51	56.43 ± 0.39
I	Sy. glucose	14.60^{a} ± 0.70	35.68^{b} ± 1.14	39.93^{b} ± 1.77	45.02^{c} ± 1.30	50.05^{d} ± 2.44
	SSGD	44.75^{a} ± 0.88	23.87^{b} ± 1.27	19.41^{b} ± 1.93	14.15^{c} ± 1.17	8.68^{d} ± 1.37
	Sr. glucose	59.64 ± 0.45	57.32 ± 0.34	58.15 ± 0.55	60.27 ± 0.38	58.97 ± 0.34
II	Sy. glucose	19.54^{a} ± 0.77	40.85^{b} ± 1.26	45.96^{c} ± 1.32	52.78^{d} ± 0.83	52.27^{d} ± 1.09
	SSGD	39.44^{a} ± 0.92	16.47^{b} ± 1.31	12.19^{c} ± 1.36	7.38^{d} ± 0.69	6.7^{d} ± 1.02
	Sr. glucose	54.44 ± 0.49	53.28 ± 0.50	50.56 ± 0.44	55.06 ± 0.43	54.02 ± 0.40
III	Sy. glucose	11.78^{a} ± 0.77	38.33^{b} ± 1.62	44.11^{c} ± 0.66	46.93^{c} ± 0.37	45.92^{c} ± 1.20
	SSGD	42.65^{a} ± 1.00	15.28^{b} ± 1.35	8.45^{d} ± 0.58	8.17^{d} ± 1.23	7.35^{d} ± 1.29

Means bearing different superscripts within a row differ significantly ($p<0.01$)

Sr.- Serum; Sy.- Synovial fluid SSGD – Serum synovial fluid glucose difference

Table 23. Synovial fluid total nucleated cell count (1000/mm^3) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Day 1	Day 7	Day 14	Day 21	Day 28
I	$33.12^{a} \pm 2.30$	$14.39^{b} \pm 2.26$	$6.60^{c} \pm 0.55$	$3.39^{d} \pm 0.27$	$2.96^{d} \pm 0.12$
II	$30.89^{a} \pm 1.90$	$8.07^{c} \pm 0.30$	$3.64^{d} \pm 0.20$	$0.99^{e} \pm 0.06$	$1.02^{e} \pm 0.05$
III	$38.17^{a} \pm 1.26$	$6.31^{c} \pm 0.47$	$0.77^{e} \pm 0.03$	$0.79^{e} \pm 0.08$	$0.72^{e} \pm 0.05$

Means bearing different superscripts differ significantly ($p<0.01$)

Table 24. Synovial fluid differential cell count (%) at different time intervals in various groups of animals (Mean ± S.E.)

Group	Cell	Day 1	Day 7	Day 14	Day 21	Day 28
I	PMN	$90.95^{a} \pm 04.98$	$54.48^{b} \pm 13.34$	$31.52^{c} \pm 12.94$	$18.76^{d} \pm 07.97$	$10.29^{e} \pm 05.81$
	MN	$09.05^{a} \pm 04.98$	$45.52^{b} \pm 13.34$	$68.48^{c} \pm 12.94$	$81.24^{d} \pm 07.97$	$89.71^{e} \pm 05.81$
II	PMN	$91.86^{a} \pm 05.53$	$35.19^{b} \pm 05.07$	$21.38^{c} \pm 04.12$	$9.52^{e} \pm 03.27$	$07.38^{e} \pm 03.73$
	MN	$08.14^{a} \pm 05.53$	$64.81^{b} \pm 05.07$	$78.19^{c} \pm 03.33$	$90.67^{d} \pm 03.54$	$93.10^{e} \pm 03.57$
III	PMN	$88.95^{a} \pm 05.75$	$25.71^{c} \pm 05.48$	$10.38^{e} \pm 03.49$	$8.52^{e} \pm 03.79$	$07.86^{e} \pm 02.99$
	MN	$11.05^{a} \pm 05.75$	$74.29^{b} \pm 05.48$	$84.62^{c} \pm 03.49$	$89.67^{d} \pm 03.79$	$93.57^{e} \pm 02.99$

Means bearing different superscripts within a row differ significantly ($p<0.01$)

Table 25. Synovial fluid cytology

A.No.	Cytology	Gram's staining	Culture
1.	Degenerate neutrophils with intracytoplasmic cocci (Plate 10)	+ ve cocci, intracellular	β- haemolytic *Streptococcus spp.*
2.	Fibrin, heavy neutrophil	–ve rods, extracellular	*Pseudomonas spp.*
3.	Neutrophil, fibrin and extracellular cocci	+ ve cocci, extra cellular	Negative
4.	Heavy neutrophils	-	Negative
5.	Heavy neutrophils	–ve rods, extracellular	Negative
6.	Fibrin, neutrophils and a few macrophages	+ ve cocci extracellular	*Staphylococcus*
7.	Heavy neutrophils	+ ve cocci, intracellular	*Staphylococcus spp.* – coagulase positive
8.	Heavy neutrophils	-	Negative
9.	Neutrophils	–ve rods, intracellular	*Pseudomonas spp.*
10.	Eosinophilic granular debris, erythrocytes and degenerate neutrophils	-	Negative
11.	Degenerate neutrophils, mononuclear cells and *Streptococci* (Plate 11)	+ ve cocci, extracellular	β-haemolytic *Streptococcus spp.*
12.	Degenerate neutrophils and few macrophages (Plate 12)	-	Negative
13.	Predominantly neutrophils	–ve rods, extracellular	*Escherichia coli*
14.	Heavy neutrophils	-	Negative
15.	Neutrophils	-	Negative
16.	Mononuclear cells with phagocytosed rods	–ve rods intracellular	*Salmonella spp.*
17.	Predominantly neutrophils	–ve rods extracellular	*Escherichia coli*

18.	Mononuclear cells with phagocytosed cocci (Plate 13)	+ ve cocci intracellular	Non β- haemolytic *Streptococcus spp.*
19.	Intact neutrophils	–ve rods extracellular	*Pseudomonas spp.*
20.	Predominantly neutrophils	–ve rods extracellular	Negative
21.	Degenerate neutrophils	–ve rods intracellular	*Escherichia coli*
22.	Neutrophils	+ ve cocci intracellular	β-hemolytic *Streptococcus spp.*
23.	Degenerate neutrophils, basophilic to eosinophilic synoviocytes	+ ve cocci extracellular	*Staphylococcus spp.* – coagulase negative

Contd.,

A.No.	Cytology	Gram's staining	Culture
24.	Predominantly neutrophils (Plate 14) and RBC	+ve cocci intracellular	Negative
25.	Predominantly neutrophils	-	Negative
26.	Predominantly neutrophils and few yeast colonies (Plate 15)	+ ve cocci extracellular	β-haemolytic *Streptococcus spp.*
27.	Degenerate neutrophils	+ ve cocci extracellular (Plate 16)	Non β-haemolytic *Streptococcus spp.*
28.	Fibrin, degenerate neutrophil and few RBC	-	Negative
29.	Neutrophils and macrophage in equal number	–ve rods extracellular (Plate 17)	*Escherichia coli*
30.	Predominantly neutrophils with few mononuclear cells	–ve rods extracellular	*Escherichia coli*
31.	More erythrocytes	+ ve cocci extracellular	β-haemolytic *Streptococcus spp.*
32.	RBC, with intact and degenerate neutrophils,	-	Negative

	basophilic to eosinophilic synoviocytes		
33.	Intact neutrophils	+ ve cocci intracellular	β-haemolytic *Streptococcus spp.*
34.	Predominantly neutrophil	-	Negative
35.	Degenerate neutrophils, a few cocci and yeast colonies	+ ve cocci intracellular, yeast colonies	Negative
36.	Neutrophils and mononuclear cells	+ ve cocci extracellular	Non β-haemolytic *Streptococcus spp.*

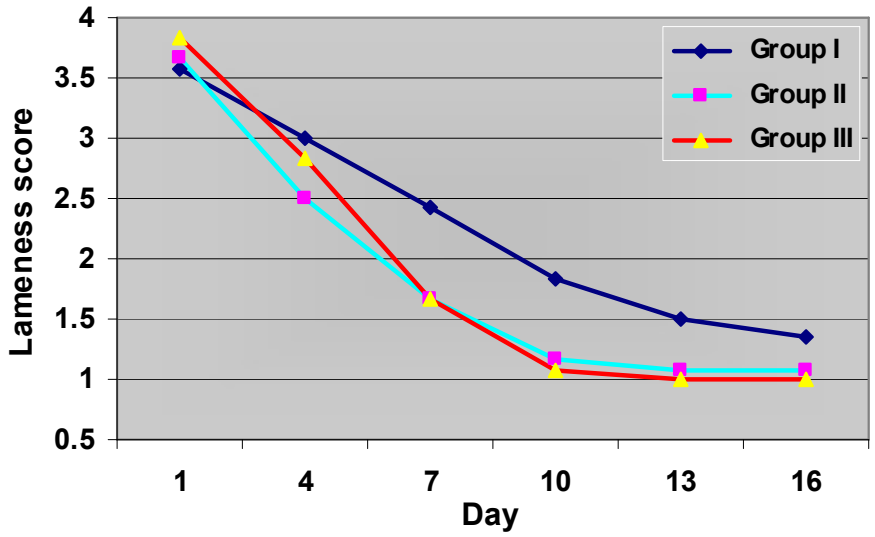


Figure 6. Mean lameness score at different time intervals in various groups

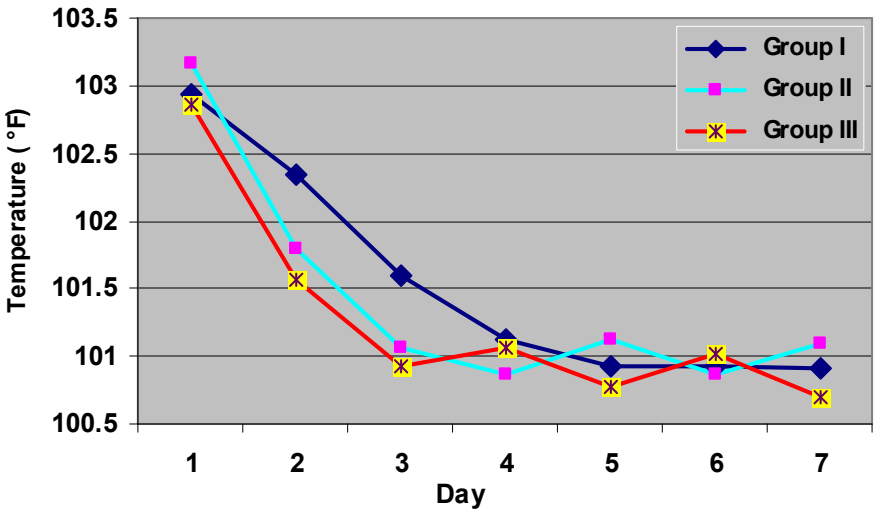


Figure 7. Mean rectal temperature (°F) at different time intervals in various groups

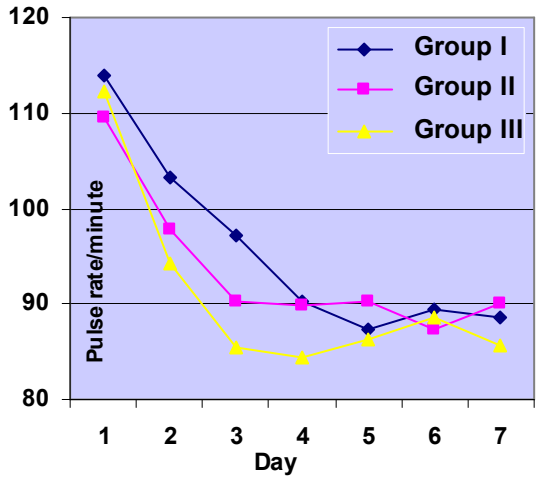


Figure 8. Mean pulse rate/minute at different rate/minute time intervals in various groups in

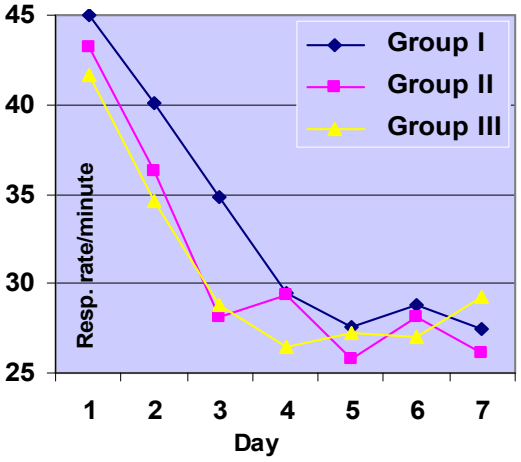


Figure 9. Mean respiratory at different time intervals various groups

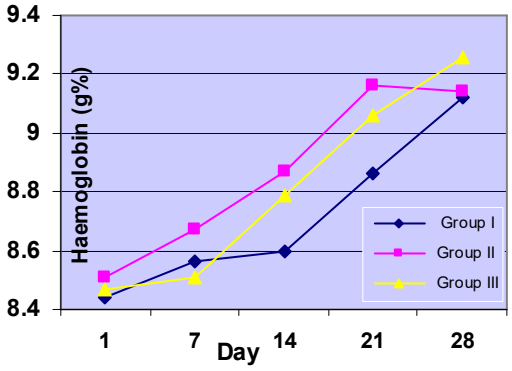


Figure 10. Mean haemoglobin (g%) in blood at

at different time intervals in various groups

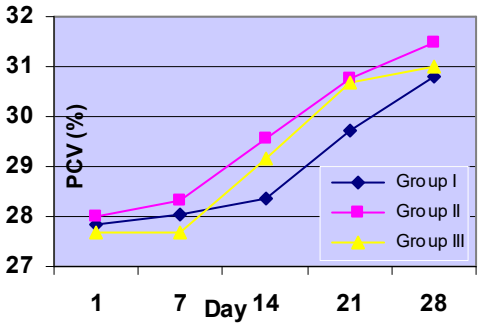


Figure 11. Mean PCV (%) in blood

different time intervals in various groups

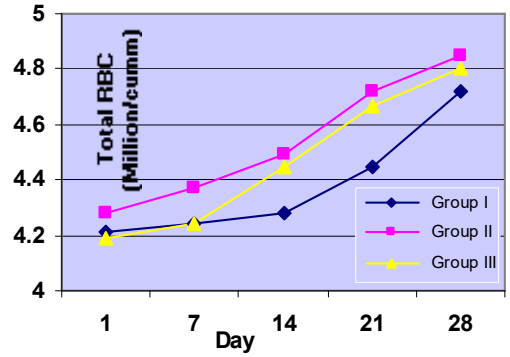


Figure 12. Total RBC count (million/cumm) blood at different time intervals in

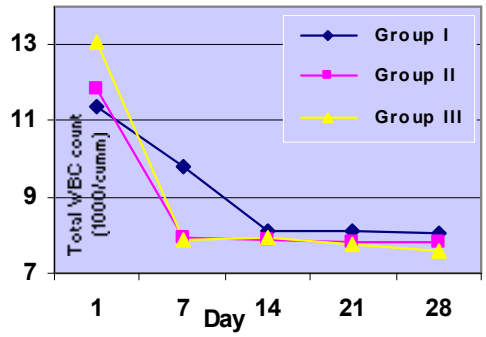


Figure 13. Mean total WBC count (1000/cumm) at different

various groups

time intervals in various groups

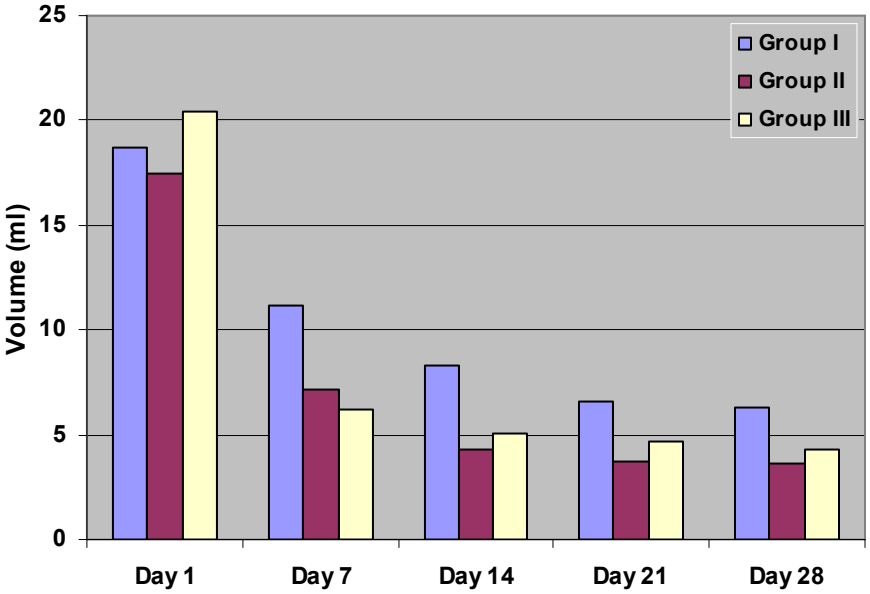


Figure 14. Mean synovial fluid volume (ml) at different time intervals in various groups

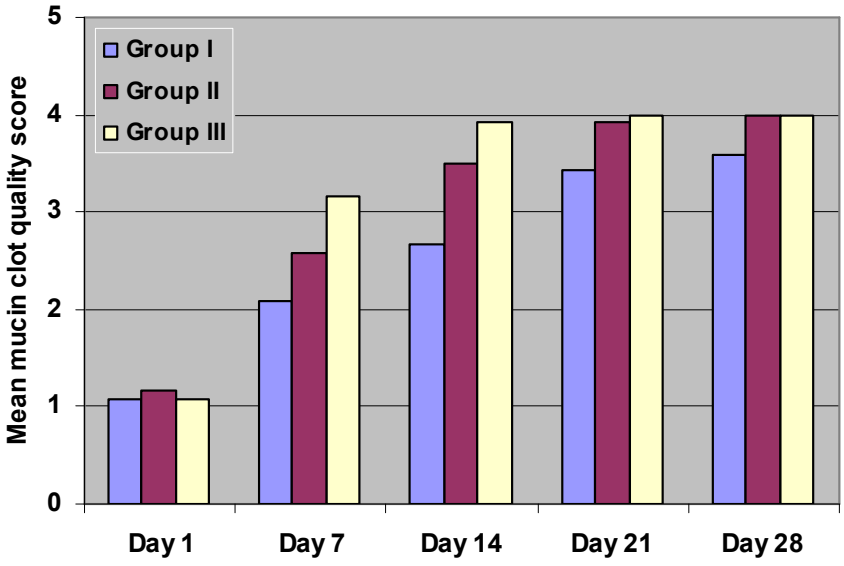


Figure 15. Mean mucin clot quality score at different time intervals in various groups

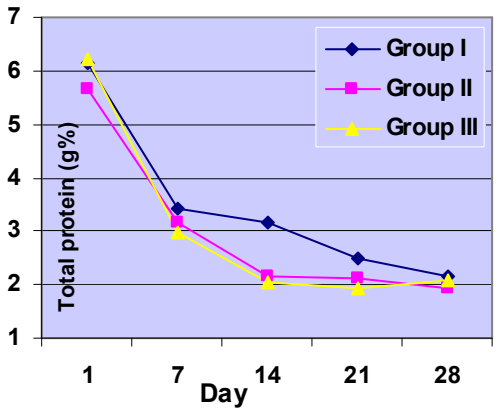


Figure 16. Synovial fluid total protein (g%) at different time intervals in various groups

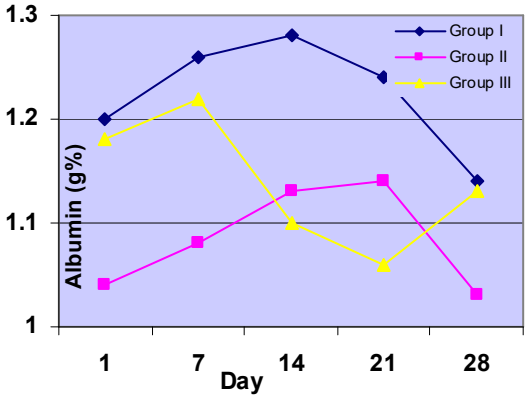


Figure 17. Synovial fluid albumin (g%) at different time intervals in various groups

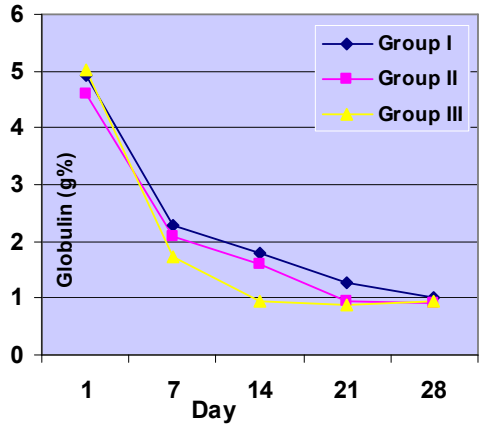


Figure 18. Synovial fluid globulin (g%) at different time intervals in various groups

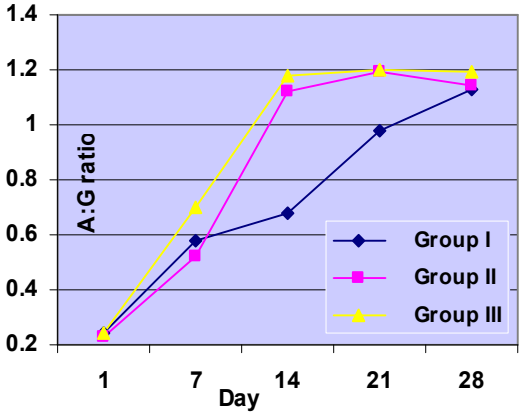
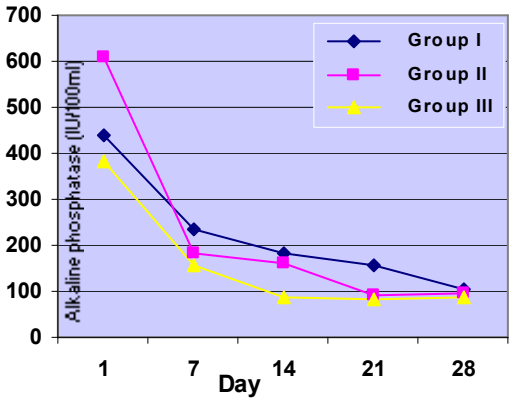


Figure 19. Synovial fluid albumin : globulin ratio at different time intervals in various groups



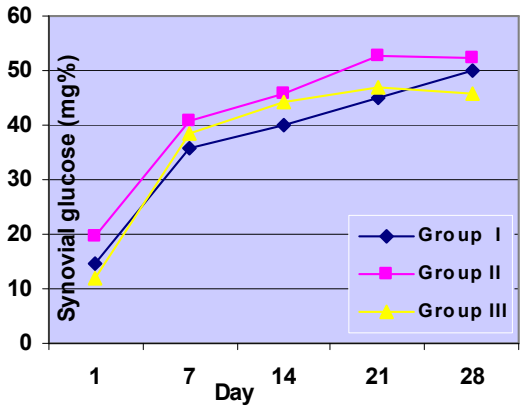


Figure 20. Synovial fluid alkaline phosphatase (IU/100ml) at different time intervals in various groups

Figure 21. Synovial fluid glucose (mg%) at different time intervals in various groups

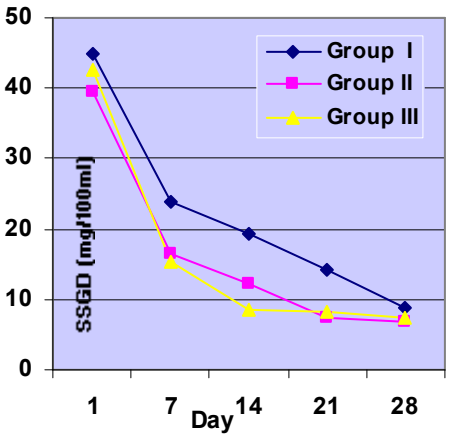


Figure 22. Serum-synovial fluid glucose difference (SSGD) (mg/100ml) at different time intervals in various groups

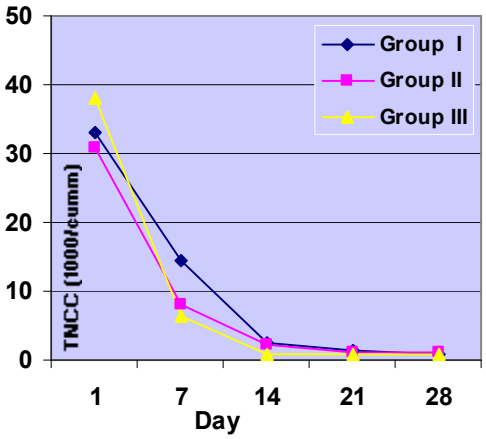


Figure 23. Mean synovial fluid total nucleated cell count (1000/cumm) at different time intervals in various groups

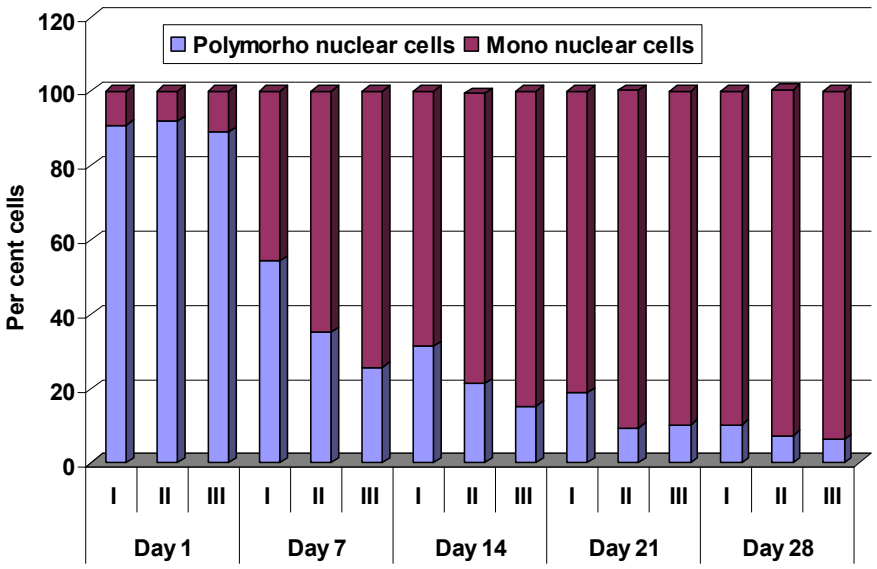


Figure 24. Mean synovial fluid differential leucocyte count (%) at different time intervals in various groups

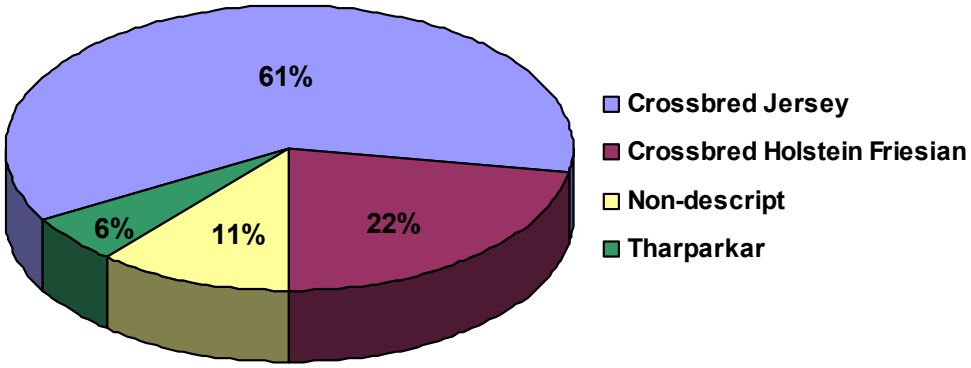


Figure 2. Breed wise distribution of septic arthritis in cattle

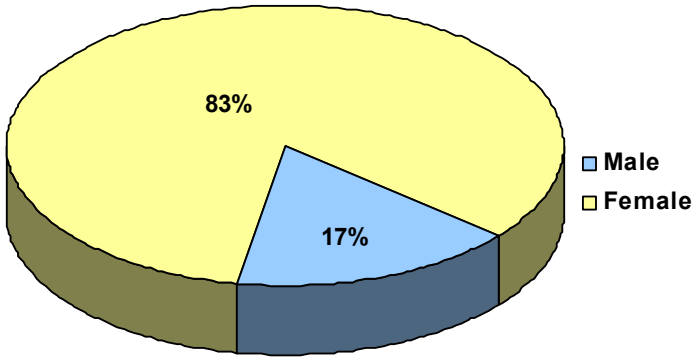


Figure 3. Gender wise distribution of septic arthritis in cattle

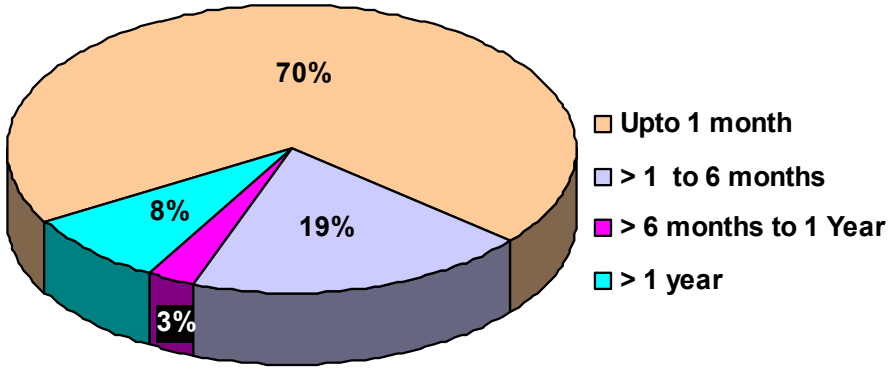


Figure 4. Age wise distribution of septic arthritis in cattle

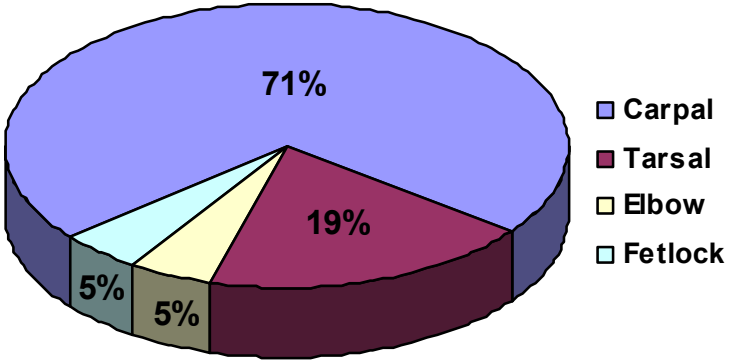


Figure. 5. Joint wise distribution of septic arthritis in cattle