PATHOLOGY OF SELECTED ENDOCRINE GLANDS IN TESTICULAR DISORDERS IN BULLS

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DECLARATION

I hereby declare that this thesis entitled **"PATHOLOGY OF SELECTED ENDOCRINE GLANDS IN TESTICULAR DISORDERS IN BULLS"** is a bonafide record of research work done by me during the course of research and that this thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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Certified that the thesis entitled **"PATHOLOGY OF SELECTED ENDOCRINE GLANDS IN TESTICULAR DISORDERS IN BULLS"** is a record of research work done independently by **Dr. B. Jothish Kumar**, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

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Introduction

1. INTRODUCTION

"The Bull is half of the herd" statement confirms the importance of breeding bull in animal population. The sire of the herd plays a major role for producing optimum fertility and genetic potentiality in the herd. Bull fertility is of paramount importance in cattle production.

A major percentage of young bulls are being culled every year due to poor semen quality. The sterility problems in bulls are mainly attributed to genetic, environmental, nutritional and endocrine factors. The association of endocrine gland disorders with the poor reproductive performance of the bulls have been well documented.

After birth, a bull enters a period of infancy during which the reproductive organs are relatively quiescent. This is followed by profound changes of hypothalamic, pituitary, and gonadal function that culminate in puberty. Puberty is the time when a bull becomes capable of participating in reproduction. There is an inductive relationship between the male gonad and the endocrine glands like pituitary, thyroid and adrenal gland.

Abnormalities of the male genital system may be expressed in the categories familiar to pathologists such as congenital anomalies, degenerative alterations, inflammatory processes, or neoplastic conditions. Most of these pathological changes are well described and reasonably understood.

Sperm fertility is the final manifestation of a complex series of events, each of which are carefully controlled and modified by the pineal, hypothalamus, pituitary, testes, epididymes and a feedback mechanism involving various hormones. In the testis, LH (Luteinizing Hormone) stimulates testosterone secretion and FSH (Follicle Stimulating Hormone) is important in the initiation and maintenance of spermatogenesis. The secreted testicular androgen

testosterone and its activated form of dihydrotestosterone (DHT) act on target end organs for the development of male secondary sexual characteristics. Sertoli cell products may serve as the mediators of interaction between germ cells, Leydig cells, peritubular myoid cells and the Sertoli cells of the testis.

The hereditary and non-hereditary abnormalities affecting the testis are prone to cause male infertility or sterility. Testicular disorders are mainly due to acquired causes than the congenital or hereditary causes. The epithelium of seminiferous tubules is highly sensitive to injury which may affect spermatogenesis and fertility. The common pathological conditions affecting testis such as aplasia, hypoplasia, testicular degeneration, testicular fibrosis, testicular calcification, orchitis and testicular tumour have been reported in bulls. In India, sporadic cases of testicular atrophy, degeneration including fibrosis and calcification and orchitis has been documented in bovines (Rao and Rao, 1979; Chaudhuri *et al.*, 1982; Pandit and Pandey, 1992).

Endocrine gland lesions are sometimes associated with gonadal change. Endocrine glands such as pituitary, thyroid and adrenal can influence the testicular function in various domestic animals.

Pituitary gonadotropins like follicle stimulating hormone and luteinizing hormone play an important role on the male reproduction. The luteinizing hormone controls the Leydig cell production of testosterone and the follicle-stimulating hormone controls the secretary activity of the Sertoli cell.

The relationship between the thyroid and the male gonad has significance in male infertility. Thyroid hormones are responsible for the testicular growth, spermatogenesis and libido.

Testosterone is the most important naturally occurring androgen, having widespread effects on the development of both sexual and nonsexual tissues in the body. More than 90 per cent of testosterone synthesis in the male occurs in

the Leydig cells of the testes while the remainder is produced in the adrenal glands.

Many diseases of the endocrine glands are characterised by dramatic functional disturbances and characteristic clinicopathological alterations affecting one or several body system.

Although endocrine diseases of animals are frequently encountered in veterinary practice, they are only scarcely reported in the literature and therefore they may go undiagnosed by the veterinary practitioner. Hence the present study is undertaken with the following objectives:

1. Identify the testicular lesions of the bull.
2. Study the pathological lesions of pituitary, thyroid and adrenal gland of the bulls with testicular disorders.
3. Correlate the pathological changes in the endocrine glands with testicular lesions.

Review of Literature

2. REVIEW OF LITERATURE

2.1 ANATOMY OF THE TESTIS

The testicle of the bull is oval in shape and closely covered by a thin serous membrane, the tunica vaginalis propria. Beneath this structure are a dense, thick connective tissue capsule, and the tunica albuginea. The septa radiate to the mediastinum to form the lobules of the testis. Within these lobules are the seminiferous tubules, which are lined by germinal epithelium that produces spermatozoa. Between the seminiferous tubules are the islands of interstitial or Leydig cells. The straight tubules enter the rete testis, a structure of anastomosing spaces located in the mediastinum testis (Roberts, 1971).

2.2 TESTICULAR DISORDERS

2.2.1 Anomalies of Development

2.2.1.1 Cryptorchidism

2.2.1.1.1 Prevalence

Blackshaw and Samisoni (1967) reported the cryptorchid testis in six mature polled Merino rams and the cases detected were unilateral.

Roberts (1971) stated that cryptorchidism occurs in all domestic species, seen most commonly in stallions, boars, dogs, less commonly in rams and bucks, uncommonly in cattle, and rarely in cats.

Saunders and Ladds (1978) examined the genital tracts of 968 slaughtered bulls derived from 24 herds in North Queensland and Northern Territory of Australia for defects of congenital or developmental nature. The overall occurrence of lesions was seven per cent. Involvement of cryptorchidism was in six cases and it was unilateral. The retained testis was located in the abdomen in

five bulls while in the remaining bulls it was found on the left side at the external inguinal ring. Two affected bulls were from the same herd.

Logue and Greig (1986) stated that cryptorchidism was most commonly seen in the young pig and ram. The condition was considered to be heritable and in the unilateral state often associated with normal fertility.

Reddy *et al.* (1999) reported seminoma in a cryptorchid Hallikar bull, aged four years. On examination, the bull was found to be a unilateral cryptorchid and the right testis in the scrotum was enlarged more than double in size.

Onu *et al.* (2002) reported six cases of cryptorchidism in rams in the age group of nine to twenty four months in Sokoto, Nigeria among 1020 abnormalities of genitalia.

Regassa *et al.* (2003) conducted a detailed study of abnormalities of genitalia in 404 bucks belonging to two breeds and 167 rams of two breeds, collected from abattoir house in Ethiopia. In the affected bucks, 18 had unilateral and 4 had bilateral cryptorchidism. In those with unilateral retention, 10 involved the right testicle and 8 the left testicle. Three cases of unilateral cryptochidism were encountered in rams.

2.2.1.1.2 Gross Pathology

Cryptorchid testes were uniformly small, being about 10 per cent of the weight of the corresponding scrotal organ, but their epididymis were relatively larger. In all the animals the right testis was abdominal and in the right sub lumbar region close to the right iliac vessels (Blackshaw and Samisoni, 1967).

The cryptorchid testis might be located in the inguinal canal or subcutaneously at the external inguinal ring. Sometimes epididymis might be located in the inguinal canal, while testis was in the abdomen. Cryptorchid testis was hypoplastic. Affected testes were small and firm to hard (Jubb *et al.*1993).

Acland (1995) described that the gross appearance of the cryptorchid testis prior to puberty and observed it as normal. After puberty, the testis becomes progressively smaller and fibrotic. Epididymal differentiation was coordinated with testicular descent.

2.2.1.1.3 Histopathology

Blackshaw and Samisoni (1967) studied histopathology of the cryptorchid testis on frozen sections. They recorded normal spermatogenesis in the scrotal testes whereas the cryptorchid organs had small-undifferentiated tubules in a considerable amount of connective tissue. The cryptorchid epididymes, although was only two third of the normal size were devoid of spermatozoa.

Ezeasor and Singh (1987) described the morphological features of the cryptorchid testes in different age groups of dwarf goats. In 1-month-old goats, the seminiferous epithelium comprised of columnar Sertoli cells with uniformly stained cytoplasm and many pale-stained pre-spermatogonia. The characteristic lesion in three months old goats consisted of dense-staining elliptical masses in the apical cytoplasm, and a few spermatogonia at the base of the epithelium. The seminiferous epithelium of the pubertal and adult goats assumed mature characteristics of the germ cell modifications associated with Sertoli cells. The basal and supranuclear cytoplasm of Sertoli cells often contained osmiophilic (lipid) droplets.

Jubb *et al.* (1993) described histological features of the cryptorchid testis. The lesions were similar to that of severe form of hypoplasia. Intratubular concrements similar to those seen in testicular hypoplasia were present. Marked fibrosis, especially of the tunics, was the feature of cryptorchid testes of old animals. In unilateral cases, compensatory hypertrophy of the normal testis was noticed.

Acland (1995) described that the cryptorchid testis were atrophied and characterised by interstitial collagen deposition, hyaline thickening of the tubular

basement membranes, atrophy of germinal epithelium with only a few spermatogonia and Sertoli cells. Interstitial cells appeared to be relatively more in cases of descended testes.

2.2.1.2 Aplasia of the Testis

Chaudhuri *et al.* (1982) observed eight cases of absence of testis out of 197 non-descript buffaloes slaughtered. Bilateral absence of testis was noted in three cases while absence of right testis was found in three and that of left in two animals. Grossly, it was characterised by one or two very small fibrous masses near the apex of the head of the respective epididymis. The cut surface of such mass revealed reddish to yellowish colour. Histologically the masses contained irregular spaces lined by flattened epithelial cells. The central portion of such mass was somewhat hyalinized.

2.2.1.3 Testicular Hypoplasia

2.2.1.3.1 Prevalence

In a survey study, Galloway (1961) recorded two cases of testicular hypoplasia on 326 bulls examined with the problem of bull wastage. The condition was noted in a young Jersey and Hereford bulls. It was bilateral in the Jersey bull and unilateral in a three year old Hereford bull.

Rao *et al.* (1966) reported unilateral left-sided testicular hypoplasia in a crossbred bull. The scrotum was distorted contained one normal and one under-sized testicle.

Kodagali *et al.* (1971) reported nine cases of hypoplasia of testis in Gir bulls. Amongst these, six were left sided, two were right sided and one was bilateral hypoplasia.

Benjamin *et al.* (1974) reported testicular hypoplasia in 14 cases out of 203 buffalo bulls examined at local municipal abattoir. Out of the 14 cases, eight

were bilateral, two were of total right-sided and the remaining were of partial left side hypoplasia.

Saunders and Ladds (1978) examined the genitalia of 968 slaughtered bulls, derived from 24 herds in Australia for congenital or development defects. The overall occurrence of hypoplasia was seven per cent. Involvement of testicular hypoplasia in two of these bulls was unilateral.

Dunn *et al.* (1980) reported bilateral testicular hypoplasia and azoospermia in a polled Hereford bull with good libido. The karyotype of this bull was 61, XXY and simulated human Klinefelters syndrome.

Chaudhuri *et al.* (1982) recorded nine cases of testicular hypoplasia out of 197 non-descript buffalo bull's genitalia that were examined at the Municipal abattoir, in which two cases were bilateral, four cases were unilateral right and three cases were unilateral left.

Ahmad *et al.* (1988) reported various forms of germinal tissue hypoplasia of the testis in 22 buffalo bulls in a herd of 135 buffalo bulls at the Semen Production Unit, Qadirabad, Pakistan. Out of 22 infertile buffalo bulls that were slaughtered, five cases were of bilateral with complete, partial or incomplete hypoplasia. Incomplete bilateral hypoplasia was seen in one case and bilateral partial hypoplasia was in another case respectively.

Madrid *et al.* (1988) studied scrotal circumference, seminal characteristics and testicular lesions of 37 yearlings Angus bulls and out these one bull had small testis with poor semen quality.

Galloway *et al.* (1992) studied the prevalence of hypoplasia of testes in poll Dorset rams born in the Southeast Australia. Out of 437 rams, 12 rams were with one large and one small testicle and 11 were with two small testicles.

2.2.1.3.2 Gross Pathology

Rao *et al.* (1966) reported testicular hypoplasia in a crossbred bull with conspicuous reduction in size and weight of the testicles. The left testicle was found to be markedly smaller than the right one and did not descend far down to the bottom of the scrotum. There were no adhesions and both the testicles were freely movable. The consistency of the left testicle was slightly firmer while the right one was normal and healthy.

The hypoplastic testis was observed on gross examination only after puberty and the size may be as small as one quarter of normal sizes and freely movable within scrotum. Consistency of the hypoplastic testis was closer to normal than that of the degenerated testes, and the cut surface usually tends to bulge on sectioning (Jubb *et al.*, 1993).

Acland (1995) described that the hypoplasia of the testis often was not apparent until after puberty. Unilateral hypoplasia was more common than bilateral hypoplasia, but this could be a reflection of the relative ease of recognizing a size abnormality when most of the causes would logically seem to act systemically and, therefore, produce the lesion bilaterally. The hypoplastic testis generally range in size from one quarter of normal to near normal. Consistency of a hypoplastic testis was near to normal.

2.2.1.3.3 Histopathology

Histologically, the testes of two bulls showed incomplete spermatogenesis in majority of the seminiferous tubules and the process appeared to have been arrested at the spermatid stage, with some of the spermatids being free in the lumina of the seminiferous tubules. Basement membrane of some of the seminiferous tubules was found slightly thickened. In total hypoplastic condition the germinal cells were completely absent. The lumina showed degenerated seminal products, where as some seminiferous tubules were empty. A single layer of Sertoli cells lined the basement membrane in majority of the tubules.

The basement membrane of some of the tubules showed hyalinisation. The peri-tubular space was occupied by fibrous connective tissue. In some areas there was aggregation of interstitial cells (Kodagali *et al.*, 1971).

Benjamin (1974) described the histological changes in hypoplastic testis of buffalo bulls, which showed different degrees of degeneration. There was increased amounts of fibrous tissue. In extreme cases the whole architecture was replaced by fibrous tissue without revealing any seminiferous tubules or Leydig cells. In mild cases, a few seminiferous tubules showed degeneration and desquamation of the germinal epithelium with no signs of mitosis. Sertoli cells were seen in between spermatogonia cells in a few tubules. The septi testis was thickened considerably. There was sclerosis of vessels underlying the tunica albuginea. In extensive damage, the seminiferous tubules did not contain the germinal epithelium lining, that was vacuolated, or the epithelium was missing altogether. The nuclei of the germinal epithelium were some times clumped together and were seen in the lumen of a seminiferous tubule. The Leydig cells were not seen. A few seminiferous tubules were completely occluded with sperms. In such tubule, the lining epithelium was absent and the basement membrane was thickened. The lumina contained sperms and cellular debris.

Dunn *et al.* (1980) described that the hypoplastic testis was composed predominantly of normal appearing Leydig cells and degenerated seminiferous tubules. The vast majority of seminiferous tubules consisted of irregular shaped masses of collagenous connective tissue. Few tubules contained Sertoli cells but no germinal cells were evident.

Veeramachneni *et al.* (1986) described the changes of seminiferous epithelium and interstitium in the hypoplastic testis of beef bulls. The seminiferous epithelium with Sertoli- cells- only tubules was seen without any vacuolation. These tubules were small and usually without a lumen or peritubular thickening. Focal areas of Leydig's cell hyperplasia were marked.

Acland (1995) histologically classified the severity of the hypoplasia by the proportion of hypoplastic tubules scattered through the organ. Hypoplastic tubules had a smaller diameter and were lined by Sertoli cells, few stem cells and spermatogonia. The tubule had a thickened basement membrane surrounded by collagen deposits. In severe hypoplasia, the tubules were small in diameter, with infrequent vacuolation of Sertoli cells and without a thickened basement membrane. In moderate hypoplasia, some tubules were smaller in size and those with normal size showed some differentiation of their seminiferous epithelium. The lumen of tubules also contained cellular debris and multinucleate giant cells. In mild hypoplastic cases, only a few small tubules, lined mostly by Sertoli cells were present; whereas most of the tubules were active. Multinucleate giant cells were present in the tubular lumina.

2.2.2 Testicular Degeneration

2.2.2.1 Prevalence

Galloway (1961) recorded 21.5 per cent cases of testicular degeneration out of 326 bulls examined with problem bull wastage in Australia. An increased incidence was found in the older age groups. 4 per cent of 173 young bulls, 30 per cent of 69 medium bulls and 50 per cent of 84 old bulls had some degree of macroscopic degenerative changes. Within the whole group 43 per cent had unilateral lesions and 57 per cent had bilateral lesions.

Turnbull and McKay (1973) observed testicular degeneration of a two-and-a-half-year-old Santa Gertrudis bull. The right testicle of the bull had been trodden on at least nine months before mating began.

Chaudhuri (1982) revealed two cases of the degeneration of the testis in 197 non-descript buffalo bulls examined at municipal abattoir, Bareilly. Out of two cases one was bilateral and the other was unilateral.

Refsal *et al.* (1983) reported a case of bilateral testicular degeneration in an 18-month-old male goat with problem of fertility.

Ahmad *et al.* (1988) reported the eight cases of testicular degeneration in 22 infertile buffalo bulls in Pakistan.

Kalicharan and Verma (2001) reported the testicular degeneration in 18.04 per cent buffaloes out of 61 river buffaloes slaughtered.

Kavani *et al.* (2001) reported testicular degeneration in a 4-year-old Jersey bull at the Regional Semen Station, Rajkot in Gujarat state. The bull when examined clinically revealed bilaterally atrophic testicles.

2.2.2.2 Gross Pathology

Galloway (1961) classified the degree of degeneration as "Very slight", "Moderate" and "Extensive" on the basis of changes noticed in the tubules.

Jubb *et al.* (1993) described the gross pathology of testicular degeneration in bulls. The testis showed decrease in size and associated with initial swelling and altered consistency. The testis did not bulge on sectioning. Fibrosis and calcification of parenchyma, and thickening of the tunica albuginea were noticed.

Atrophy of the testes, close adhesions to a thickened tunica vaginalis and fluid filled cyst in the head of epididymis were characteristic features observed (Turnbull and McKay, 1973).

In testicular degenerative condition, the testis was reduced in size and soft in consistency in unilateral cases. The cut surface did not bulge and no gross changes were observed in bilateral cases (Chaudhuri *et al.*, 1982).

The most striking gross lesions of testicular degeneration were white, granular foci involving segments of individual seminiferous tubules of both testes. These lesions were located in close proximity to the rete testes and

radiated out into the testicular parenchyma in a plane perpendicular to the rete testes (Refsal *et al.*, 1983).

Ladds (1993) described gross lesions of the testis undergoing degeneration. In early stages the testis was enlarged by oedema and then reduced in size. In early or rapidly progressing degeneration, the testis was soft and flabby, lacked turgor, and the cut surface did not showed evidence of bulging. Distinct wrinkling of the tunica albuginea was present. Decrease in stroma resulted in a decrease in size and there was change of firm consistency. With progression of degeneration and fibrosis, the testis became hard and mineralised. The cut surface of such a testis had a coarse granular appearance.

The degenerated testis will be swollen and softer than normal; followed by progressive shrinkage. The cut surface of degenerated testis bulge slightly. After the acute phase, the consistency became firmer. Small flecks or large areas of calcification were present (Acland, 1995).

Kavani *et al.*. (2001) described that in the testicular degeneration both testicles were smaller with shrivelled epididymis in proportion to the age and body size and the testicular parenchyma did not bulge out on sectioning through the capsule.

2.2.2.3 Histopathology

Galloway (1961) noticed calcification in some of the straight collecting tubules as well as the seminiferous tubules in testicular degenerative condition. Congestion and impaction with sperm were also evident in the seminiferous tubules. Degeneration of the epithelium had occurred with hyalinisation of the central mass of debris, sperm and Sertoli cells. Zones of calcified tubules showed some degree of intratubular fibrosis. There were other areas in the same testicle, which appeared to be producing sperm normally. Cytological abnormalities in the form of vacuolation of spermatocytes, pyknosis and karyorrhexis, giant cell formation were also evident. Atrophy of the tubules were also noticed.

Turnbull and McKay (1973) noticed that the parenchyma of affected testis was completely fibrosed with no functional seminiferous tissue evident. Head and tail of the epididymis showed a mass of degenerated tubules embedded in fibrous tissue. The tubules of the unaffected testis produced sperm but there were many degenerated cells in the lumina and varying degrees of hypertrophy of the interstitial tissue.

In buffaloes, Chaudhuri *et al.* (1982) reported that there was complete degeneration of most seminiferous tubules, the presence of giant cells in some of the tubules and mononuclear cell reaction in the interstitial tissue and around the tubules

Refsal *et al.* (1983) observed that the presence of dense mineralised debris occluding affected seminiferous tubules, accompanied by complete loss of germinal epithelium. Sperm heads were imbedded in the debris. The remainder of the seminiferous tubules had no spermatozoa within their lumen and few spermatids in the germinal epithelium. Some seminiferous tubules showed sloughing of the germinal epithelium into the lumen. No inflammatory response accompanied these lesions.

Veeramachaneni *et al.* (1986) described lesions in testicular degeneration in beef bulls. The testis showed loss of germinal cells, leaving vacuolated epithelium and atrophy of cells in degenerated tubules. Hyperplasia of Leydig's cells was also observed. Sertoli's cell showed degeneration or hypoplasia.

Ahmad *et al.* (1988) described histological changes of testicular degeneration associated with three types of infertility. He classified infertility as bulls without sexual libido; bulls that produced poor quality semen; and bulls in which the quality of semen deteriorated. In the first category it was characterised by extensive peritubular infiltration of mononuclear cells. In the second category, there was degeneration and intertubular fibrosis of varying intensity at different areas of the testes. Fibrosis and inflammatory reactions, fibrosis of

intertubular spaces and tubular stasis were other features. In the third category, testicular degeneration and marked intertubular fibrosis associated with tubular stasis were seen.

Acland (1995) described microscopical lesions of the testicular degeneration. The predominant lesions were small seminiferous tubules with thickened basement membrane which had decreased numbers of seminiferous epithelial cells, fenestrated tubular lining, intratubular giant cells, and interstitial fibrosis. The degree of vacuolation of the lining cells was more severe in degeneration than in hypoplasia. Basement membrane in degeneration had much more irregular outline. Calcification of intratubular cellular debris, tubular basement membranes, or the interstitium was also noticed.

Degeneration of majority of seminiferous tubules showed partial to complete loss and, or desquamation of cellular structures. The lumen contained either pink homogenous proteinaceous material mixed with degenerated cells or completely calcified masses. Increased cellular proliferation and mild to moderate degree of fibrous tissue proliferation were noticed in the interstitial tissue. Calcified masses were evident around seminiferous tubules (Kavani *et al.*, 2001).

2.2.3 Testicular Fibrosis

2.2.3.1 Prevalence

McEntee (1958) attributed the ventral testicular fibrosis to degenerative vascular changes most commonly seen in older bulls.

Turnbull and Mckay (1973) reported fibrosis of the right testis in a Santa Gertrudis bull.

Rao and Rao (1979) reported the bilateral testicular fibrosis in a young Red Dane Bull.

2.2.3.2 Gross Pathology

The affected testicles were firm in consistency and appeared to be one-third of the normal size. The testes were slightly flaccid and lacked the normal tension (Fincher *et al.*, 1942).

On gross examination fibrosed testes were small in size and firm in consistency. The cut surface was dark brown in colour interspersed with grey white streaks appeared in the parenchyma (Rao and Rao, 1979).

Ladds (1993) described varying degrees of fibrosis from mild diffuse change with increased consistency to extreme fibrosis where the testis is small and hard with an obvious, grossly visible stroma. Ventral testicular fibrosis in old bulls was attributed to degenerative vascular changes.

2.2.3.3 Histopathology

Fincher *et al.* (1942) examined the histological changes in fibrosis and tubular atrophy. The tubules consisted of a single layer of pale staining cells with prominent nucleoli. The basement membranes of the tubules were thickened by fibrous tissue. Broad bands of dense connective tissue separated the tubules. Thick-walled vessels were prominent in this tissue. Circular areas of progressing fibrosis were present where tubules were obliterated and replaced by connective tissue. Groups of interstitial cells were present in the fibrous tissue. There were occasional small groups of tubules in which spermatogenesis was taking place.

Humphrey and Ladds (1975) found a higher proportion of stromal tissue in the ventral part of normal bull testes, and demonstrated testicular fibrosis, which they opined as the histological feature of ageing.

Rao and Rao (1979) described histopathological features in testicular fibrosis. There was marked increase of interstitial tissue together with marked degenerative changes in the germinal epithelium of the seminiferous tubules. In certain areas the tubules could be observed with several layers of germinal

epithelium, although mostly degenerated and usually without formation of sperm. In certain areas, lymphocytic infiltration in the intratubular connective tissue was observed.

Ladds (1993) stated that the ventral testicular fibrosis in old bulls is attributed to degenerative vascular changes. As the germinal epithelium is avascular, progressive fibrosis may also be consequent to basement membrane changes restricting diffusion from underlying vessels.

2.2.4 Testicular Calcification

2.2.4.1 Prevalence

Maurya *et al.* (1968) reported 43 cases of testicular calcification out of 450 adult buffalo-bulls of non-descript breed obtained from the slaughterhouses. Of these 40 were bilateral and 3 were unilateral.

Venkataswamy and Pattabiraman (1970) recorded the calcareous testicular degeneration in an adult Jersey bull brought for treatment of oligospermy associated with necrospermy.

Damodaran and Parthasarathy (1972) reported a case of testicular calcification in Jersey bull, aged five and half years, in Tamil Nadu. The animal was reported to have lost its libido and was not producing good quality semen.

Barnabe *et al.* (1973) encountered a case of testicular calcification in a 3-year-old Santa Gertrudis bull. On palpation the testes were clearly asymmetrical. The left testis was affected.

Kaikini and Patil (1978) revealed unilateral testicular calcification in one healthy Berari buffalo bull aged 11 years.

Chaudhari *et al.* (1982) recorded 61 cases of testicular calcification in 197 cases examined. Bilateral calcification was observed in 39 cases where as

calcification of either right or left testis was found in 10 and 12 cases respectively.

Pandit and Pandey (1992) reported a case of testicular calcification in a seven year old Haryana bull presented with a history of scrotal injury.

Kalicharan and Verma (2001) reported 9.84 per cent of degeneration of seminiferous tubule with calcification in buffalo bulls out of a total of 61 pairs of male gonads, collected from slaughterhouse at Bareilly. A total of 38 organs had histopathological changes.

2.2.4.2 Gross Pathology

Maurya *et al.* (1968) found that the tunica albuginea in the affected testicle was thickened and the glistening appearance lost. Extensive deposition of calcium in the affected testicles was noticed and had a distinctive whitish appearance in 17 cases. Of other 26 cases, the distribution of calcium was only focal and limited to superficial areas.

Venkataswamy and Pattabiraman (1970) observed that the affected testicle was smaller than the unaffected one. The tunica albuginea was thickened and wrinkled with loss of its glistening appearance. Sagittal section showed a rough surface with grating of mid-testicular portion that was coarsely granular, opaque and flecked in appearance. The periphery was apparently normal containing miliary white granules.

Damodaran and Parthasarathy (1972) reported a case of testicular calcification in bull. The left testis weighed 470 gms and the right testis was 210 gms. Grossly the testis was yellowish-white in colour, hard and gritty.

Barnabe *et al.* (1973) described that the testes were asymmetrical and the left testis was hard. The scrotal connective tissue was thickened and oedematous. The serous cavity was obliterated by adhesions, and the tunics were thickened. The entire parenchyma was replaced by a hard yellowish mass.

Kaikini and Patil (1978) described the gross pathology of testicular calcification in a buffaloe bull. The testes were hard. Metallic sounds were produced while incising the testicular substance. The cut surface showed evenly spaced pin-head-sized spots of calcification throughout the substance of both the testes.

The calcified testes were hard and on incision a grating sound was emitted. The cut surfaces of the testes showed whitish to yellowish gritty masses which were most observed mostly towards the ventral portion. In extreme cases, the whole of the cut surface showed densely packed calcified masses leaving small areas of parenchyma unaffected (Chaudhuri *et al.* 1982).

Pandit and Pandey (1992) described on gross examination, the testes were shrunken, fibrotic in consistency with absence of tonicity. Cut section showed calcified masses within the tissues.

2.2.4.3 Histopathology

Maurya *et al.* (1968) classified the degree of testicular calcification as low, moderate, marked and high. In low degree of calcification, the seminiferous tubules had normal shape and size, but were slightly congested and showed mild degeneration of the lining epithelial cells and the interstitial connective tissue. The calcium was present in the form of small granules in the seminiferous tubules.

In moderate degree, the seminiferous tubules were generally irregular in shape and size. At few places the walls of the seminiferous tubules were broken and two or three adjacent tubules had joined together. At other places, the tubules were compressed and were smaller in size. The cells lining the tubules were detached from basement membrane and were placed in the centre of the tubule. There were focal areas of degeneration, necrosis, calcium deposition and mild leucocytic infiltration.

In marked degree, the prominent features were extensive degeneration, focal necrosis, fibroblastic proliferation and pronounced calcification. The seminiferous tubules had lost their morphology and their walls were disintegrated.

In high degree, the calcification was distributed throughout the specimen. At places, a few degenerated spermatogonial cells could be seen in the area of calcification. There was no evidence of connective tissue proliferation or inflammatory cells in the areas of calcification. The cells lining tunica albuginea were showing degenerative changes.

Venkataswamy and Pattabiraman (1970) recorded different stages of progressive calcareous seminiferous tubular degeneration resulting calcification and distortion of tubules widely separated by fibrous tissue. The initial stage was characterised by desquamation of tubular epithelium. The tubule was irregular and distorted with the basement membrane and widely separated from the interstitial tissue. The typical pattern of cellular layers of normal spermatogenesis was lost with distinct desquamation of cells. The cells of germinal epithelium were pushed away from the basement membrane and had lost their nuclei and were characterised by pyknosis and cytoplasmic vacuolation. Few spermatidic giant cells were present at the centre of the tubule. Some of the spermatogonial and Sertoli cells still persisted at the basement membrane and desquamated in the later stages.

The second stage was the stage of coalescence of desquamated cells, forming a cellular matrix in the centre of the tubule. There was stenosis and stagnation of tubular contents. In later stages, Sertoli cells were also involved.

The third stage was the stage of hyalinisation. The cells in the stagnated cellular matrix lost their nuclei and became distinctly hyalinized with large vacuoles. The stenotic tubules were occluded with denuded germinal epithelium. The fourth and final stage was the stage of calcification of the hyalinized mass.

The bare basement membrane was thickened and tubules were distorted. Proliferation of tubular tissue was also evident. Calcified and distorted tubules were widely separated by fibrous tissue.

Damodaran and Parthasarathy (1972) described the histological picture of calcification. Marked intratubular fibrosis and lymphocytic infiltration were prominent. The seminiferous epithelium was atrophied and reduced in thickness with only basal layer remaining in many of the tubules. Extensive intratubular calcification in several tubules was observed. There was no histological evidence of spermatogenesis.

Barnabe *et al.* (1973) described the testicular calcification on tissue section of which indicated complete absence of spermatogenesis, with tubular collapse and replacement by connective tissue in most areas. In some sections there was tubular calcification.

Chaudhuri *et al.* (1982) described that the affected seminiferous tubules were found to contain calcified masses. The adjacent tubules showed variable degeneration and at times cystic dilatation. The interstitial connective tissue was greatly increased which caused thickening of the septum. In certain areas, a few tubules showed distension of their lumina with presence of sperm where as majority of the tubules of the same area showed complete absence of sperms with no calcification.

2.2.5 Testicular Cyst

2.2.5.1 Prevalence

Charan and Verma (1997) recorded a case of unilateral monocentric testicular cyst in the nondescript buffalo bull. On screening, the right testis was comparatively larger and assumed an oval appearance than the contra lateral testis.

Kalicharan and Verma (2001) reported 1.64 per cent of testicular cysts in buffalo bull out of a total of 61 pairs of male gonads examined. A total of 38 organs had histopathological changes.

2.2.5.2 Gross Pathology

Damodaran and Thanikachalam (1977) noticed a cyst in the posterior extremity, near the ventral border of the right testis and it was about 2 cm. in diameter and filled with brownish, viscid and glairy fluid. The cyst wall was thick and hard.

Charan and Verma (1997) described the monocentric testicular cyst in which the right testis was viewed comparatively larger and assumed an oval appearance than the contra lateral testis. The incision divulged a centrally located cavity containing clear watery sap that easily flowed out from the right testis. The cyst appeared to be non-parasitic in origin as there was no evidence of parasitic scolices submerged in the fluidy contents.

2.2.5.3 Histopathology

Damodaran and Thanikachalam (1977) observed a single large cyst and several small cysts. The cyst was lined by a single layer of columnar epithelium which in some places, abruptly transcended to pseudostratified arrangement. The columnar cells were occasionally ciliated. The epithelium was thrown into folds and was highly mucigenous. Several goblet cells were present. The cavity contained mucus mixed with red blood cells. The cyst wall was made up of a thick collar of fibro-vascular tissue that contained mucous secreting glands, medullated nerve fibres and multiple lymphoid follicles.

Charan and Verma (1997) described that the cyst was lined by a thin connective tissue stroma abutting the atrophied seminiferous tubules.

2.2.6 Orchitis

2.2.6.1 Prevalence

Weidlch (1963) reported the sterility in bulls due to chronic tuberculosis orchitis and the lesions were characterised by necrosis, tuberculous nodules and fibrosis of the testicles.

Konig (1964) reported an unusual form of orchitis in 10 bulls which was a focal intra lobular orchitis with granuloma like lesions of non-tuberculous origin.

McIlwain and Bolin (1967) reported the mycoplasma-associated orchitis in three rams with enlarged testes.

Chaudhuri *et al.* (1982) reported two cases of orchitis in 197 cases buffaloes examined.

Trichard *et al.* (1982) reported a case of brucella orchitis in a Brahman bull with an acute, necrotic; granuloma which contained the *Brucella abortus* organisms.

Chand *et al.* (2002) reported three cases of epididymo-orchitis in breeding rams. The involvement of *Brucella melitensis* was serologically confirmed. In two of the rams, the enlargement was bilateral while in the other ram it was unilateral.

2.2.6.2 Gross Pathology

Jubb *et al.* (1993) described the intratubular orchitis grossly as a solitary or multiple white-yellow foci of up to about 1cm size.

Orchitis of the testis was scarcely distinguishable from testicular degeneration due to gross suppurative or necrotic destruction of the organ (Arthur *et al.*, 1996).

Radostitis *et al.* (2000) described orchitis due to brucellosis, in which the testes were not grossly enlarged. The testis underwent liquefaction necrosis and eventually got destroyed.

Chand *et al.* (2002) described gross lesions of the orchitis caused by *Brucella melitensis* in ram. The most consistent change observed was an enlargement of the epididymis and testis, which was highly conspicuous in the unilateral case. The capsule of the testes was markedly thickened and fibrous adhesions were seen between the skin, tunica dartos, and layer of tunica vaginalis. The testes were turgid and thick fluid filled material was seen. On incision, the testes were found replaced by thick, greyish-white caseous material that contained small remnants of testicular tissues. In one ram, despite enlargement of the epididymis and testis, the fibrotic adhesions between layers were less marked. However, when incised the testis of this animal revealed necrotic material under the thickened capsule.

2.2.6.3 Histopathology

Mycoplasma associated orchitis histologically revealed small necrotic areas throughout the tissue cuffed by mixed inflammatory exudate with polymorphonuclear leukocytes and occasional mononuclear cells. In addition to the inflammatory lesions, congestion was scattered throughout the tissue (McIlwain and Bolin, 1967).

Atrophy of the seminiferous tubules and mononuclear cell reaction in the interstitial tissue was evident in local interstitial orchitis (Chaudhuri *et al.*, 1982).

Gimbo *et al.* (1989) described chronic periorchitis in a bull. It was characterised by periarteritis nodosa, fibrosis of albuginea, spermatocytes vacuolation, multinucleate spermatids and epithelial shedding within the testis.

In interstitial orchitis in bulls, small mononuclear infiltrates were frequently observed adjacent to seminiferous or rete tubules or efferent ducts of

the testis. The intratubular orchitis showed the retention of tubular outline in the affected area. Seminiferous epithelium was obliterated and replaced centrally by neutrophils and detritus (Jubb *et al.*, 1993).

Chand *et al.* (2002) revealed the salient microscopic changes, which included diffuse infiltration of inflammatory cells into the tubular epithelium, as well as thickening of the tubular walls, the presence of a purulent exudate in the lumen, hyperplasia and intraepithelial cyst formation, spermatic granuloma with occasional calcification, and progressive fibrosis in the interstitial tissue.

2.2.7. Testicular Neoplasm

2.2.7.1 Prevalence

Dabholkar *et al.* (1967) examined two cases of Sertoli cell tumour from a nondescript 3-year old bull and a 10-year old Khalihar bull and one case of interstitial cell tumour from a 9-year old Konkani bull at slaughter in the Municipal Corporation Abattoir, Bombay. All cases were unilateral.

Shortridge and Cordes (1969) reported two seminomas in sheep in New Zealand. The occurrence was unilateral.

Bhagwat *et al.* (1972) recorded a rare case of bovine seminoma in a bull calf at the Department of Pathology, Nagpur Veterinary College, Nagpur. Palpation revealed a hard mass, of the size of a fist in the scrotal sac.

Ladds and Saunders (1976) reported Sertoli cell tumours in one mature and five aged bulls out of 1598 bulls slaughtered in abattoir in North Queensland. Tumours were located in the left testis of 3 bulls and in the right testis of 2 bulls; in one bull the side affected was not recorded.

Zanwar *et al.* (1982) studied 54 gonads from bulls and 97 buffaloe bulls in the age group of nine to fourteen years old in Deonar abattoir. Except in one

bull wherein seminoma was encountered and all others tumours were of Sertoli cell origin.

Dass *et al.* (1987) reported a case of Sertoli cell tumour in a 6-year old bullock with a large swelling in the scrotal and preputial region on the left side. On palpation, the left testicle was found hard and enlarged to a coconut size and the spermatic cord was abnormally thick. The right testicle was completely atrophied and asymmetric.

Gelberg (1987) reported the testicular interstitial cell tumour from nine stallions. In all but one horse, the tumours were found in undescended testes. Five animals had bilateral tumours. Two animals showed increased aggression.

Kagawa *et al.* (1998) reported a testicular yolk sac carcinoma (YSCA) in 28 day old male Japanese black calf clinically and confirmed by histopathologic and immuno histochemical techniques.

Reddy *et al.* (1999) reported the seminoma in a four-year-old Hallikar bull associated with cryptorchidism.

Joshi *et al.* (2005) reported a case of seminoma in 14-year old crossbred bullock with the history of enlarged testicle.

2.2.7.2 Gross Pathology

Dabholkar *et al.* (1967) described two cases of Sertoli cell tumour. In the first case the left testicle was enlarged and attained the size of 15.2 x 10.1 cm. and weighed 900 Gms. In the second case the right testicle was affected and the tumour weighed 1 kg and measured 45 x 20 cm.

In interstitial cell tumour, the tumour mass was yellow and had completely replaced the normal testicle. It weighed 2 kg and measured 37.5 x 35 cm. A number of haemorrhagic and necrotic spots could be seen on its surface.

Shortridge and Cordes (1969) observed seminomas wherein the testicle of one side was normal in size, shape, colour and consistency, and was approximately two-thirds the size of the enlarged testicle. The enlarged testicle measured 13.5 cm dorso-ventrally and 11.8 cm antero-posteriorly. It was of approximately normal shape, colour and consistency. The cut surface bulged and had a uniform glistening yellow-grey colour. In the second case the enlarged testicle measured 10 cm dorso ventrally, and 8 cm antero-posteriorly and was firmer than normal. The tail of the epididymis was enlarged, firmer than normal and the surface had smooth, nodular prominences. The body of the epididymis was thickened and contained palpable nodules. A medial longitudinal section through the testicle, epididymis and spermatic cord revealed a spherical mass 5 cm. in diameter in the testicle. This lesion was firm and pale brown with irregular red and yellow areas. It contained numerous small-mineralised areas and a fine stroma. It was surrounded by a fibrous capsule about 3 mm in thickness. A column of tissue approximately 8 mm in diameter penetrated the capsule from the central mass extended into the adjacent tissue. The remainder of the testicular tissue was firmer than the normal. It was pale brown, with red and yellow areas that produced a mottled appearance. Numerous veins adjacent to the tunica albuginea were thrombosed. The section of the epididymis showed bulging nodules surrounded by connective tissue septa. The thickened spermatic cord contained firm, pale brown tissue.

Bhagwat *et al.* (1972) observed the seminoma of the testis in bull. The growth was encapsulated. On incision of the scrotum, oedematous fluid oozed out, leaving a cauliflower-like, dark-red-coloured growth.

Ladds and Saunders (1976) recorded six numbers of Sertoli cell tumour in bulls. The tumours appeared as discrete pale nodules of the size of 1-3 cm in diameter and easily palpable in the intact testis. The tumour mass bulged from its cut surface. Single nodules were seen in the testes of 3 bulls but several nodules were present in the others. In three cases the tumours were in contact with tunica albuginea, while in two they were associated with the mediastinum. In other

cases the nodules were surrounded by parenchyma. Nodules were found both in the proximal and distal thirds and in the mid-testis region. Foci of calcification within nodules or at their periphery were evident. In one case small cysts were present in the parenchyma.

Zanwar *et al.* (1982) observed Sertoli cell tumour in cow and buffalo bulls. The affected gonads were usually enlarged, firm, and nodular and showed an irregular contour. The tunica vaginalis was moist and shining with its blood vessels standing out prominently. The cut surface was bulging and revealed a greyish yellow lobulated tumour mass. Epididymis was firm and swollen. A solitary seminoma in bull grossly observed in the study was in the form of two greyish white, firm nodules, each 3 cm in diameter with a homogenous pink cut surface.

Dass *et al.* (1987) recorded a case of Sertoli cell tumour in a bullock. It was greyish white, encapsulated in highly vascular glistening tunica vaginalis and its external surface had multiple nodular elevation of 2 to 5 cm diameter. It weighed 925 gms and was about 27 cm x 10 cm in size and the tail of epididymis and spermatic cord were abnormally thick. The right testicle weighed 11 gms. On cutting the hard exterior, the tumour bulged and contained soft yellowish tissue scattered in the hard fibrotic tissue. At some places, cysts with albuminous fluid were found. A large size of haematoma was also located towards the proximal end of the tumour.

Gelberg (1987) described testicular interstitial cell tumour in nine stallions. On sectioning it revealed multifocal indistinctly bordered nodules of varying sizes. The tumour was firm, gray, and irregularly shaped.

Reddy *et al.* (1999) reported a case of seminoma in a cryptorchid bull. The tumour appeared grey, lobulated and was bulging out from the testis.

Joshi *et al.* (2005) described seminoma of the testicle in two crossbred bullocks, weighed 2.5 kg with intact skin and reddish brown in colour. The

growth was encapsulated in a thick fibrous connective tissue capsule with septa invading the testicular mass. Growth appeared grayish yellow with small cysts containing watery fluid.

2.2.7.3 Histopathology

Dabholkar *et al.* (1967) described that the Sertoli cell tumour originated from the nonspermatic component of seminiferous tubules, the Sertoli cells. The growth had a thick capsule composed of adult connective tissue that was partially hyalinized. The seminiferous tubules were mostly atrophied, irregular in size and shape and were separated by a large amount of dense fibrous connective tissue. From this connective tissue, septa were seen to enter the tumour parenchyma, dividing it into number of lobules. These septa showed extensive hyalinisation. The collagenous stroma was seen to contain large number of thin walled capillaries that were congested. Few areas of haemorrhage and necrosis were also seen. The parenchyma of the tumour was composed of cuboidal or low columnar cells with pale cytoplasm and an oval nucleus containing fine chromatin and a prominent nucleolus. Mitotic figures were few. The cells were arranged irregularly in the lobules, formed by connective tissue septa. The tumour cells were arranged in palisade fashion in the periphery of the lobule. In interstitial cell tumour, the neoplasm was seen to be composed of cords of epithelial cells that were polyhedral and faintly eosinophilic with foamy vacuolated cytoplasm. Mitotic activity appeared to be rather low. Nucleus was vesicular with fine chromatin and a prominent nucleolus. The cell cords were separated by sinusoidal spaces, some of which showed large quantities of PAS positive homogenous material. Macronucleated hyperchromatic giant cells were seen in some areas. Extensive areas of haemorrhage and necrosis were other prominent features.

Shortridge and Cordes (1969) described the histopathological features of seminoma in two rams. In the first case, diffuse sheets of neoplastic cells had replaced the seminiferous tubules. The cells were spherical or polyhedral and most of them were of uniform size. The cytoplasm was sparse, eosinophilic and granular. Some cells contained large cytoplasmic vacuoles. The nuclei were large, approximately spherical, and vesicular with granular chromatin and had one or two distinct nucleoli. There were numerous mitotic figures with some bizarre forms. The neoplastic tissue was supported by connective tissue trabaculae and a vascular system, both of which appeared to have been part of the original testicular parenchyma. Reticulin fibres surrounded groups of neoplastic cells but did not invade individual cells. Few seminiferous tubules were present. Most of those which found were lined by single layer of degenerated cells. Many of these cells had pyknotic nuclei and swollen, granular, feathery cytoplasm. Formation of eosinophilic hyaline droplets in the cytoplasm was a prominent feature of the degenerative process. Sheets of neoplastic cells were present within the lumen of some veins and lymphatic vessels in the testicle. In the second case, almost all the tissue in the central mass showed coagulative necrosis and only a ghost outline was discernible. This tissue had contained many thin walled vascular spaces. Sheets of neoplastic cells were present between these spaces. Several rows of neoplastic cells lined the lumen of some vessels. The neoplastic cells had a small amount of eosinophilic cytoplasm that was feathery and vacuolated in many cells. The nuclei were large and approximately spherical, with finely granular chromatin and distinct nucleoli. There were few mitotic figures.

Histological examination of the seminoma revealed highly vascularised stroma with haemorrhages distributed throughout the growth. Abortive structures, not unlike seminiferous tubules, were observed. These were separated by massive fibrous tissue stroma. The type cells of the growth was highly anaplastic. The anaplastic cells revealed large vesicular nuclei characterised by

coarse chromatin distribution. The cytoplasm was faintly basophilic (Bhagwat *et al.*, 1972).

Ladds and Saunders (1976) described that all the Sertoli cell tumours exhibited an expansive pattern of growth, and early encapsulation was apparent in several of them. Degenerative changes in seminiferous epithelium were evident in tubules. These changes were vacuolation, spermiostasis, occasional mineralization and in one case early ossification, aspermia, and loss of germinal elements. A distinct tubular pattern was maintained in the tumours in all six bulls but tubule size, regularity, thickness of basement membrane and degree of intertubular fibrosis were all subject to considerable variation. Tubules, all with a very distinct basement membrane, ranged in size from those containing only a few Sertoli cells to those containing several hundred. Typically, tumour cells lined the basement membrane in palisade fashion; the nuclei were round or ovoid but always more basophilic and were usually smaller than the nuclei of normal Sertoli cells. The presence and prominence of nucleoli varied considerably. The occurrence of mitoses also varied. Tumour cells often filled the tubule. A striking histological feature in 5 of the 6 cases was the presence of intratubular laminated concrements within the tumour or at its periphery.

Zanwar (1982) described that the Sertoli cell tumour comprised seminiferous tubules of varying sizes and possessed a thick collagenous capsule. The tubules were occupied by oval or elongated Sertoli cells arranged in 2 or 3 layers. Mitotic figures were scanty. The unaffected testicular parenchyma was atrophic. Leydig cells were however unaffected. Seminoma histologically, comprised seminiferous tubules crowded with large polyhedral cells with a pink eosinophilic cytoplasm and round vesicular nucleus. Mitosis was rare. The adjacent non-neoplastic parenchyma was atrophic.

Dass *et al.* (1987) found that histologically, the Sertoli cell tumour was highly undifferentiated, consisting mostly of palisaded fusiform cells having indistinct cell boundaries and cytoplasmic projections. In some areas there was a

tendency to form primary tubule like structure with abundant fibrous tissue between them. In some areas the cells formed multiple layers. The cytoplasm of the tumour cells was distinctly vacuolated. Mitotic figures were abundant in the areas of solid tumour cells. At some places haemorrhagic areas were conspicuous.

Gelberg (1987) described that the testicular interstitial cell tumour was histologically the degeneration of seminiferous tubules and lined by a single layer of Sertoli cells. In some specimens, normal tissue architecture had been effaced by the neoplastic masses. Abundant fibrous tissue, much of it collagenous, was present in the interstitium. Connective tissue septa also dissected between sheets of neoplastic cells. Interstitial cells were found in nodular accumulation to broad sheets and were indistinctly delineated from surrounding parenchyma even though some growths were fairly discrete.

Reddy *et al.* (1999) described seminoma in a cryptorchid bull. Histologically, the tumour had highly vascular stroma with haemorrhages. The neoplastic cells were arranged as islands, separated by connective tissue. The neoplastic cells were large, rounded with granular cytoplasm. The nucleus was hyperchromatic. One or two nucleoli and few mitotic figures were present.

Kagawa *et al.* (1998) described a testicular yolk sac carcinoma (YSCA) in a Japanese black calf. Histologically, the mass consisted of tissues showing a variety of patterns; loose reticular network, pseudopapillary arrangement, festoon, solid nest, and labyrinthine pattern. Tumor cells were round to oval, with single central or polar nuclei with sharply defined nuclear borders and deeply basophilic chromatin.

Joshi *et al.* (2005) described seminoma as an irregular shaped and varying sized seminiferous tubules in sheet forms. At few places thick connective tissue stroma was observed. Neoplastic cells showed round vesicular pleomorphic

nuclei with pink eosinophilic cytoplasm. Nuclei were hyperchromatic with course chromatin material and moderate number of mitotic figures.

2.3 ENDOCRINE LESIONS IN TESTICULAR DISORDERS

2.3.1 General

The process of growth in animals is under endocrine control for most part of life and reproductive processes are primarily under endocrine control (MacDonald, 1969).

Ahmad *et al.* (1988) opined that genetic and endocrine abnormalities are the important causes for differing infertility patterns in buffalo bulls like testicular hypoplasia, arrested spermatogenesis, lack of sexual libido and small accessory sex glands.

2.3.2 Diseases of Pituitary Gland and Testicular Disorders

2.3.2.1 Incidence

Mudge (1955) reported that in the male, the pituitary-gonadal axis dysfunction was associated with gonadal dysfunction and various types of sexual precocity. Adrenal hyperplasia is attributed to a metabolic block in the normal synthesis of adrenal steroids.

MacMillan and Hafs (1968) studied the pituitary and hypothalamic endocrine relation in reproductive development of Holstein bulls from birth till one year of age.

Roberts (1971) reported that the hypophysectomy resulted in involution of the seminiferous epithelium and a lack of libido. Administering injections of FSH and LH restored spermatogenesis, libido and ejaculation of normal motile spermatozoa.

Schanbacher (1981) reported that the cryptorchid bull testes have a reduced capacity to secrete testosterone and the near-normal level of serum testosterone was maintained in the presence of increased serum gonadotrophins. In calves made cryptorchid at birth, serum Luteinizing Hormone concentrations were elevated over those of intact bulls.

The high concentrations of gonadotropins indicated the failure of negative feedback from the hypoplastic testicles on the hypothalamic, pituitary system. In man, high concentrations of LH and FSH with the presence of normal concentrations of testosterone and inhibin were interpreted as compensated Leydig and Sertoli cell failure (DeKrester *et al.*, 1989).

Capen (1993) reported the genetically determined failure of adenohypophysis development in Gurnsey and Jersey cattle, although the neurohypophysis developed normally. This resulted in a lack of foetal pituitary tropic hormone secretion during the last trimester and hypoplasia of target endocrine organs, the adrenal cortex, gonads, and follicular cells of the thyroid gland.

Peters *et al.* (2000) concluded that the seminomas were not endocrinologically active although both Sertoli cell tumours and Leydig cell tumours could cause increased oestrogen production leading to signs of feminisation. These tumours also had considerable amounts of inhibin-like immuno-reactivity. However Sertoli cell tumours produced a reduction in FSH concentrations.

Sweeney *et al.* (2000) reported that the maternal exposure to octylphenol inhibits the secretion of FSH in the foetus with a concomitant decrease in testis size and Sertoli cell number at birth of the lambs.

2.3.2.2 Panhypopituitarism

Paulsen (1962) described adult panhypopituitarism in which gonadotropins secretion ceases after puberty and secondary testicular atrophy follows. The germinal elements slough, the germinal epithelium atrophies, and finally the seminiferous tubules become fibrosed and hyalinized. Leydig cell function ceases and the cells revert to mesenchymal elements.

William (1962) reported that hypogonadism as the earliest sign of hypopituitarism occurred in 69 per cent of the male with chromophobe adenomas.

Jubb *et al.* (1993) reported the juvenile panhypopituitarism in German shepherd dogs resulting from secondary endocrine dysfunction such as hypothyroidism and hypoadrenocorticism resulted in infantile genitalia in which the testis and penis are small.

2.3.2.3 Hyperplasia of Pituitary

ElEtreby *et al.* (1980) reported that 10 male dogs of different breeds had diffuse hyperplasia and hypertrophy of somatotroph or lactotroph cells in the pituitary gland.

Sikdar and Bhowmik (1993) reported hyperplastic lesions (9.0%) in the pituitary glands of 300 goats and the highest prevalence in male goats belonging to the Black Bengal breed in the age group of more than 5 years.

Emmakeeble (2001) reported that pituitary hyperplasia and adenomas are extremely common in older rats (70 per cent incidence in older Sprague-Dawley rats).

Abraham *et al.* (1987) reported the enlargement of the pituitary and microscopically the vacuolation and hyperplasia of the basophil cells in focal areas in hypothyroidism of male calves.

2.3.2.4 Pituitary Adenoma

Capen (1993) reported that craniopharyngioma is a benign tumor derived from epithelial remnants of the oropharyngeal ectoderm of the craniopharyngeal duct (Rathke' s pouch). This resulted in the lack of secretion of pituitary tropic hormones and tropic atrophy, subnormal function of the adrenal cortex and thyroid, atrophy of the gonads, and failure to attain somatic maturation due to lack of growth hormone.

2.3.2.4.1 Pathology

Capen (1993) described that the craniopharyngiomas were large and grow along the ventral aspect of the brain, where they can surround several cranial nerves. In addition they extend dorsally in to the hypothalamus and thalamus.

2.3.4 Diseases of Thyroid Gland and Testicular Disorders

2.3.4.1 Hypothyroidism

Sreekumaran (1976) observed that the seminiferous tubules contained only few primary and secondary spermatocytes in experimentally induced hypothyroid state in male kids and there was complete absence of spermatozoa and germinal layer in some seminiferous tubules and the lumen contained a network of fibres, scattered round cells and marked interstitial oedema.

Reddy and Rajan (1985) observed hypothyroidismin in goats, the testis grossly showed decrease in relative weight, reduced size, smooth in consistency and pale in colour. Histologically, the seminiferous tubules were small and were lined by a single layer of spermatogonial cells. The tubules contained only a few

inactive primary and secondary spermatocytes. Spermatogenesis was completely absent. Some of the tubules contained pink stained granular material; only few of the tubules contained sperms. There was moderate degree of interstitial oedema. The interstitial cells were scattered and few in number. There was no evidence of Sertoli cells in most of the tubules; with only a few of them containing scattered Sertoli cells.

Abraham *et al.* (1987) reported hypothyroidism in male calves and observed the significant increase in the weight of the thyroid gland that was characterised by the absence of colloid. Some of the follicular epithelial cells were hyperplastic and degenerated. In testes, most of the seminiferous tubules were lined with single layer of epithelial cells without mitotic activity. Some of the tubules showed degenerated and desquamated epithelial cells. There was moderate interstitial oedema. The seminiferous tubules were slightly smaller in size and lining cells were scanty. In few, scattered spermatocytes were seen, without any evidence of proliferation of the spermatogonial cells. In some of the tubules a few degenerated and desquamated cells along with a few giant cells were seen. Interstitial cells were loosely scattered in the interstitial spaces, which was slightly oedematous and appeared relatively prominent.

Tahmaz *et al.* (2000) studied the effects of hypothyroidism on the testes in mature rats by using propylthiouracil and histopathologically observed that maturation arrest of spermatogenesis, a reduced number of Sertoli and Leydig cells, a decreased tubular diameter, interstitial oedema, and thickening of basal membrane of the testis

2.3.4.2 Thyroid Hyperplasia (Goitre)

Sharma and Ramkumar (2001) described the enlargement of thyroid gland in goitrous male goats and it was congested, tough in consistency, and cut surfaces were meaty in appearance. Histologically, the thyroid gland showed variable sized spherical or irregular follicles and lined by multilayered high

cuboidal cells, partially or completely occluding lumen. The testes showed degeneration of spermatogonial cells and atrophy of seminiferous tubules.

2.3.5 Diseases of Adrenal Gland and Testicular Disorders

2.3.5.1 General

Kelly (1984) reported that the androgenic hormone named dehydroepiandosterone was produced by zona reticularis in adrenal gland in normal level has little effect on testis and the dysfunctional status could result in production of large amounts of derivative sex steroids which led to feminising syndromes in men.

2.3.5.2 Adrenal Hyperplasia

Yarrington and Capen (1981) reported the unilateral and bilateral adrenal hyperplasia in four aged bulls and consisted of multinodular or diffuse areas of hyperchromatic chromaffin cells that were non-encapsulated and compressed adjacent cortical tissue.

Nair *et al.* (1981) recorded cortical hyperplasia of the adrenal gland in a five year old buck. There was gross enlargement of the adrenal to almost double in size. Both testes and pituitary of the animal showed no abnormalities on gross and histopathological examination.

Abraham *et al.* (1987) reported experimentally induced hypothyroidism in male calves by using thiourea and there was a significant increase in relative weight of the adrenal glands. Histologically, proliferation and depletion of fat in the cells of zona fasciculata was recorded.

McDonald and Pineda (1989) reported that adrenal cortices normally produce androgens, and adrenal androgen secretion may be greatly increased in a variety of pathological condition, including adrenal hyperplasia or neoplasia, nymphomania in cattle, and adrenal virilism in women.

Ava *et al.* (1997) studied testicular histopathology in two cases with congenital lipoid adrenal hyperplasia in which seminiferous tubules was normal in diameter and consisted of an age-appropriate number of type A spermatogonia and an increased number of Sertoli cells. The interstitial space was abundant and contained normal to increased number of Leydig cells that were filled with lipid droplets. Lipid accumulation in Leydig cells was consistent with defective conversion of cholesterol to prednenolone and the other appeared to be common disorders associated with hypergonadotrophic hypogonadism.

2.3.5.3 Adrenal Neoplasms

2.3.5.3.1 Incidence

Roberts (1971) reported that the steroid producing tumours of the testes and adrenal gland caused degeneration and atrophy of the seminiferous epithelium and the interstitial cells.

Capen (1993) reported that cortical adenomas in castrated male goats had greater incidence than the intact males.

2.3.5.3.1 Pheochromocytoma

2.3.5.3.1.1 Incidence

Capen (1993) described that pheochromocytoma was the most common neoplasms in the adrenal medulla of animals, occurring most often in cattle and dogs and infrequently in other species. In bulls and human beings, pheochromocytomas developed concurrently with calcitonin secreting cell neoplasms of the thyroid gland.

2.3.5.3.1.2 Gross Pathology

Wilkie and Krook (1970) revealed a mass 16 to 18 cm in diameter adjacent to the anterior pole of the right kidney that apparently replaced the right adrenal. It was soft, spongy, and, on cut surface, dark brown with numerous

blood vessels containing thrombi. The tumour was histopathologically confirmed as pheochromocytoma.

West (1975) described that the adrenal gland at the point of caval penetration was constricted. Except for a shaggy area on the cephalic aspect, the surface of the intravascular portion of the gland was smooth and appeared to be encapsulated. The edges bulged when the gland was sectioned. A central mass of mottled, grayish brown, and haemorrhagic tissue was surrounded by a thin light brown band; grayish white striae coursed through the extra-and intravascular tissue.

2.3.5.3.1.3 Histopathology

Yarrington and Capen (1981) described that the four bulls had either diffuse on multinodular proliferation of chromaffin cells, resulting in expansion of the adrenal medulla. These lesions were hypercellular and non-capsulated, and they compressed the adjacent normal adrenal cortical tissue. The hyperplastic cells in the adrenal medulla were polygonal and had a lightly basophilic cytoplasm. They were subdivided into small packets by a fine fibrovascular stroma, or formed tubules. The pheochromocytomas were composed of large, densely packed, well-differentiated chrommaffin cells arranged in columns in close apposition to numerous capillaries.

Materials and Methods

3. MATERIALS AND METHODS

The present study was conducted at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy to investigate the pathology of selected endocrine glands in testicular disorders in bull.

3.1 MATERIALS

3.1.1 Data Collection

The history and details regarding the animals were documented after getting information from the records maintained in the concerned farm.

3.1.2 Sample Collection

A total of 110 bulls from the Meat technology unit, College of Veterinary and Animal Sciences, Mannuthy and 190 bulls from the Corporation slaughter house, Thrissur were screened for testicular disorders after antemortem examination. Out of these 105 and 156 cases were examined for gross lesions respectively after postmortem examination. From the above, that 20 and 25 cases of testis, pituitary, thyroid, adrenal and accessory sex glands of bulls with and without a history of reproductive failure respectively were chosen for further studies.

3.2 METHODS

3.2.1 Morphometry

The morphometry of the animals brought for slaughter at the Meat Technology Unit and the Corporation slaughterhouse, Thrissur were studied. The body weight of the carcasses and the weight of the both testis were recorded. The relative weight of the testes was determined. Collection of testis, pituitary, thyroid, adrenal and accessory organs was based on the methods described by Ahmad *et al.* (1985).

Pituitary gland was collected and weight of the gland was recorded. Thyroid gland was dissected out from the caudal larynx on the first or second tracheal ring. The weight of the thyroid gland was also recorded. Adrenal glands were collected and the weight of the gland was recorded. The accessory glands of the bull which comprised of the seminal vesicles, ampullae, prostate and bulbourethral glands were collected and their respective weight and size recorded.

3.2.2 Gross Examination

3.2.2.1 Testis

The right and left testis was examined for gross lesions if any. The size, shape, colour and consistency were recorded. The organ was examined for any congenital or hereditary anomalies, acquired defects, degenerative changes, inflammatory conditions and neoplasms. The lesions were recorded.

3.2.2.2 Endocrine Glands

The size and weight of the pituitary gland, thyroid and adrenal glands were recorded and examined for any gross pathological changes including hypertrophy, hypoplasia, cyst, haemorrhage and tumour.

3.2.2.3 Accessory Sex Glands

The seminal vesicle, prostate, bulbourethral gland and ampullae were examined grossly for any congenital and acquired defects and the pathological changes were noted.

3.2.3 Histopathology

Testis was halved mid-sagittally. Transversely cut slices from the dorsal, middle and ventral parts of each testis were fixed in Bouin's fluid immediately after collection. After 36-48 hours, the fixed slices were transferred to 10 per

cent aqueous formalin for 12 hours and then stored in 70 per cent alcohol (Ahmad *et al.*, 1988).

The pituitary gland was preserved in Bouin's fluid and the tissues were processed by routine histological techniques (Luna, 1968). The thyroid gland was cut into pieces and fixed in the 10 per cent neutral buffered formalin and the tissues were processed by conventional method to obtain 4-6 microns thick paraffin sections (Reddy and Rajan, 1985). Samples of adrenal tissue were collected and preserved in Bouin's fluid and were processed for paraffin embedding (Luna, 1968). The accessory glands were cut into pieces and preserved in 10 per cent formalin and were processed for paraffin embedding (Luna, 1968).

Paraffin wax sections were cut at 3-4 microns thickness and stained with Harris haematoxylin and eosin as described by Disbrey and Rack (1970). Stained sections of each testis, pituitary, thyroid, adrenal and accessory glands were examined under low and high magnifications. Histopathological lesions of these organs were recorded and classified. Special stains such as PAS, Trichrome and Alizarin red were used wherever necessary as per methods described by Luna (1968). Gross and histopathological lesions were correlated with history and details (age, breed and weight) of the animals.

Results

4. RESULTS

A total of 110 bulls from the Meat technology unit, College of Veterinary and Animal Sciences, Mannuthy and 190 bulls from the Corporation slaughter house, Thrissur were screened for testicular disorders at the time of antemortem examination between January 2004 and June 2005. Out of these 105 and 156 cases were examined for gross lesions at the time of post mortem examination respectively. Gross and histopathological examination were carried out on testis, pituitary, thyroid, adrenal and accessory sex glands of bulls obtained from the above two places in which 20 cases with history of reproductive failure and 25 cases without a history of reproductive failure were chosen for further studies respectively. The carcasses were grouped based on age and breed. Lesions were classified according to age, breed and pathological findings.

4.1 PREVALENCE OF LESIONS

4.1.1 Testis

Out of 261 samples examined 45 samples of the testis revealed pathological changes on gross and histopathological examination. The prevalence of the testicular disorders in bulls is shown in table 1 and Fig.1. Unilateral right-sided testicular aplasia was recorded in one case. Testicular hypoplasia was recorded in 10 cases and testicular degeneration in 25 cases. Varicosity of the testis was seen in 2 cases. Fibrosis in three cases and calcification in four cases were recorded.

4.1.1.1 Influence of Age

The age wise prevalence of testicular disorders in bulls is shown in the Table 2 and Fig.2. A higher prevalence of lesion in the corporation slaughterhouse was seen in the age group of above three to five years that was

31.11 per cent and higher prevalence in the Meat Technology Unit was seen in the age group of above five to seven years that was 33.33 per cent. The age group above one and half years to three years of age showed only a lower prevalence that was 4.44 per cent and 2.22 respectively.

4.1.1.2 Influence of Breed

The prevalence of testicular disorders based on the breed of the bull is shown in Table 3 and Fig.3. The study revealed a higher incidence of testicular disorders in nondescript breed. Out of 25 cases at Corporation Slaughter House examined 12 revealed testicular disorders. In the Meat Technology Unit the crossbred Holstein-Friesian showed highest prevalence of testicular lesion. Eight out of 20 animals showed testicular disorders.

4.1.2 Endocrine Glands

The lesions observed in pituitary, thyroid and adrenal were classified and tabulated in the table 4.

4.1.2.1 Pituitary Gland

Lesions of pituitary glands were recorded in 16 out of 45 samples examined. Cyst was seen in nine cases and haemorrhage, congestion and oedema was seen in seven cases.

4.1.2.2 Thyroid Gland

Lesions of thyroid gland were recorded in 27 cases out of 45 samples examined. Hypoplasia was recorded in seven cases. Degeneration was seen in ten cases, cyst was seen in five cases, colloid goitre in three cases and nodular goitre was recorded in two cases.

4.1.2.3 Adrenal Gland

Lesions of adrenal gland were seen in 21 cases out of 45 samples examined. Cortical hypoplasia in two cases, cortical hyperplasia in seven cases and medullary hyperplasia in twelve cases were observed.

4.1.2.4 Accessory Sex Glands

Lesions of accessory glands were seen in 21 cases out of 45 samples examined. Hyperplasia of seminal vesicle was seen in eight cases and hypoplasia of seminal vesicle was seen in six cases. Prostatic hyperplasia was seen in two cases and atrophy of the prostate was seen in five cases.

4.2 HISTORY OF THE ANIMALS

Twenty bulls from the Meat Technology Unit, Mannuthy were between the age group of one and half years to seven years with known history of decreased libido and congenital anomalies of the testis. The semen was watery in consistency, reduced concentration and motility. In many cases the semen contained abnormal and defective sperms. In some cases increased mucopurulent and haemorrhagic flakes in semen were noticed.

4.3 RELATIVE WEIGHT OF ORGANS

The relative weight of the testis; pituitary, thyroid and adrenal glands are given in the Table 5 and 6 respectively. The weight of the animal and the weight of the testis, pituitary, thyroid and adrenal glands were measured and the relative weight of the above said organs were calculated.

The relative weights of the testis were decreased in testicular hypoplasia and degeneration and increased in the varicosity, fibrosis and calcification. The relative weight of the pituitary, thyroid and adrenal gland was increased in pituitary cyst, cystic goitre, nodular goitre and adrenal hyperplasia. The relative

weight of the thyroid and adrenal gland was decreased in testicular hypoplasia condition.

4.4 GROSS PATHOLOGY

4.4.1 Testis

4.4.1.1 Aplasia

The right testis of the bull was absent in one case. The head, body and tail of the epididymis were only prominent without evidence of testicular tissue except for the presence of a pale yellow mass of fibrous tissue between the head and tail of epididymis on the right side and it was soft in consistency (Fig. 4). The left testis was normal.

4.4.1.2 Hypoplasia

In two cases unilateral hypoplasia (Fig. 5) and eight cases bilateral hypoplasia (Fig. 6) was encountered. The testis was reduced in size and weight. The capsule was thickened. The size of the testis ranged between 4.3 cms x 2.1 cms and 9.0 cms x 4.9 cms and the weight of the testis ranged between 15.5gms to 65 gms. The relative weight ranged in between 0.0007 and 0.02 percent.

4.4.1.3 Degeneration

The testis was smaller than the normal and hard in consistency. The thickening and wrinkling of tunics was seen in most cases. The cut surface was coarse granular in appearance. In few cases marked oedema was seen. Grossly there was reduction in size and weight of the testis. The size ranged from 6.7 cms x 5.4 cms to 14.2 cms x 7.2 cms. The weight ranged from 105 gms to 370 gms. The relative weight ranged between 0.0352 and 0.08 per cent.

4.4.1.4 Varicosity

Varicosity was seen in two cases. Testis was reddish coloured and the consistency hard. There was severe congestion over the tunics. All the blood vessels appeared highly engorged and tunics were filled with blood with prominent varicosity of vein (Fig. 7). The testis was enlarged and oedematous in appearance. The size of the testis ranged between 12.2 cms x 5.6 cms and 15.4 cms x 8.0 cms. The weight of the testis ranged between 210 gms and 424 gms. The relative weight of the testis ranged between 0.05 and 0.06 per cent.

4.4.1.5 Fibrosis

Fibrosis was seen in three cases. The size of the testis ranged between 5.8 cms x 4.4 cms and 8.4 cms x 6.7 cms and weight ranged between 115 gms and 175 gms respectively. The relative weight ranged between 0.0588 and 0.0625 per cent. The tunics were thickened. The testis was too hard in consistency. The cut surface was dark brown and white streaks seen within the parenchyma.

4.4.1.6 Calcification

Calcification was seen in four cases. The thickening of the tunics of the testis was seen. The calcium salts were deposited in the stroma and the superficial areas of the testis (Fig. 8). The testis appeared wrinkled and the glistening appearance disappeared. The affected part was whitish in colour. On incision grittiness was felt. The size of the testis ranged from 6.6 cms x 4.5 cms and the weight ranged between 185 gms and 225 gms. The relative weight ranged between 0.049 and 0.064 per cent.

4.4.2 Endocrine Glands

4.4.2.1 Pituitary gland

4.4.2.1.1 Cyst

Cyst was noticed in nine cases. The pituitary gland was enlarged. The size of the gland ranged between 2.7 cms x 1.9 cms and 3.2 cms x 2.1 cms. The weight of the gland ranged between 2.7 gms and 3.4 gms. The relative weight of the gland ranged between 0.0009 and 0.001 per cent. The diameter of the cyst ranged between 0.6 cms and 1.2 cms and was deep seated. On incision one cyst was seen in between the pars distalis and pars intermediary part of the pituitary gland (Fig. 9). The cyst caused distension of the gland. The cyst contained straw-coloured turbid fluid.

4.4.2.1.2 Haemorrhage

Haemorrhage was noticed in seven cases. The gland was soft and reddish in colour. Cut section revealed haemorrhages in the pars distalis part of the gland (Fig. 10). The size and weight of the gland was increased. The relative weight of the gland was ranged between 0.0005 and 0.0013.

4.4.2.2 Thyroid Gland

4.4.2.2.1 Hypoplasia

Hypoplasia of thyroid gland was seen in seven cases. The gland was reduced in size and weight. The relative weight ranged between 0.0019 and 0.0051 percent. The gland was elongated, pale and hard in consistency. There was loss of glistening appearance. Moderate fibrosis was evident (Fig. 11).

4.4.2.2.2 Hyperplasia

Hyperplasia was seen in three cases. There was increase in size and weight of the gland. The gland appeared dark brown. The consistency was soft

to pliable (Fig. 12). Copious quantity of pale yellow to brown coloured gelatinous material oozed out from the cut surface.

4.4.2.2.3 Cystic Thyroid Gland

Cyst of the thyroid gland was seen in five cases. There was increase in size and weight of the gland. The relative weight of the gland ranged between 0.0066 and 0.0099 per cent. The colour was dark brown and the consistency pliable. A cyst of 2.8 cms diameter filled with light brown to purple gelatinous material was seen embedded in the parenchyma (Fig. 13).

4.4.2.2.4 Nodular Goitre

Nodular goitre was seen in two cases. There was increase in size and weight of the gland. The size of the gland was 6.5 cms x 3.8 cms and the weight was 26 gms. The relative weight was 0.014 per cent. Nodules of varying size were seen deeply embedded or projecting from the surface of the gland (Fig. 14). A large sized nodule measuring 3.2 cms was seen in one of the glands and the cut surface of which appeared granular (Fig. 15). The colour of the gland was dark brown and the consistency was firm.

4.4.2.3 Adrenal Gland

4.4.2.3.1 Adrenal Hyperplasia

Adrenal hyperplasia was seen in 21 cases. The adrenal gland was enlarged. The size and weight of the gland was increased. The colour of the glands was brown to dark brown and the consistency was hard (Fig. 16). Moderate fibrosis was seen in three cases. Oedema of the gland was seen in five cases

4.4.2.3.2 Adrenal Hypoplasia

Adrenal hypoplasia was seen in two cases. The adrenal gland was smaller in size and the weight also decreased. The size was 4.1 cms x 1.3 cms and

weight was 3.8 gms. The relative weight ranged between 0.0008 and 0.009 per cent. The gland was pale brown and soft in consistency (Fig. 17). On section, the gland revealed cortical zone to be much narrow compared to the medullary zone.

4.4.3 Accessory Sex Glands

4.4.3.1 Seminal Vesicle

4.4.3.1.1 Hyperplasia

The gland appeared enlarged. There was loss of lobulation and the consistency of the organ was firm. Petechiae could be seen on the serosal surface of the organ (Fig. 18).

4.4.3.1.2 Hypoplasia

The gland was small in size and lower in weight. The gland appeared rudimentary without any lobulation (Fig. 19).

No lesion was seen in the prostate, ampullae and bulbo-urethral gland.

4.5 HISTOPATHOLOGY

4.5.1 Testicular Disorders

4.5.1.1 Aplasia

Aplasia was seen in one case. There was total absence of any developed seminiferous tubules. Fibrous tissue proliferation was predominant. Amidst the proliferating fibrous tissue irregular slit like spaces lined by flattened cells could be seen (Fig. 20). In some areas the peritubular fibrosis was too extensive that the slit like structures appeared occluded. There was thickening of the vessel wall with variable degree of hyalinization of vessels were evident amidst the fibrous tissue mass (Fig. 21).

4.5.1.2 Hypoplasia

Testicular hypoplasia was recorded in 10 cases. Variety of changes could be observed in the histopathology of hypoplastic testes. In some cases, groups of seminiferous tubules with thickened basement membrane and complete absence of lining cells were seen amidst tubules of varying functional activity (Fig. 22). The hypoplastic tubules appeared collapsed. Vacuolation of the scanty cells lining the tubules was seen in some. There was predominance of interstitial cells of Leydig (Fig. 23). Very small sized seminiferous tubules to medium sized were seen. Seminiferous tubules of very small size with complete absence of spermatogonial cells and in some others only a single layer of cells could be seen. In some cases basement membrane were lined only by Sertoli cells (Fig. 24). In two cases, peripheral to groups of hypoplastic tubules, papillary proliferation of tubular cells could be seen (Fig. 25). Thickened and hyalinized basement membrane with collapsed tubules and focal infiltration of inflammatory cells were seen in some other cases (Fig. 26). Peritubular and perivascular fibrous tissue proliferation, vascular congestion and thrombosis of the vessels were the other lesions observed (Fig. 27).

4.5.1.3 Degeneration

The testicular degeneration was recorded in 25 cases. According to the intensity of the degenerative change it was classified as mild, moderate and severe. Mild degeneration was seen in six cases, moderate in 12 cases and extensive or severe degeneration in seven cases.

Mild degeneration was seen in certain groups of tubules or in the same tubules, certain regions or part (Fig. 28). Vacuolation of the cells and nuclear pyknosis were evident. Lesions were seen tubular epithelial cells of seminiferous tubules in small foci of amidst the tubules showing normal spermatogenesis (Fig. 29).

In moderate cases, the affected tubules were devoid of any lining. The membrane appeared wrinkled. There was hyalinisation of the cells that had degenerated and appeared as homogenous mass in the centre of the lumen (Fig. 30). Some affected tubules were clear and there was collapse of the membrane. Detachment, clumping of cells and formation of giant cells was seen in some (Fig. 31). Incomplete layers, thickening of the tunica albuginea and predominance of Sertoli cells were also seen.

In cases with extensive degeneration, only the basement membrane remained without any evidence of germinal elements (Fig.32). There was widening of the interstitium and diffuse collections of interstitial cells were observed. Degenerative changes were also observed in the interstitial cells.

4.5.1.4 Varicosity of the testis

In two cases, varicosity of the testis was noticed. There was thickening of the tunics. The blood vessels of the tunics (Fig. 33) and the parenchyma (Fig. 34) appeared dilated and tortuous. The vessel wall was highly thickened. Degeneration and desquamation of the seminiferous epithelium, oedema and haemorrhage around the dilated vessels were characteristics (Fig. 35).

4.5.1.5 Fibrosis

The testicular fibrosis was recorded in three cases. There was loss of normal architecture of the seminiferous tubules. The tubular basement membrane appeared highly thickened due to peritubular fibrosis (Fig. 36). The dense fibrous tissue in the interstitium caused wide separation of the tubules (Fig. 37). Some of the tubules appeared contracted due to peritubular fibrosis. The tubules were filled with clumps of degenerated and desquamated spermatogonial cells. Seminiferous tubules were scanty and those present appeared greatly reduced in their size and were distorted. The tubules in some regions were seen obliterated by the proliferating fibrous tissue that formed a circular profile (Fig. 38). In certain areas the interstitial cells of Leydig were predominant as aggregates of

cells where as in other location they were individually separated and scattered amidst the proliferating fibrous tissue. The lumen of certain tubules was distorted and there was absence of any stages of the cell layers. There was absence of spermatogenesis. The seminiferous tubular cells were flattened in some wherein there was no differentiation in to spermatogonial cells.

4.5.1.6 Calcification

The testicular calcification was recorded in four cases. The basement membrane showed increased mineralization in most of the tubules adjacent to the tunics (Fig. 39) and some of the tubules had irregular lining with necrosis and mineralization. Intra luminal necrosis and mineralization was seen in some of the tubules (Fig. 40). Infiltration of inflammatory cells was recorded in some cases. Complete necrosis of the tubular cells which appeared as homogenous pink staining masses over which was deposited calcium were observed in majority of the testicular tissue. The deposited calcium salts appeared orange red with Alizarin red stain (Fig. 41).

4.5.2 Endocrine Disorders

4.5.2.1 Pituitary Gland

4.5.2.1.1 Pituitary Cyst

Pituitary cyst was seen in nine cases. The cyst was seen both in the adenohypophysis and neurohypophysis. Cyst was either single or multiple (Fig. 42), small or large. Most of the cysts were seen in the pars tuberalis and pars distalis. The cystic cavity was either empty or filled with homogenous pink staining exudate (Fig. 43). The cyst wall was lined by flattened cells. Degeneration, necrosis and hyalinisation of acidophils and basophils were seen in the vicinity of the cysts (Fig. 44). Widespread haemorrhage was seen adjacent to the cystic spaces.

4.5.1.2 Pituitary Haemorrhage and Oedema

Haemorrhage was seen in seven cases. Haemorrhage was seen within the pars distalis. Homogenous pink staining fluid separated the pituicytes within the parenchyma (Fig. 45). There was diffuse hypertrophy of the acidophils and basophils in the pars distalis. Acidophils were proportionately much less. Severe congestion was seen in most of the areas of adenohypophysis.

4.5.2 Thyroid Gland

4.5.2.1 Thyroid Degeneration

Thyroid degeneration was seen ten cases. The follicles were found to be varying in size and many were devoid of colloid. Focal areas revealed degeneration and desquamation of follicular epithelium (Fig. 46). Most of the colloid contained partially or completely infiltrated cells and mild degree of fibrosis was seen. Interstitial tissue appeared prominent. Dilatation of blood vessels with haemorrhage was seen. The colloid was seen red and fibrous tissue was seen as green colour with trichrome stain (Fig. 47).

4.5.2.2 Nodular Goitre

Nodular goitre was seen in two cases. Follicles of varying size lined by many layers of cells were seen. Coalesion of some of the follicles with nodular aggregates of cells appeared in some regions (Fig. 48). Compression of adjacent thyroid follicles was seen. Proliferation of lining cells forming papillary projections in to the lumen was also seen (Fig. 49).

4.5.2.3 Thyroid Cyst

Thyroid cyst was seen five cases. The follicles were of varying shape and size with irregular outline. Many of the follicles were either empty or contained small quantity of pale or dark acidophilic colloid and few showed cystic

dilatation. A large cyst lined by flattened cells was seen in the parenchyma (Fig. 50). The follicles around the cyst were compressed with scanty colloid.

4.5.2.4 Colloid Goitre

The colloid goitre was seen in three cases. Highly distended and irregular follicles lined by flattened cells and filled with pale staining colloid were seen (Fig. 51).

4.5.2.5 Thyroid hypoplasia

Thyroid hypoplasia was seen seven cases. More micro follicles were seen. The lumen was filled with pale staining colloid in some follicles whereas in the majority of the follicles there was no colloid (Fig. 52). Flattened cells lined the follicular epithelium. The connective tissue stroma appeared prominent.

4.5.3 Adrenal Gland

4.5.3.1 Adrenal Hyperplasia

Both cortical and medullary hyperplasia were seen. Cortical hyperplasia was seen in seven cases. Increase in the width of all zones of the cortex narrowing the medullary zone was evident (Fig. 53). In certain cases, there was thickening of the capsule which was seen proliferating and invading the zona glomerula in closing group of glomerular cells which appeared encapsulated in nodules (Fig. 54). Medullary hyperplasia was seen in 12 cases. The medullary zone was very much distended and there was hyperplasia of the cells (Fig. 55). Hyperplasia was so great that the cortical segment was reduced to a narrow rim under the capsule. Dilatation of the vessels, sinusoids and haemorrhage were seen in the medulla (Fig. 56).

4.5.3.2 Adrenal cortical hypoplasia

Adrenal cortical hypoplasia was seen in two cases. There was great reduction in the various zones of the cortex which appeared narrow. There was

no clear demarcation of the different cortical zones and vacuolation of the cells were seen (Fig. 57).

4.5.4 Diseases of Accessory Gland

4.5.4.1 Seminal vesicle

4.5.4.1.1 Hyperplasia

Hyperplasia was seen in eight cases. There was proliferation of glandular cells. Most of the cells appeared columnar and formed intraluminal projections. There was increased secretory activity and the lumen contained large quantities of secretory fluid (Fig. 58).

4.5.4.1.2 Hypoplasia

Hypoplasia was seen in six cases. The lining cells were flat and there was reduction in the size of the glandular lumen which appeared devoid of any secretion. Moderate fibrosis was also evident (Fig. 59).

4.5.4.2 Prostrate

4.5.4.2.1 Hyperplasia

Hyperplasia was seen in two cases. Dilated acini of varying sizes were scattered throughout the gland. They were lined by flattened epithelium and the lumen contained homogenous pink staining secretion (Fig. 60).

4.5.4.2.2 Atrophy

Atrophy was seen in five cases. Microscopical examination revealed the presence of shrunken and collapsed acini with obliteration of the lumen, which was seen as slit like spaces. There was fibrous tissue proliferation in the interstitial tissue (Fig. 61).

No histopathological lesions were seen in the ampullae and bulbo-urethral gland.

4.5.5 Concurrent Lesions of Endocrine Gland and Testis

The most prominent gross and histopathological findings of pituitary, thyroid, adrenal glands associated with various testicular disorders are given in Table 5. The pituitary lesions included pituitary cyst, pituitary haemorrhage, congestion and oedema. The thyroid gland lesions included hypoplasia, degeneration, cyst, nodular goitre and colloid goitre. The adrenal gland lesions were cortical hypoplasia, cortical and medullary hyperplasia.

Lesions of pituitary glands in 16 cases were recorded. Cyst was seen in nine cases and haemorrhage, congestion and oedema was seen in seven cases. Cyst was seen both in testicular hypoplasia and degeneration. Haemorrhage, congestion and oedema were seen in hypoplasia, degeneration, fibrosis and calcification of the testis.

Lesions of thyroid gland were recorded in 27 cases. Hypoplasia was recorded in seven cases. Degeneration was seen in ten cases, cystic goitre in five cases, colloid goitre in three cases and nodular goitre was recorded in two cases. The hypoplasia was recorded in the testicular hypoplasia and degeneration. Thyroid degeneration was seen in the degeneration, fibrosis and calcification of testis. The cystic goitre was seen in the testicular hypoplasia and degeneration. The colloid goitre was seen in the degeneration of testis. The nodular goitre was recorded in the hypoplasia and degeneration of the testis.

Lesions of adrenal gland were seen in 21 cases. Cortical hypoplasia was seen in two cases, cortical hyperplasia was seen in seven cases and medullary hyperplasia was seen in 12 cases. The cortical hypoplasia was seen in the testicular hypoplasia and degeneration. The cortical and medullary hyperplasia was seen in hypoplasia, degeneration, fibrosis and calcification of the testis.

4.5.6 Concurrent Lesions of Accessory Gland and Testis

Hyperplasia and fibrosis of seminal vesicle was seen in eight cases and hypoplasia of seminal vesicle was seen in six cases. Prostatic hyperplasia was seen in two cases and atrophy of the prostate was seen in five cases. The hypoplasia of seminal vesicle was seen in the testicular hypoplasia and the hyperplasia of seminal vesicle was seen in degeneration, varicosity, fibrosis and calcification of the testis. Hyperplasia of the prostate was seen in degeneration of testis. Atrophy of the prostate was seen in degeneration and fibrosis of testis.

Table 1. Prevalence of testicular lesions in bulls

Sl. No.	Place of materials examined	Total number of bulls screened	Total number of samples examined	Total number of cases with disorders	Aplasia	Hypoplasia	Degeneration	Varicosity	Fibrosis	Calcification
1.	Corporation Slaughter House, Thrissur	190	156	25 (16.03)	1 (4.0)	9 (36.0)	11 (44.0)	1 (4.0)	1 (4.0)	2 (8.0)
2.	Meat Technology Unit, College of Veterinary and Animal Sciences, Mannuthy	110	105	20 (19.05)	--	1 (5.0)	14 (70.0)	1 (5.0)	2 (10.0)	2 (10.0)
Grand Total		300	261	45 (17.24)	1 (2.22)	10 (22.2)	25 (55.56)	2 (4.44)	3 (6.67)	4 (8.89)

Figures in parenthesis shows the percentage

Table 2. Prevalence of testicular lesions in various age groups of bulls

Sl. No.	Place of materials examined	Sl. No.	Age group (years)	Testicular lesions						Total
				Aplasia	Hypoplasia	Degeneration	Varicosity	Fibrosis	Calcification	
I.	Corporation Slaughter House, Thrissur	1	>1½ yr-3 yr.	--	2	--	--	--	--	2
		2	>3yr-5yr	1	5	5	1	1	1	14
		3	>5yr-7yr	--	2	6	--	--	1	9
		Total		1	9	11	1	1	2	25
II	Meat Technology Unit	1	>1½ yr-3 yr.	--	1	--	--	--	--	1
		2	>3yr-5yr	--	--	4	--	--	--	4
		3	>5yr-7yr	--	--	10	1	2	2	15
		Total		--	1	14	1	2	2	20
Grand Total				1	10	25	2	3	4	45

Table 3. Prevalence of testicular lesions in various breeds of bulls

Sl. No.	Place of materials examined	Sl. No.	Breed	Testicular lesions						Total
				Aplasia	Hypoplasia	Degeneration	Varicosity	Fibrosis	Calcification	
I.	Corporation Slaughter House, Thrissur	1	CBJ	1	2	3	1	--	1	8
		2	CBBS	--	1	1	--	--	--	2
		3	CBHF	--	2	1	--	--	--	3
		4.	ND	--	4	6	--	1	1	12
		Total		1	9	11	1	1	2	25
II	Meat Technology Unit	1	CBJ	--	--	5	1	--	--	6
		2	CBBS	--	1	3	--	1	1	6
		3	CBHF	--	--	6	--	1	1	8
		4.	ND	--	--	--	--	--	--	--
		Total		-	1	14	1	2	2	20
Grand Total				1	10	25	2	3	4	45

CBJ - Crossbred Jersey
CBBS - Crossbred Brown Swiss
CBHF - Crossbred Holstein-Friesian
ND - Non Descript

Table 4. Prevalence of endocrine gland lesions in testicular disorders of bulls

Sl. No.	Testicular disorders (Number of cases)	Endocrine gland lesions									
		Pituitary		Thyroid					Adrenal		
		Cyst	Haemorrhage	Hypoplasia	Degeneration	Cyst	Colloid goitre	Nodular goitre	Cortical hypoplasia	Cortical hyperplasia	Medullary hyperplasia
1.	Aplasia (1)			1							
2.	Hypoplasia (10)	3	2	5		3		1	1	1	
3.	Degeneration (25)	6	3	1	7	2	3	1	1	6	10
4.	Varicosity (2)				1						
5.	Fibrosis (3)		1		1						1
6.	Calcification (4)		1		1						1
Grand total		9	7	7	10	5	3	2	2	7	12

Figures in parenthesis shows number of cases

Table 5. Relative weight of the testis in various testicular disorders in bull

Sl. No.	Testicular lesion (n)	Relative weight of the testis in %	
		Range	Mean ± S.D.
1.	Hypoplasia (10)	0.0007-0.02	0.01 ± 0.007
2.	Degeneration (25)	0.0352-0.08	0.042 ± 0.014
3.	Varicosity (2)	0.05-0.06	0.06 ± 0.011
4.	Fibrosis (3)	0.0588-0.0625	0.05 ± 0.008
5.	Calcification (4)	0.049-0.064	0.06 ± 0.008

Table 6. Relative weight of pituitary, thyroid and adrenal gland in various endocrine lesions in bulls

Sl. No.	Name of the organ	Endocrine gland lesions (n)	Relative weight in %	
			Ranges	Mean ± S.D.
I.	Pituitary	Cyst (9)	0.00097-0.0010	0.001 ± 0.002
		Haemorrhage (7)	0.0005-0.0013	0.0009 ± 0.003
II.	Thyroid	Hypoplasia (7)	0.0019-0.0051	0.004 ± 0.001
		Degeneration (10)	0.0039-0.0091	0.005 ± 0.002
		Cyst (5)	0.0066-0.0091	0.008 ± 0.001
		Colloid goitre (3)	0.00696-0.014	0.009 ±0.004
		Nodular goitre (2)	0.0099-0.014	0.012 ± 0.003
III	Adrenal	Hypoplasia (2)	0.0008-0.0009	0.0009 ± 0.001
		Hyperplasia (19)	0.0027-0.0083	0.0065 ± 0.001

Figures in parenthesis shows number of cases
S.D. - Standard deviation

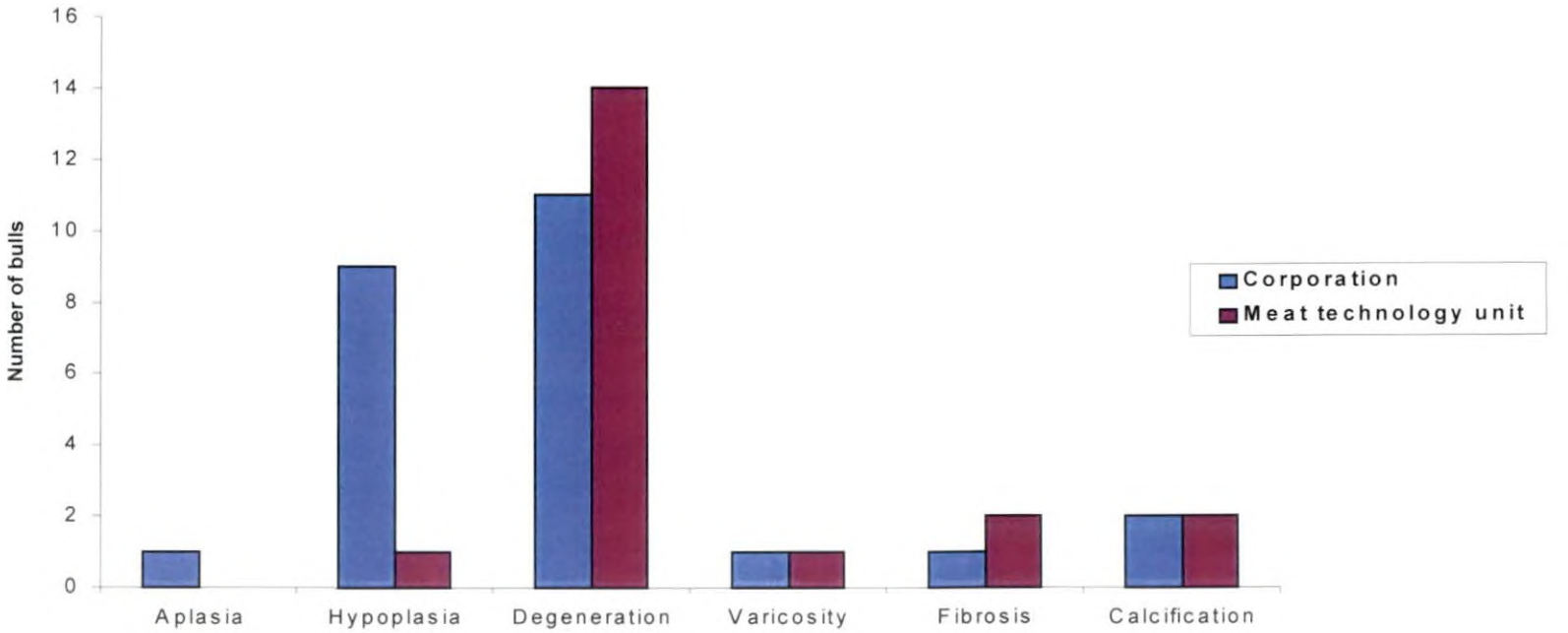

Fig. 1 Prevalence of testicular lesions in bull

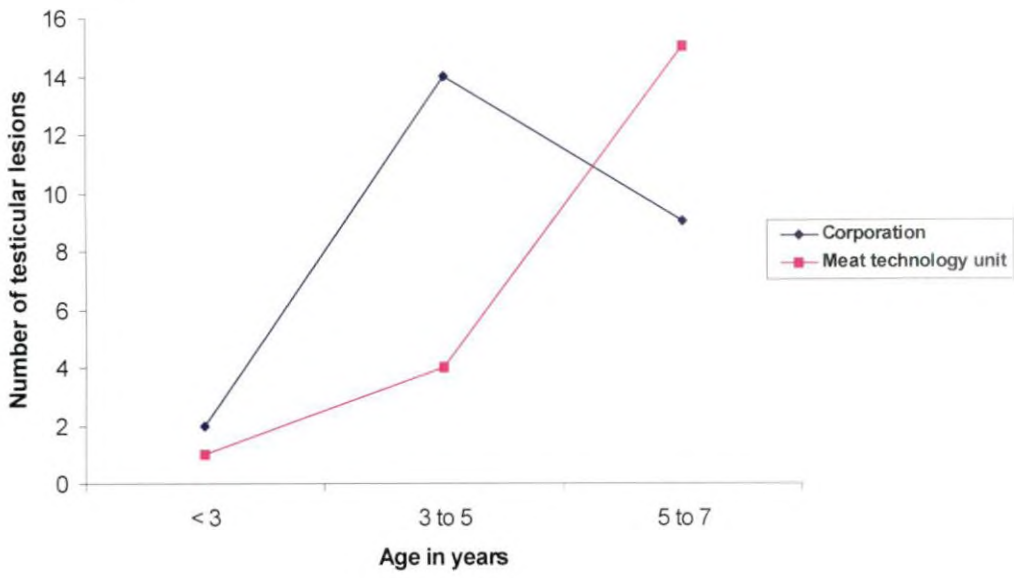

Fig.2 Prevalence of testicular lesions in various age group of bulls

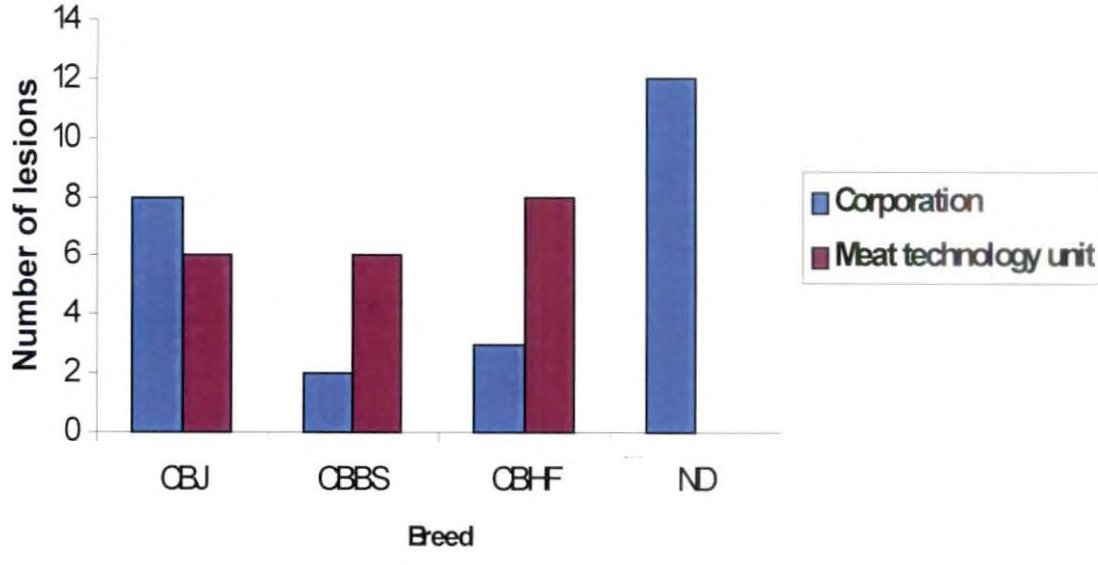

Fig.3 Prevalence of testicular lesions in various breeds of bulls

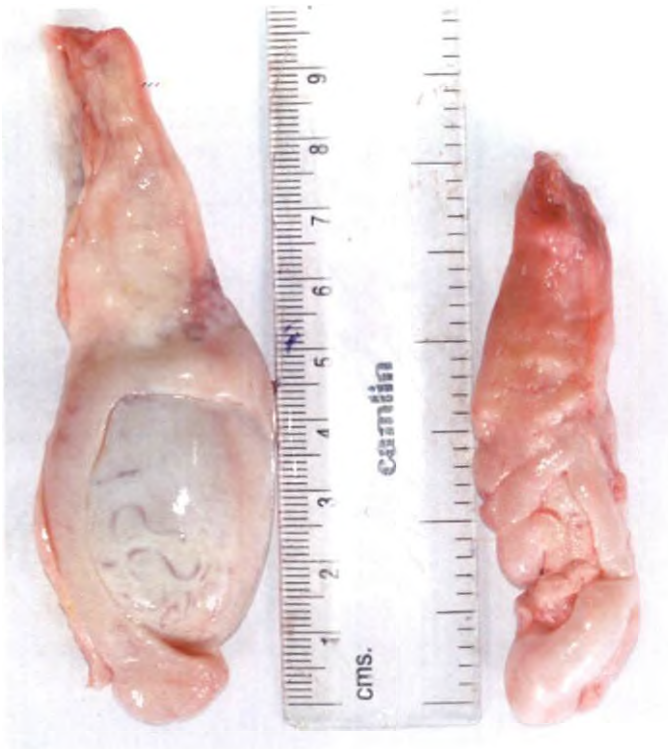

Fig. 4

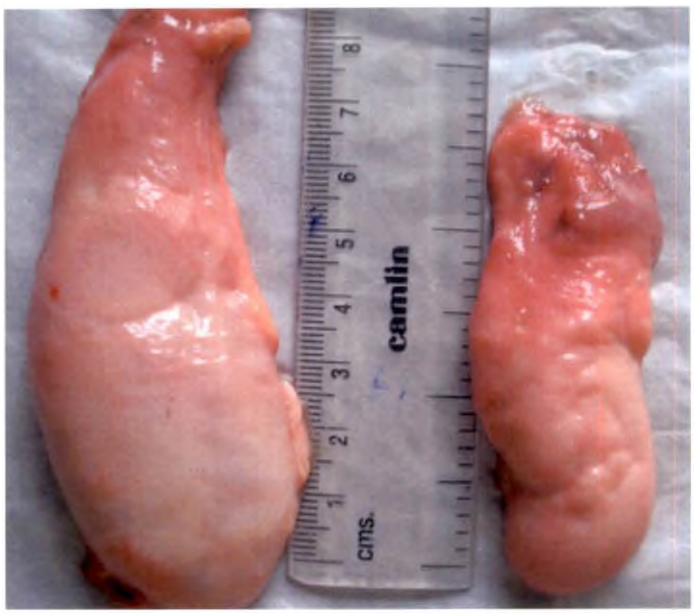

Fig. 5

Figure- 4.

Unilateral testicular aplasia: fibrous tissue mass between head and tail of epididymis

Figure-5.

Unilateral testicular hypoplasia

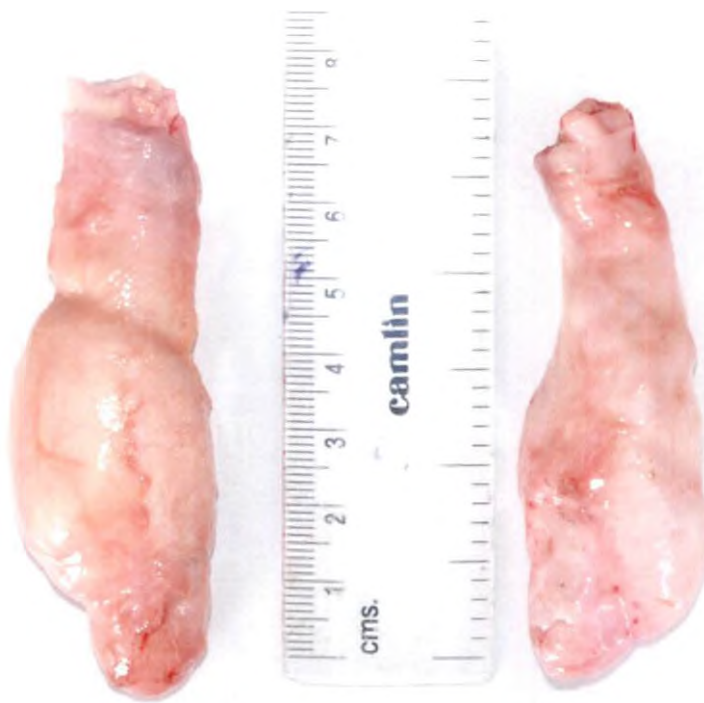

Fig. 6

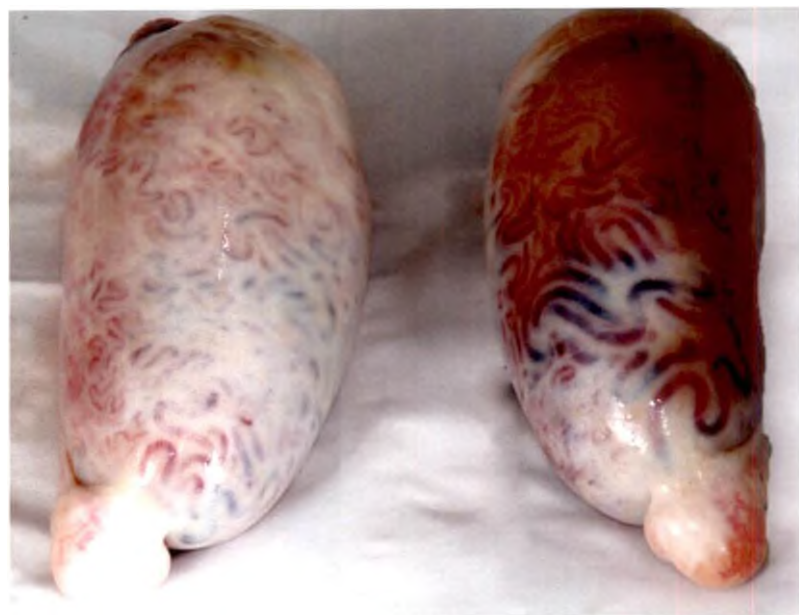

Fig. 7

Figure- 6.
Bilateral testicular hypoplasia

Figure-7.
Varicosity of testis

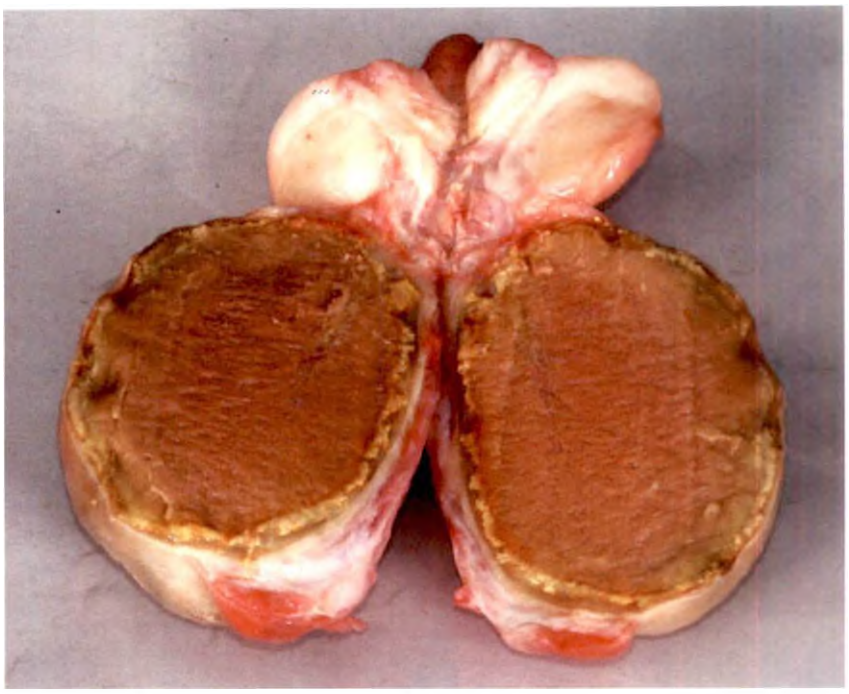

Fig. 8

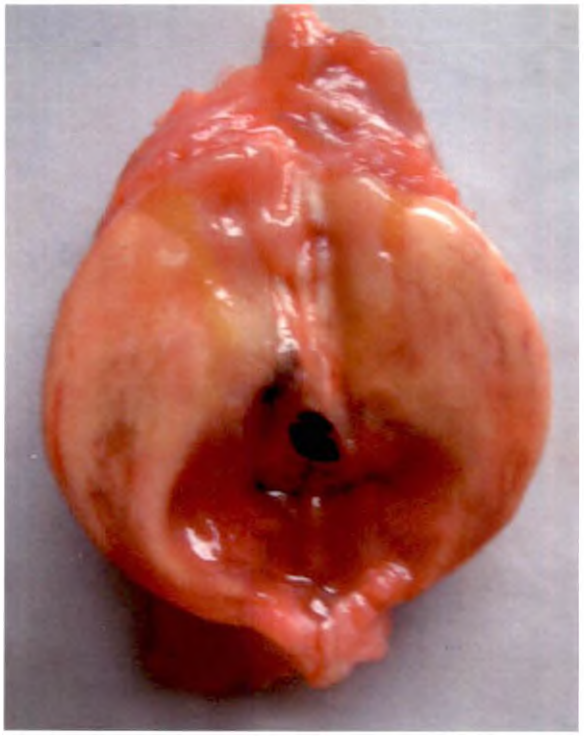

Fig. 9

Figure- 8.

Calcification of testis: pale yellow coloured of calcium deposits in the stroma and superficial areas.

Figure-9.

Pituitary cyst: cyst in the adenohypophysis.

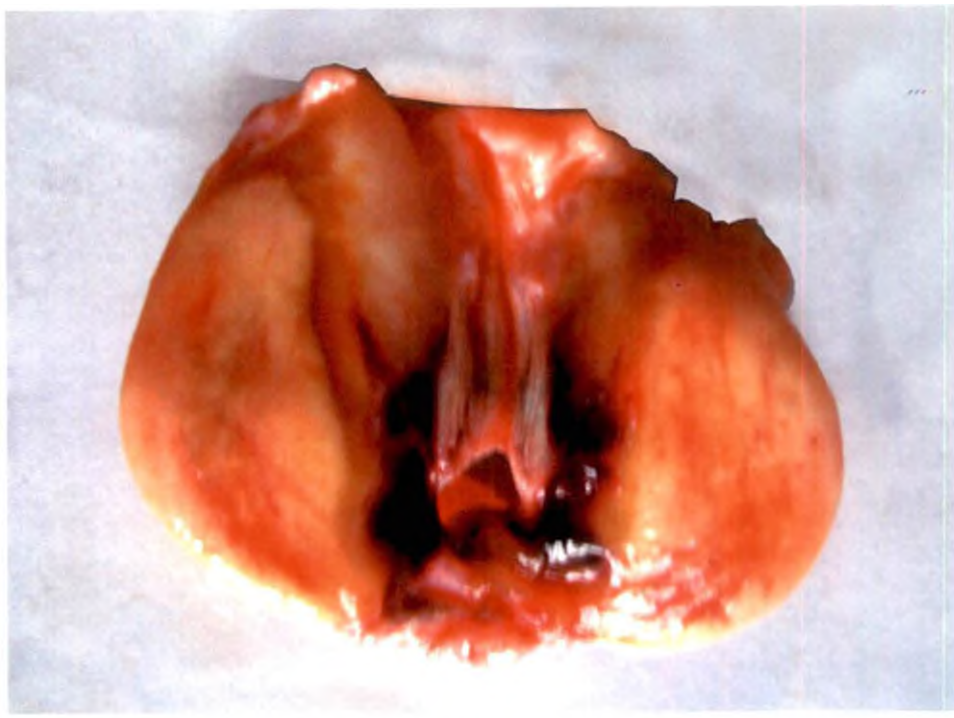

Fig.10

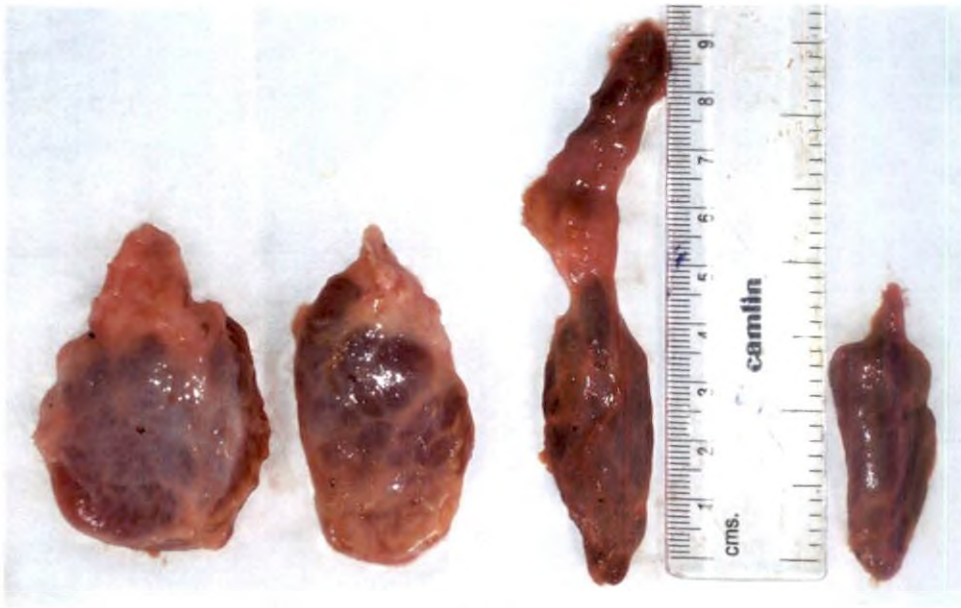

Fig.11

Figure-10.

Pituitary haemorrhage.

Figure-11.

Normal thyroid gland (left) and hypoplasia of thyroid gland (right).

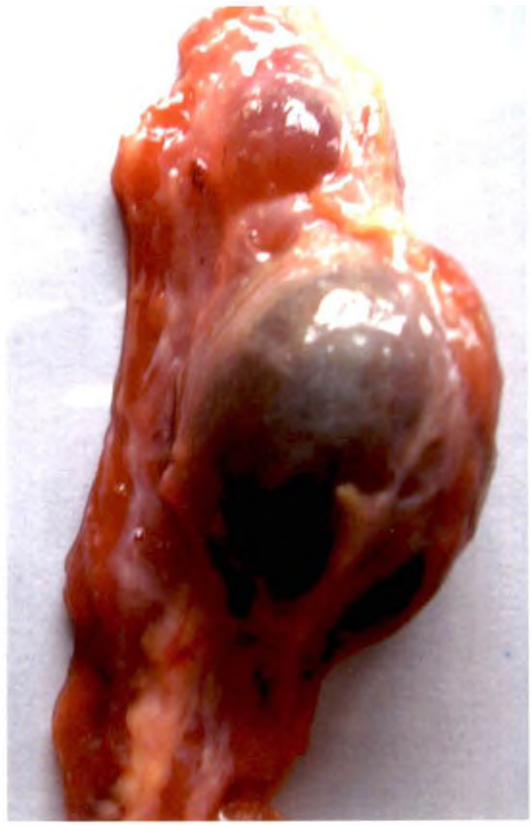

Fig. 12

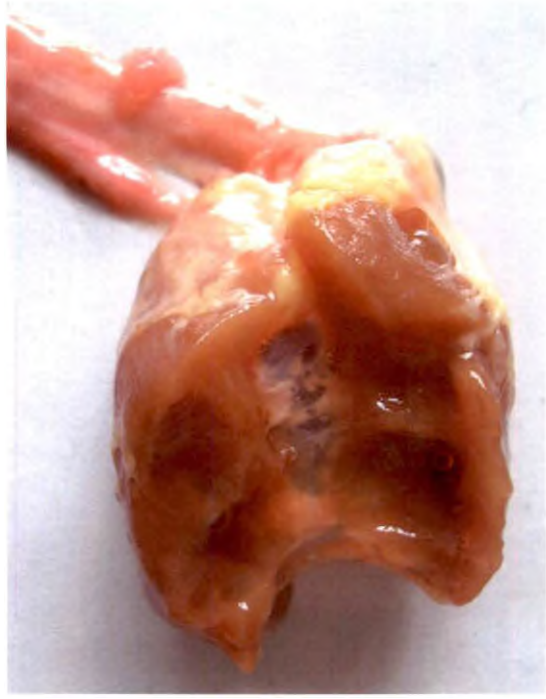

Fig. 13

Figure- 12.
Thyroid hyperplasia (goitre).

Figure- 13.
Thyroid cyst.

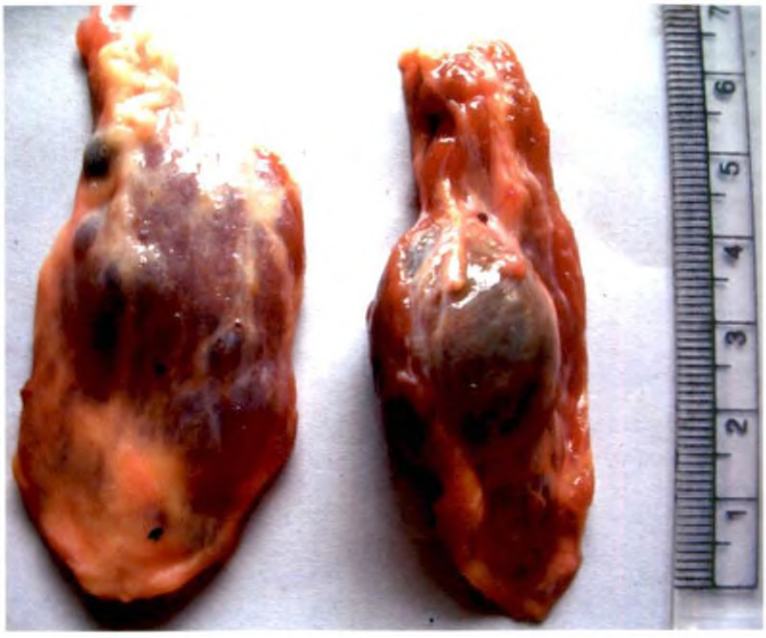

Fig. 14

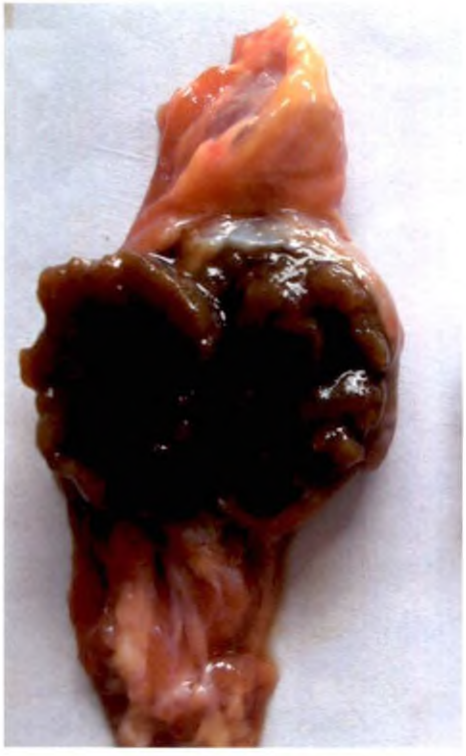

Fig. 15

Figure- 14.

Nodular goitre: thyroid gland showing multiple small nodules in one and single large nodule in the other.

Figure- 15.

Nodular goitre: cut surface appears granular.

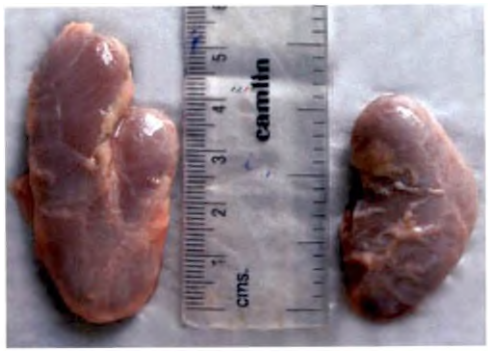

Fig. 16

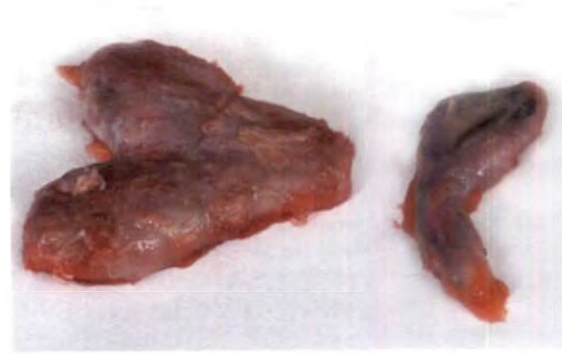

Fig. 17

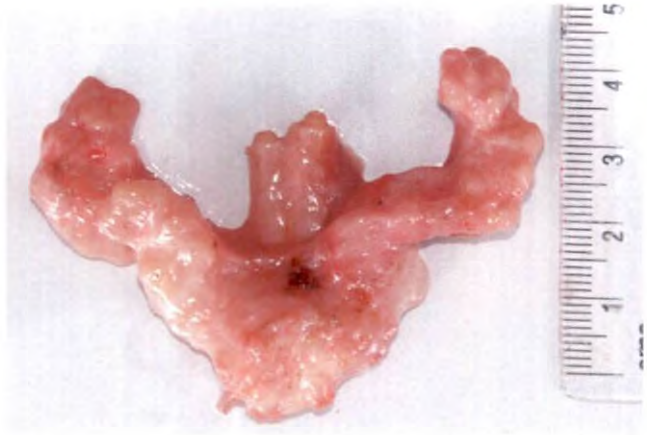

Fig. 18

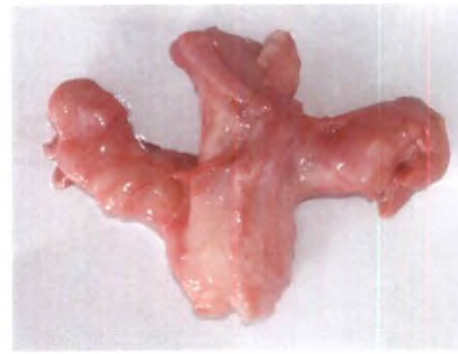

Fig. 19

Figure- 16.

Adrenal hyperplasia.

Figure- 17.

Adrenal hypoplasia.

Figure- 18.

Seminal vesicle hyperplasia.

Figure-19.

Seminal vesicle hypoplasia.

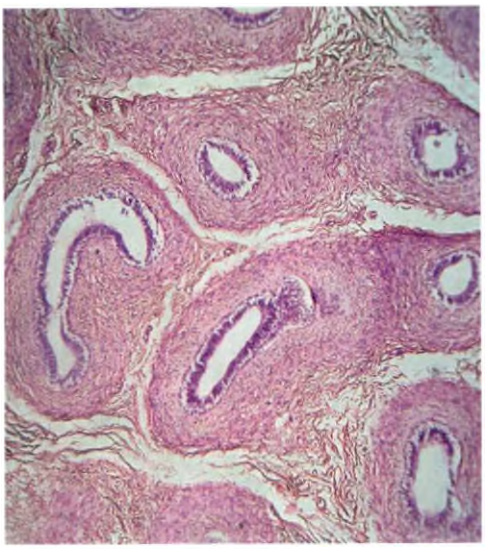

Fig. 20

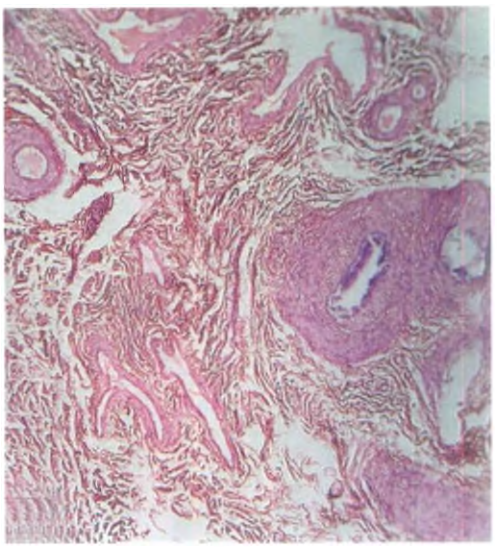

Fig. 21

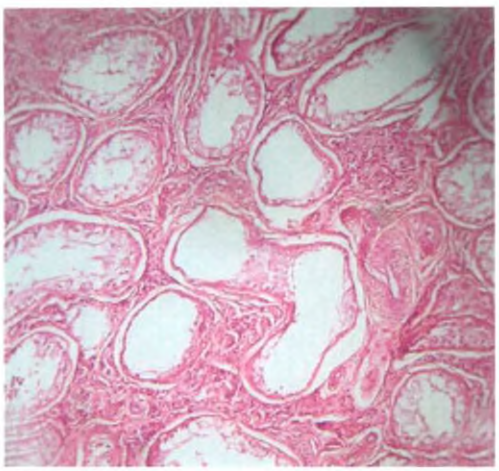

Fig. 22

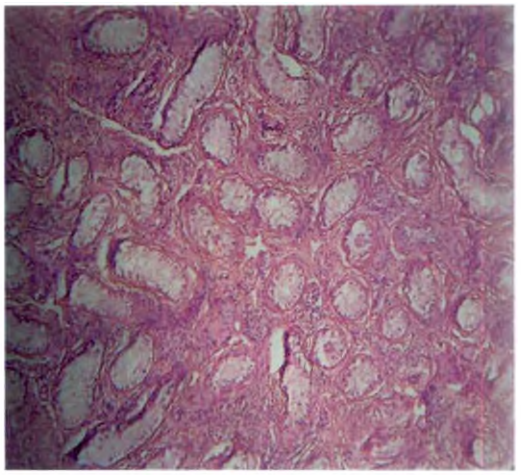

Fig. 23

Figure- 20.

Testicular aplasia: slit like seminiferous tubules with peritubular fibrosis- H&E x 250.

Figure- 21.

Testicular aplasia: fibrous tissue proliferation and hyalinization of vessel- H&E x 250.

Figure- 22.

Testicular hypoplasia: thickened basement membrane and complete absence of lining cells- H&E x 250.

Figure- 23.

Testicular hypoplasia: tubules of small size and predominance of Leydig cells- H&E x 100.

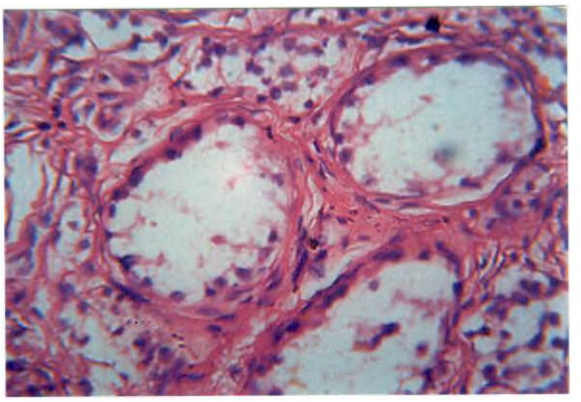

Fig. 24

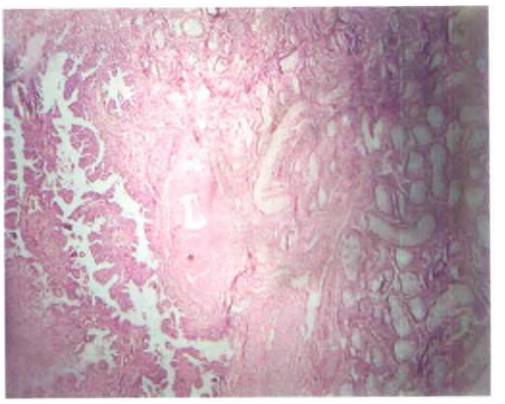

Fig. 25

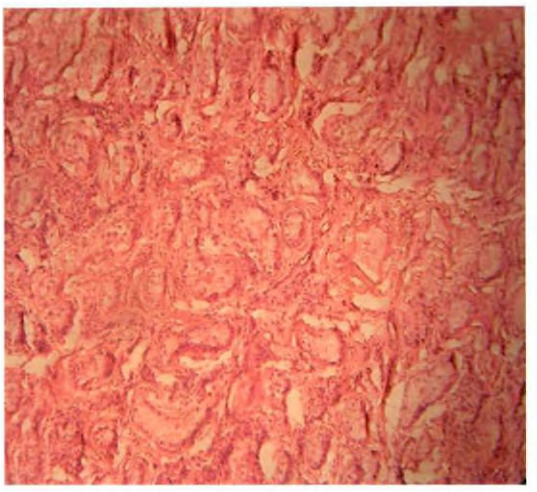

Fig. 26

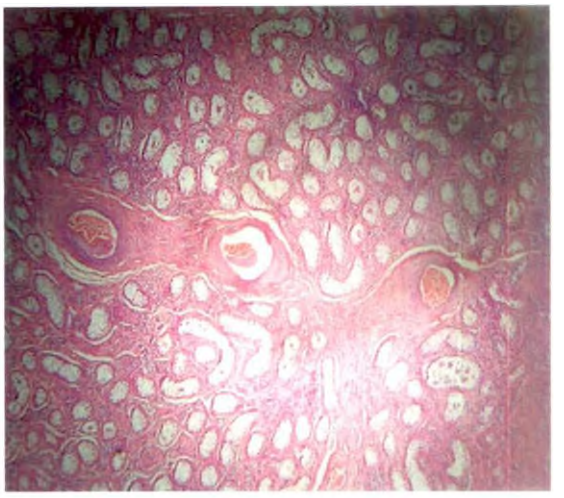

Fig. 27

Figure- 24

Testicular hypoplasia: absence of spermatogonial cells and basement membrane lined only by Sertoli cells- H&E x 400.

Figure- 25

Testicular hypoplasia: papillary proliferation of tubular cells adjacent to hypoplastic tubules and hyalinization of vessel- H&E x 100.

Figure- 26

Testicular hypoplasia: hyalinized basement membrane and infiltration of inflammatory cells- H&E x 100.

Figure- 27

Testicular hypoplasia: vascular congestion and thrombosis of vessels- H&E x 100.

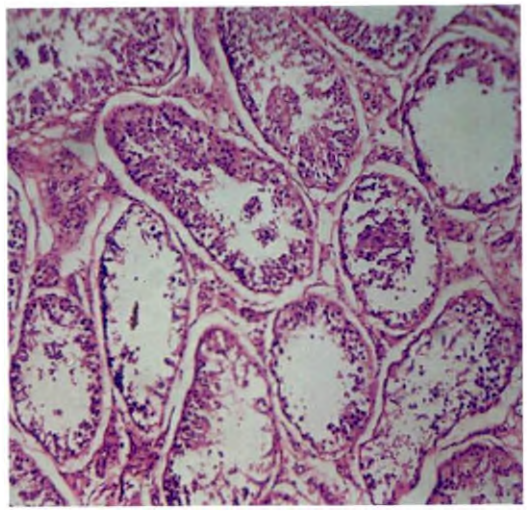

Fig. 28

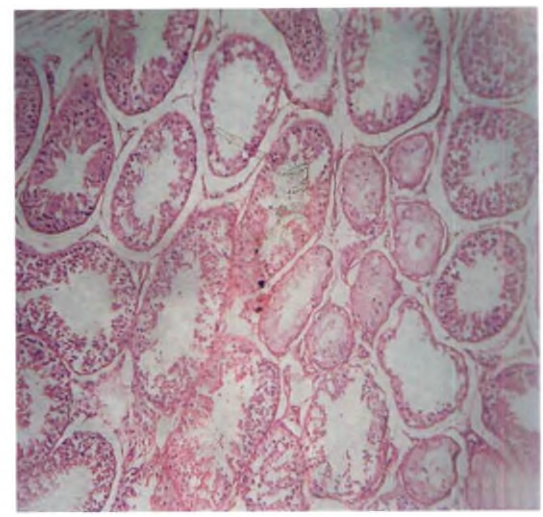

Fig. 29

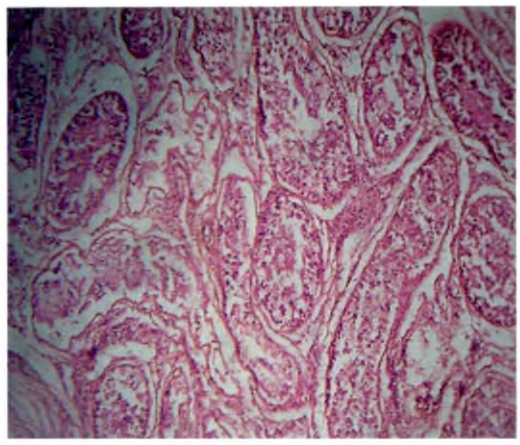

Fig. 30

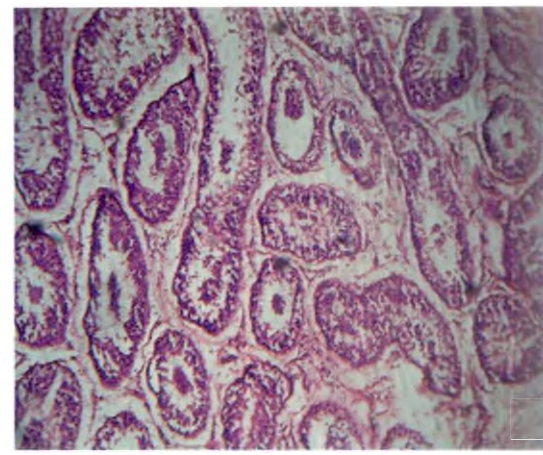

Fig. 31

Figure-28.

Mild degeneration of testis: degeneration of groups of seminiferous tubule- H&E x 250.

Figure-29.

Mild degeneration of testis: vacuolation of cells and nuclear pyknosis- H&E x 250.

Figure-30.

Moderate degeneration of testis: wrinkling of basement membrane, degeneration and hyalinization of the cells- H&E x 250.

Figure-31.

Moderate degeneration of testis: detachment, clumping of cells and formation of giant cells- H&E x 250.

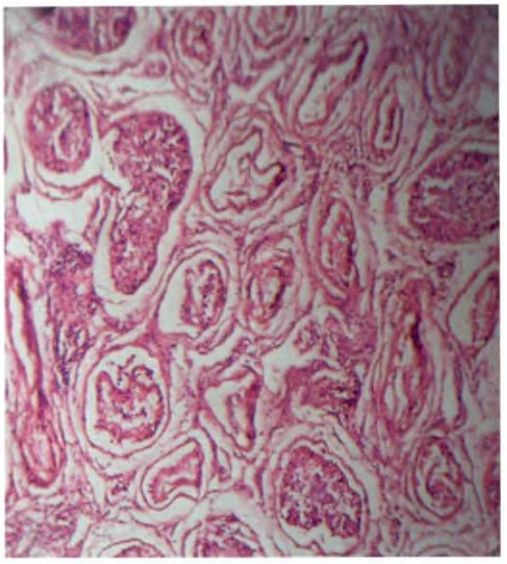

Fig. 32

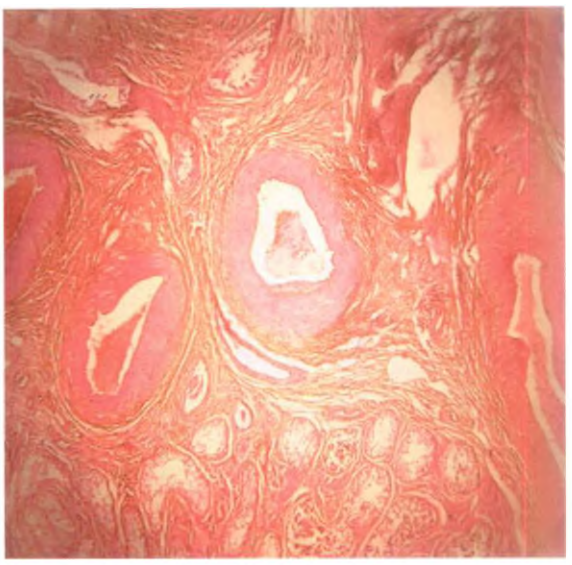

Fig.33

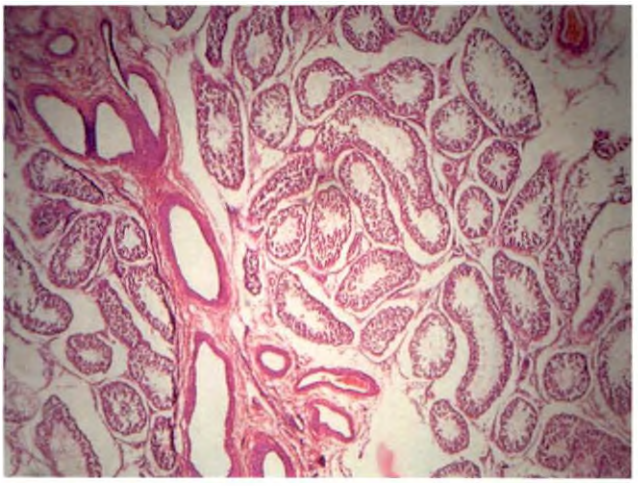

Fig. 34

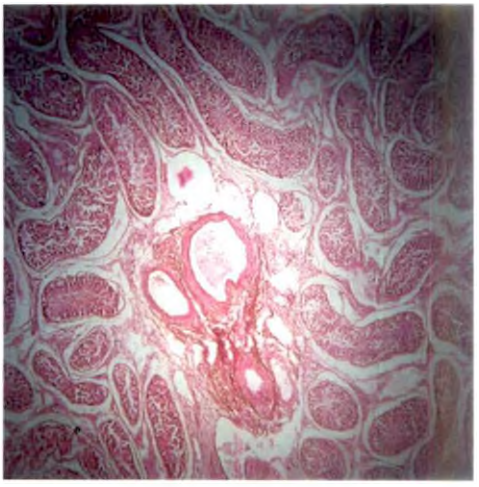

Fig. 35

Figure-32.

Extensive degeneration of testis: most of the tubules with absence of germinal layers- H&E x 250.

Figure-33.

Varicosity of testis: thickening of the tunics and presence of dilated and tortuous vessels in the tunics- H&E x 250.

Figure-34.

Varicosity of testis: presence of dilated and tortuous vessels in the parenchyma- H&E x 100.

Figure-35.

Varicosity of testis: degeneration, desquamation of seminiferous tubules and oedema around the vessels- H&E x 250.

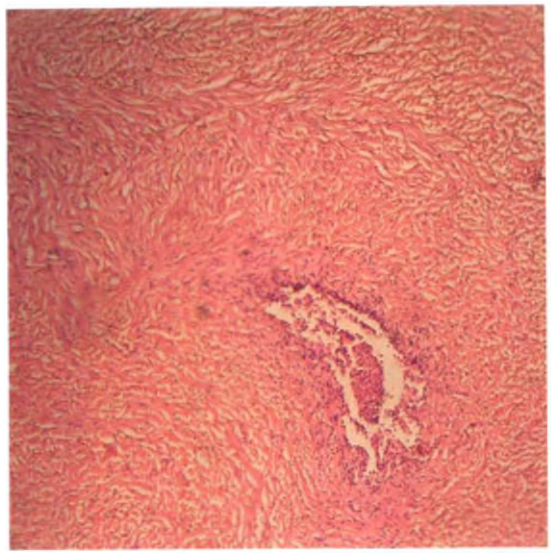

Fig. 36

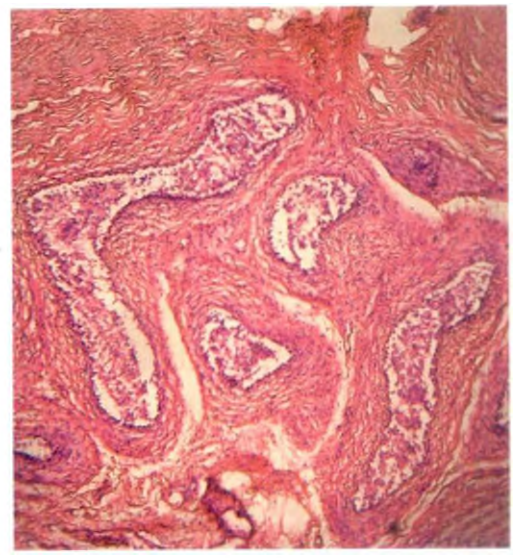

Fig. 37

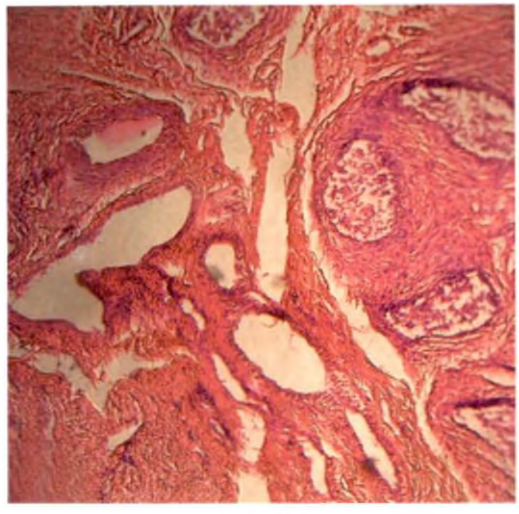

Fig. 38

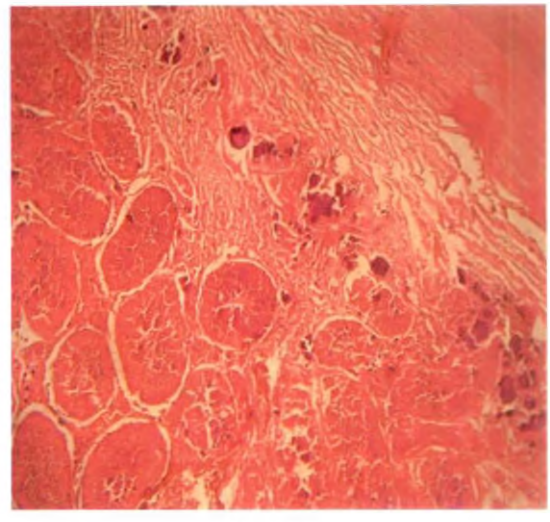

Fig. 39

Figure-36.

Fibrosis of testis: distorted seminiferous tubule with clumps of cells and extensive proliferation of fibrous tissue- H&E x 250.

Figure-37.

Fibrosis of testis: dense fibrous tissue in interstitium and wide separation of tubules- H&E x 250.

Figure-38.

Fibrosis of testis: distorted tubular profile with absence of any stages of the cell layers- H&E x 250.

Figure-39.

Calcification of testis: necrosis and mineralisation of tubules and mineralisation of tunics- H&E x 250.

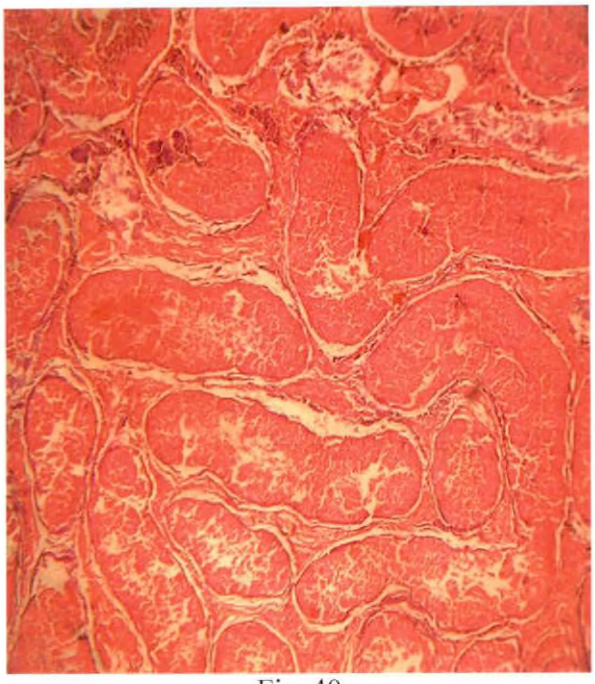

Fig. 40

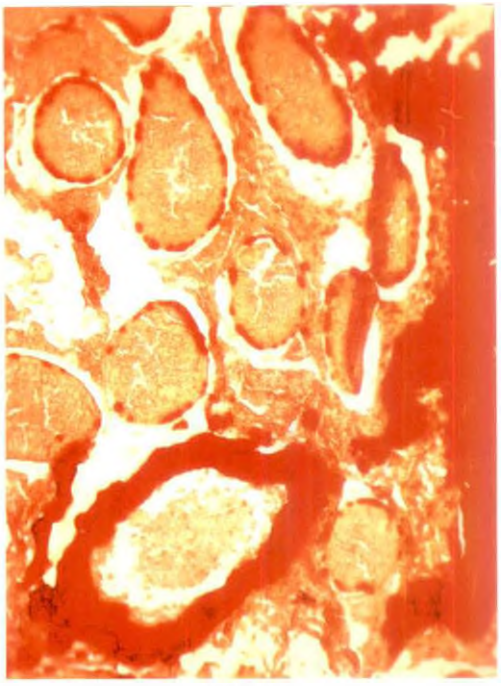

Fig. 41

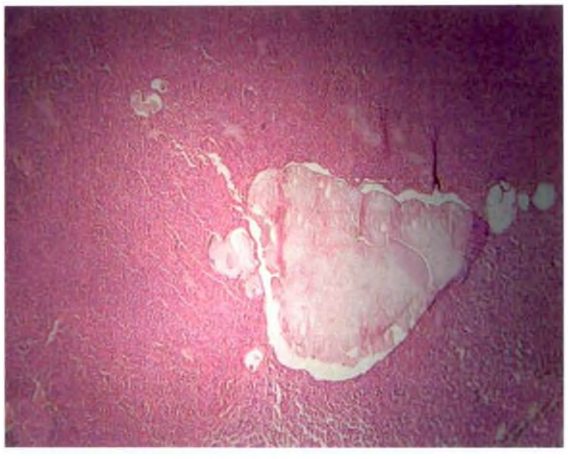

Fig. 42

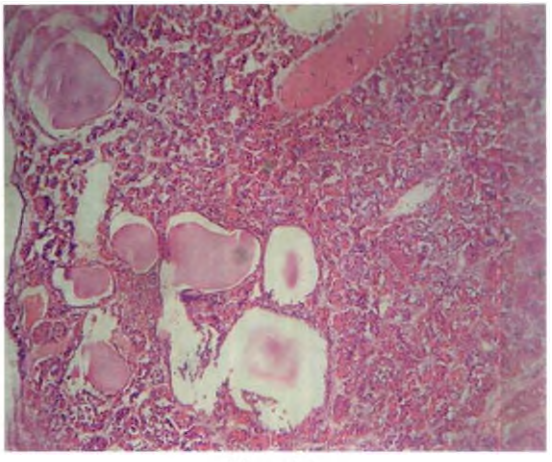

Fig. 43

Figure-40.

Calcification of testis: necrosis of tubular cells, homogenization and intraluminal calcium- H&E x 250.

Figure-41.

Calcification of testis: calcium appeared orange red in the basement membrane and the vessel wall- Alizarin red x 250.

Figure-42.

Cyst-pituitary gland: homogenous pink staining material in the cystic cavity- H&E x250.

Figure-43.

Cyst-pituitary gland: multiple, small to large cyst with or without pink staining materials- H&E x250.

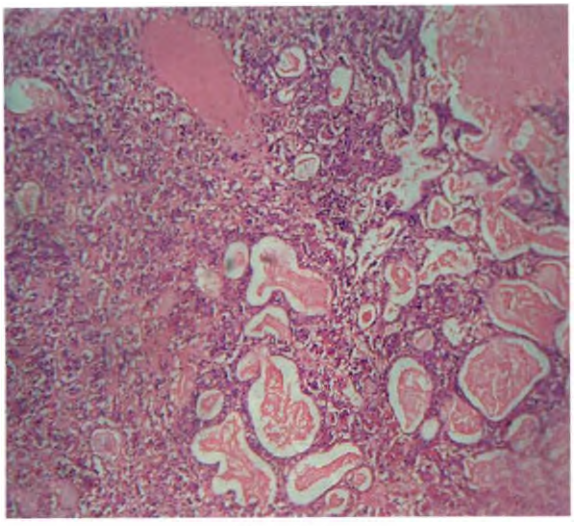

Fig. 44

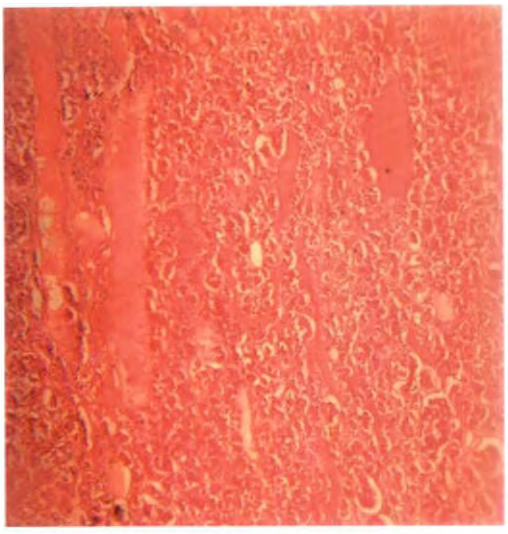

Fig. 45

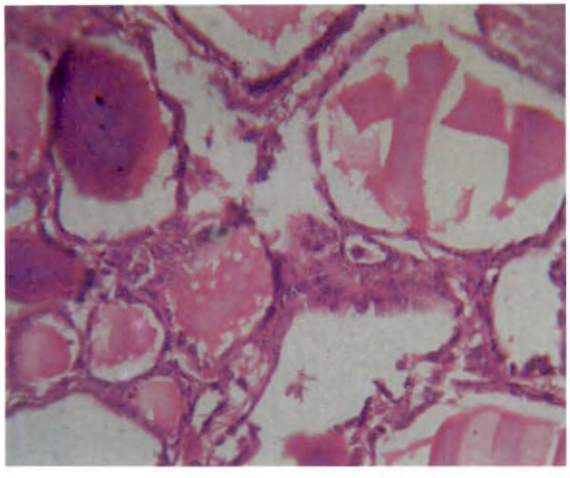

Fig. 46

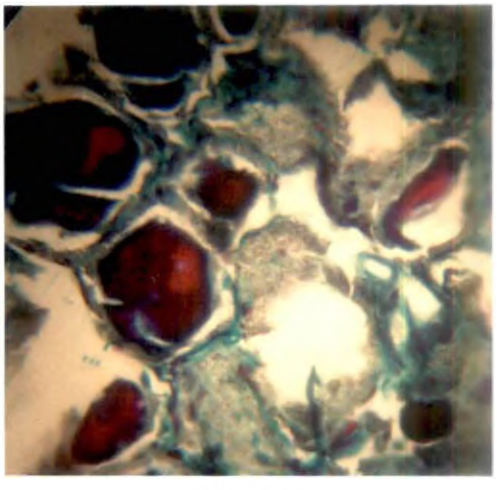

Fig. 47

Figure-44.

Cyst-pituitary gland: Multiple cysts with degeneration, necrosis and hylainization of acidophils- H&E x250.

Figure-45.

Haemorrhage and oedema-pituitary gland- H&E x250.

Figure-46.

Degeneration of thyroid gland: degeneration and desquamation of follicular epithelium- H&E x400.

Figure-47.

Degeneration of thyroid gland: colloid seen red and fibrous tissue seen green-Trichrome x 400.

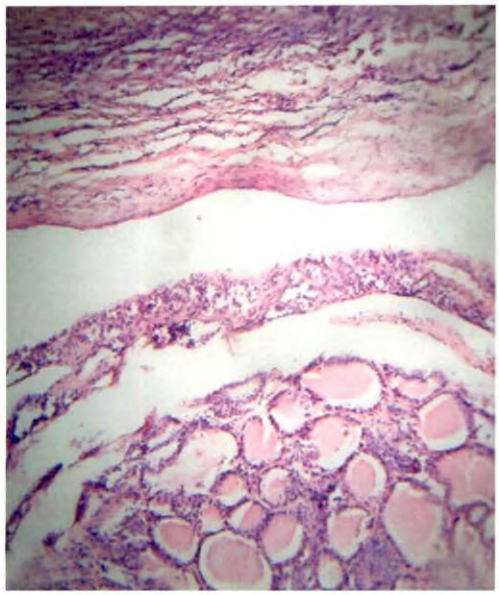

Fig. 48

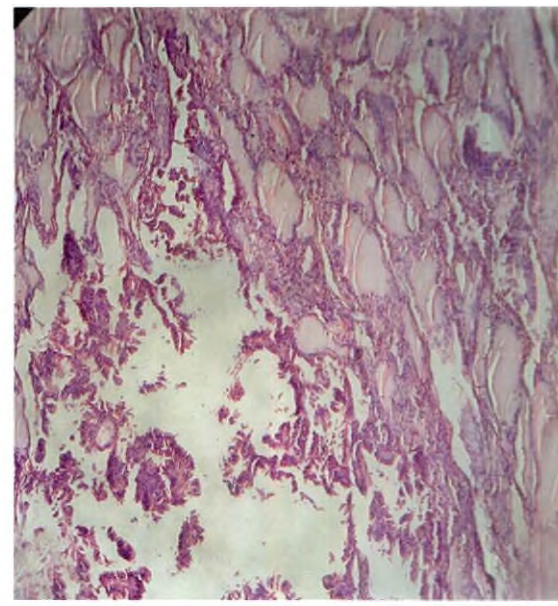

Fig. 49

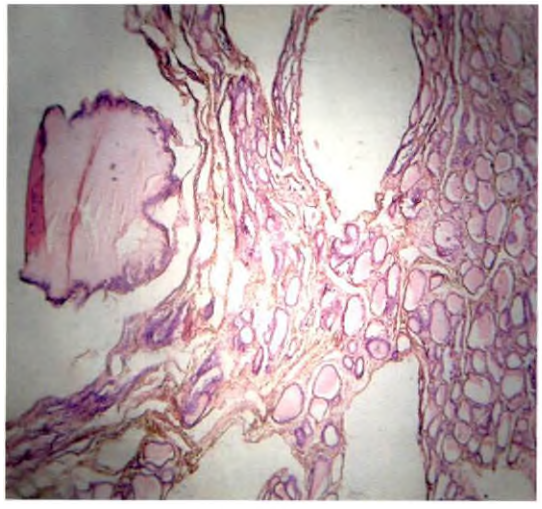

Fig.50

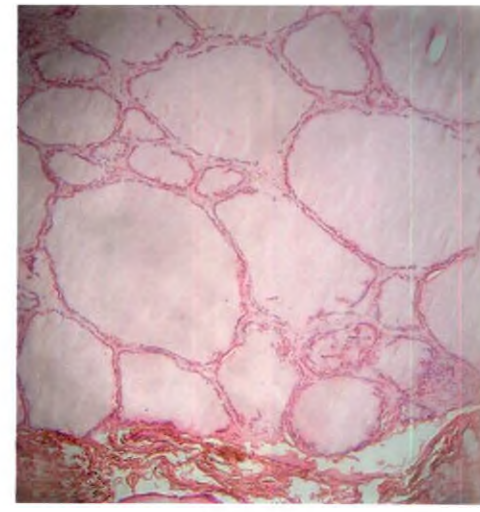

Fig.51

Figure-48.

Nodular goitre: follicles lined by many layers of cells and compression of adjacent thyroid follicles-H&E x 250.

Figure-49.

Nodular goitre: papillary proliferation of lining cells projecting in to the lumen- H&E x250.

Figure-50.

Cyst-thyroid gland: cysts lined by flattened cells with compression of thyroid follicles- H&E x250.

Figure-51.

Colloid goiter: distended follicles lined by flattened cells- H&E x 400.

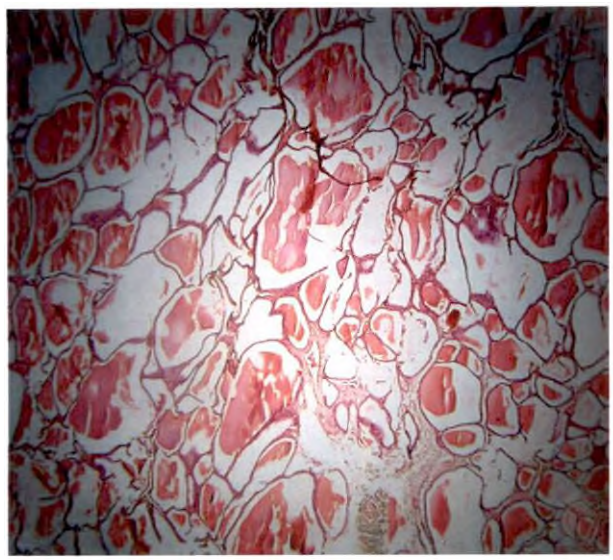

Fig. 52

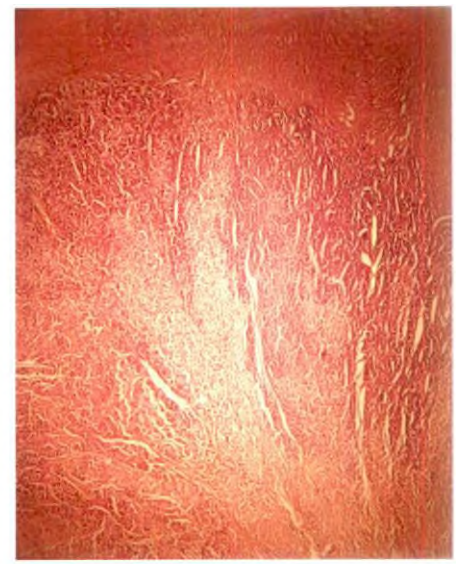

Fig. 53

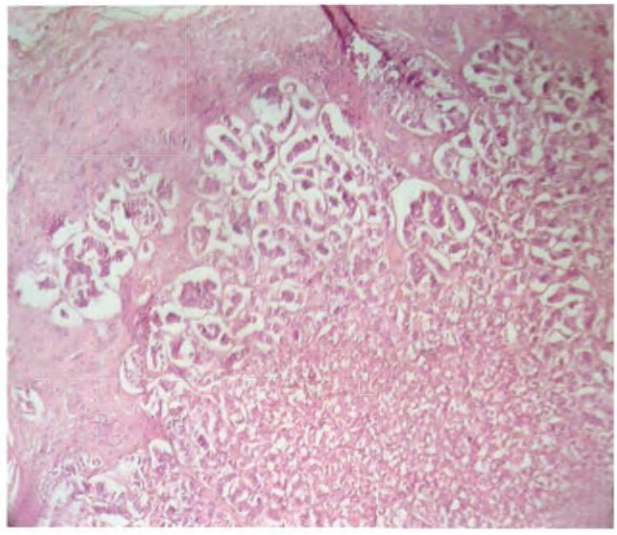

Fig. 54

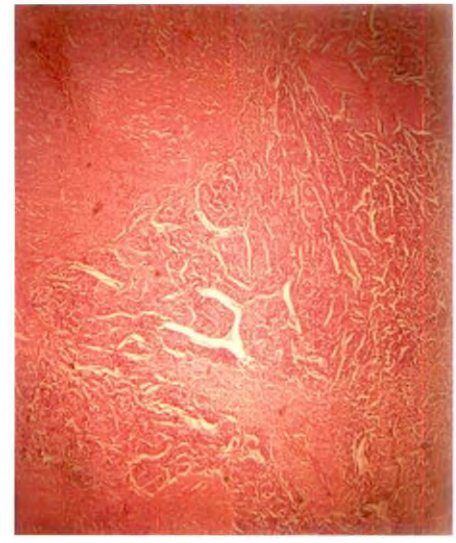

Fig. 55

Figure-52.

Hypoplasia-thyroid gland: follicles with scanty or no colloid and lining cell indistinct- PAS x 250.

Figure-53.

Cortical hyperplasia-adrenal gland: increase in the width of all zones of cortex- H&E x100.

Figure-54.

Cortical hyperplasia-adrenal gland: accessory cortical nodule separated by fibrous tissue- H&E x250.

Figure-55.

Medullary hyperplasia-adrenal gland: distended medullary zone with hyperplasia of cells- H&E x 250.

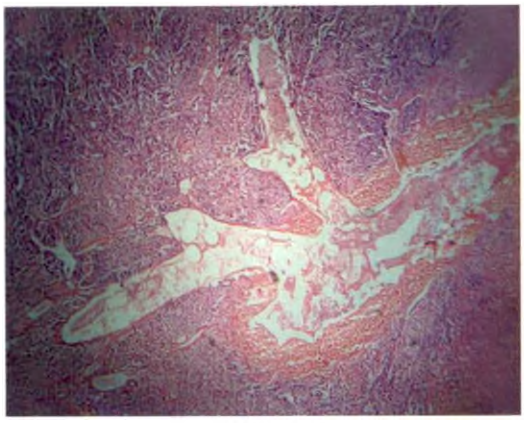

Fig. 56

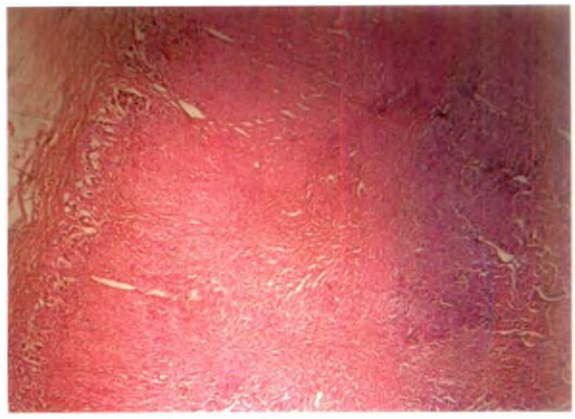

Fig. 57

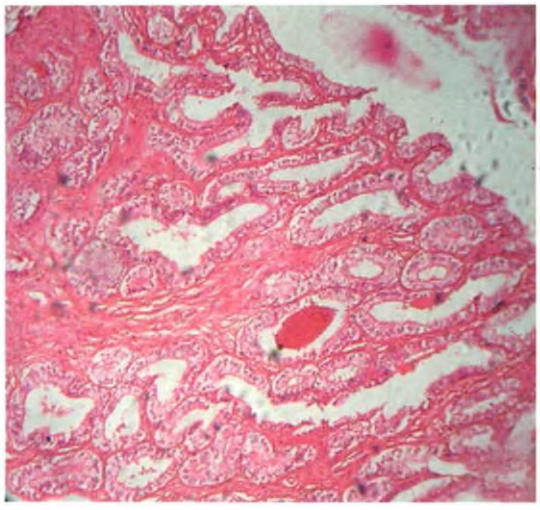

Fig. 58

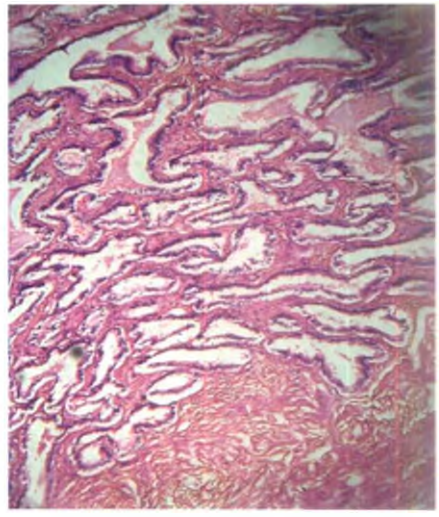

Fig. 59

Figure-56.

Medullary hyperplasia-adrenal gland: dilatation of vessels, sinusoids and haemorrhage in medulla- H&E x250.

Figure-57.

Cortical hypoplasia-adrenal gland: narrow cortical zone- H&E x 250.

Figure-58.

Hyperplasia-seminal vesicle: glandular cells appeared columnar and forming intraluminal projections- H&E x250.

Figure-59.

Hypoplasia-seminal vesicle: reduction in size of glandular lumen and flattened lining cells - H&E x 250.

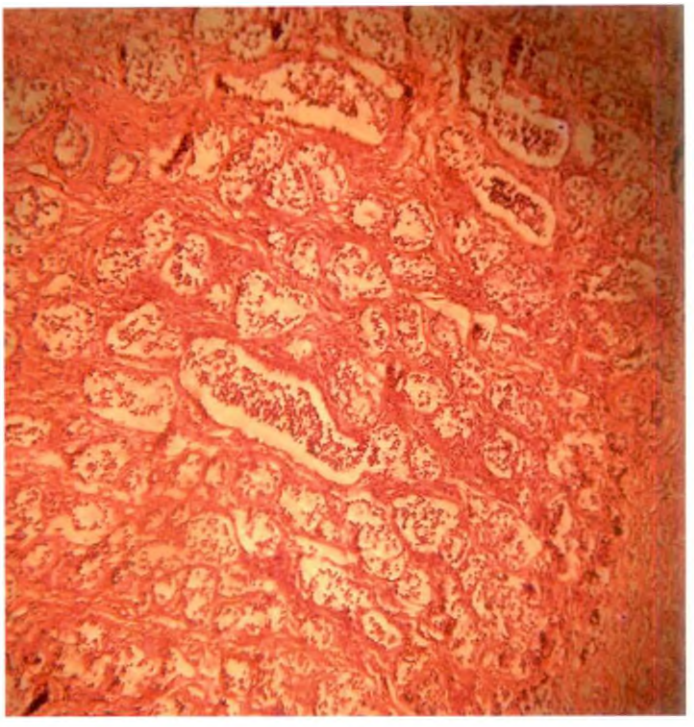

Fig. 60

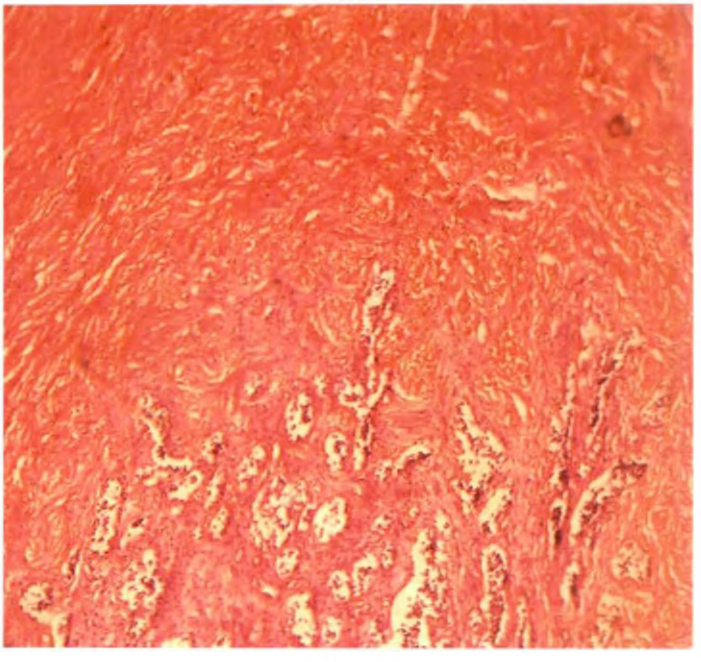

Fig. 61

Figure-60.

Hyperplasia-prostate gland: dilated acini of varying size- H&E x 100.

Figure-61.

Atrophy-prostate gland: collapsed, shrunken acini with fibrous tissue proliferation- H&E x 100.

Discussion

5. DISCUSSION

The studies initiated in the field of pathology of endocrine glands related to the infertility problems in bulls yielded only little information in elucidating bovine infertility. Hence the present investigation was undertaken to evaluate the prevalence and pathological conditions of pituitary, thyroid and adrenal gland in the various forms of testicular disorders in bull.

The systematic gross and histopathological examination revealed testicular lesions of varying forms and degrees to the extent of 17.24 per cent. Ladds *et al.* (1973) observed testicular disorders up to 15.64 percent.

The influence of age and breed on the testicular pathology was studied. The percentage of incidence of testicular disorders in the age groups between 1-3 years, 3-5 years and 5-7 years were 1.15%, 6.51 % and 9.58% respectively. Ladds *et al.* (1973) noticed 1.82 % testicular disorders in young animals, 2.55 % in mature animals and 9.82 % in old animals. The animals brought from the Government farms had higher incidence of testicular disorder in the age group between five to seven years. The higher incidence in the groups might be due to the repeated collection of semen and exhaustion that it caused. The younger animals in the age group of three to five years brought from different places other than the government farms had higher incidence of testicular disorders. This could be attributed to poor management and multiple nutritional deficiencies as most of the animals presented for slaughter had poor body condition. Malnutrition constitutes great stress to spermatogenesis in prepubertal males than in post pubertal males. Energy deficient diets adversely affect gonadotrophin secretion and damage the seminiferous epithelium (McDonald and Pineda, 1989). Jubb *et al.* (1993) reported exhaustion, environmental effects, nutritional and hormonal affections as factors responsible for causing testicular disorders.

Among the breeds the testicular disorders were found higher in Nondescript breeds and Crossbred Holstein-Friesian, which were 36 % and 46 %, respectively. This indicates breed as one of the predisposing factors for testicular disorders. Chaudhuri *et al.* (1982) observed testicular lesions in 40.10 per cent non-descript buffaloes. The higher incidence of testicular disorders in ND breeds might be due to improper selection of pedigree, nutritional deficiencies and stress.

In the present study, the percentages of incidence of various testicular disorders were testicular hypoplasia (4.21%), degeneration (9.96%), varicosity (0.77%), fibrosis (1.15%) and calcification (1.15%). Ladds *et al.* (1973) recorded testicular hypoplasia and atrophy in 1.45 % cases, oedema and thrombosis in 1.09 % cases and calcification in 12.36 % cases of bulls. Galloway (1961) found 0.6 % of testicular hypoplasia and degeneration in 21.47 % in bulls.

Relative weight of the testis in relation to various disorders was recorded in the study. In testicular hypoplasia and degeneration the relative weight was decreased whereas in fibrosis and calcification the relative weight was moderately increased. Despite the findings of testicular hypoplasia and degeneration, increased loss of germinal epithelium was associated with reduction in relative weight observed. Madrid *et al.* (1988) observed smaller testicular size in hypoplasia and degeneration cases due to loss of germinal epithelium. Sreekumaran (2000) found the measurement of scrotal circumference as a predictor of testicular weight in relation to various pathological conditions of the testis. Kodagali *et al.* (1971) observed that the hypoplastic testicles had wide variation in relative weight. Hence the relative testis weight and scrotal circumference might be taken as a criteria for the accurate diagnosis of various testicular disorders as opined by many earlier workers.

The history was collected from the different farms, where in there was a higher incidence of testicular disorders among bulls. It was observed that bulls with various testicular disorders had poor semen quality and sexual libido.

Madrid *et al.* (1988) observed poor semen quality in association with small testes. Ahmad *et al.* (1988) correlated bulls with decreased sexual libido to underdeveloped seminiferous tubules and poor quality of semen to testicular degeneration and fibrosis. Frazer (1968) suggested that the sexual libido in bulls were genetically controlled and may be associated with androgen levels.

In the present study various gross and histopathological lesions of the testis were encountered. Unilateral right-sided aplasia of the testis was recorded in a mature crossbred Jersey bull between the age group of three to five years. Ladds (1973) observed the absence of testis in mature or aged bulls. Grossly the testis was absent and very small fibrous masses were observed near the head and tail of the epididymis. Histologically the fibrous mass contained irregular spaces lined by flattened epithelial cells and hyalinised vessels were seen. The gross and histological appearance was in agreement with the findings of Kaikini and Patil (1978), Chaudhuri *et al.* (1982) and Kay and Meyer (1985). Konig (1964) stated testicular aplasia in bovines is rare in occurrence and usually associated with other anomalies. Konig *et al.* (1972) considered an autosomal recessive gene to be involved in the incidence of testicular aplasia.

Two cases of unilateral hypoplasia and eight cases of bilateral hypoplasia were encountered in the study. The percentage of incidence was 4.44 and 17.78 respectively. The cases were mostly seen in three to five years' age groups. The breeds affected were ND, CBJ and CBHF and least affected was CBBS. This findings correlate with the observation of Sreekumaran (2000) who noticed 4.48 and 13.96 percentage of unilateral and bilateral testicular hypoplasia respectively in CBJ, CBBS and CBHF breeds of bull. Galloway (1961) reported one case of testicular hypoplasia in a young Jersey bull and one case in Hereford cattle. Rao *et al.* (1966) reported testicular hypoplasia in a crossbred Jersey bull of three years of age. Jubb *et al.* (1993) reported that excess scrotal fat leading to thermal degeneration might be one of the reasons for hypoplasia. Lundgren (1972) described inheritance of the condition in Swedish Red-and-White cattle. In the present study, the hypoplastic testis showed small sized seminiferous tubules with

thickened basement membrane histologically. A single layer of Sertoli cells lined the basement membrane in majority of the tubules and the interstitial cells of Leydig appeared predominant. In total hypoplastic condition, the germinal cells were completely absent. Kodagali *et al.* (1971) and Dunn *et al.* (1980) described that the hypoplastic testis contained predominantly of normal appearing Leydig cells and degenerated seminiferous tubules. The vast majority of seminiferous tubules consisted of irregular shaped masses of collagenous connective tissue. Few tubules contained Sertoli cells but no germinal cells were evident. Most of the workers agree that testicular hypoplasia is congenital and possibly hereditary in origin and is caused by marked lack of or reduction in spermatogonia in the gonads during foetal life.

In the present study degeneration of testis was seen in 25 cases. The breeds predominantly affected were ND, CBHF, CBJ and CBBS. It was mostly seen in adult animals. No cases of testicular degeneration were seen below 3 years. Galloway (1961) observed testicular degeneration mostly in older animals. This clearly indicated that the older animals are prone to testicular degeneration compared to young animals probably due to excessive use in service, susceptibility to disease at the time of collection and long survivability. Jubb *et al.* (1993) and Acland (1995) reported that age-associated degeneration of testis might largely be secondary to degenerative vascular lesions within the testis in bulls. Higher incidences of degenerative changes recorded in older animals are in agreement to the above observations. Various acute infections and toxic diseases affecting the testis invariably produce vascular lesions in the testicular tissue (Cohrs, 1967). Degeneration of the epithelium had occurred with hyalinisation of the central mass of debris, sperm and Sertoli cells. There were other areas in the same testicle, which appeared to be producing sperm normally. Cytological abnormalities in the form of vacuolation of spermatocytes, pyknosis and giant cell formation were also evident. Konig (1964) recognised the basement membrane changes as most helpful in differentiating testicular hypoplasia from degeneration. Tubular degeneration and interstitial fibrosis are

the main response of testicular tissue to multiple noxious influences (Knudsen, 1954 and Konig, 1959). Roberts (1971) reported that severe vitamin A deficiency precedes testicular degeneration and poor semen quality. Some of the nutritional deficiency might be prone to various diseases affecting the testis and endocrine glands resulting in various disorders. Mineral deficiencies and the feeding of excessive amount of phytoestrogen, goitrogens, and nitrates are associated with impaired testicular function in males (McDonald and Pineada, 1989). Jubb *et al.* (1993) reported that general or specific nutritional deficiencies or excesses, could lead to testicular degeneration. They reported that the effect on the gonads of hypovitaminosis A was probably indirect and due to suppressed release of gonadotrophic hormone from the pituitary gland. Jubb *et al.* (1993) reported that malnutrition, debility and cachexia and also infectious diseases are common causes of degeneration and atrophy of the seminiferous epithelium causing a suppression of the release of gonadotrophic hormones from the pituitary. They also reported that chemical, metal and rare earth salts can produce degeneration of seminiferous epithelium due to their interference with hypophyseal-gonadal axis. Elhordoy and Cavestany (1986) observed in outbreak of aflatoxicosis with testicular degeneration in bulls.

Localized or systemic diseases of the animal may have contributed to various testicular and endocrine gland disorders observed in the present study. Karagiannis and Harsoulis (2005) reported that the gonadal function was significantly affected in many acute and chronic diseases determined through the integrity of the hypothalamic-pituitary-gonadal axis. In acute stress reactions, the testicular function is harmed indirectly by way of gonadotrophin suppression and directly by the action of cytokines upon the testes. In chronic stress reactions, testicular dysfunction was due to primary testicular failure with reduced production of testosterone and elevated gonadotrophin levels. Garcia *et al.* (1996) described protozoan infections of the male genital tract that are likely to produce testicular damage or secondary hypogonadism by way of hypothalmic-hypophyseal axis alterations due to hypofunction of gonadotropic hormones.

Varicosity of testis was observed in two cases. Mature and older animals were affected. Vigorous exercise and excessive use of the animal at the time of repeated collection of semen might have predisposed the animal to develop severe vascular lesions of the testis.

Fibrosis was observed in three cases. The breeds affected were ND, CBHF and CBBS. Fibrosis was seen in the age group of above five to seven years. The tunics were thickened. The testis was too hard in consistency. The cut surface was dark brown and white streaks seen within the parenchyma. Similar conditions were recorded in older bulls (Raja *et al.* (1973), Rao and Rao, (1979) and Jubb *et al.* (1993). Such fibrosis may be the end result of inflammatory processes, degeneration and hypoplasia (Konig, 1959).

In this study testicular calcification was reported in four cases. The breeds affected were CBJ, CBHF, CBBS and ND. Calcification was seen in animals of five to seven years of age. The testis appeared wrinkled and the glistening appearance disappeared. The affected part was whitish in colour. The calcium salts were deposited in the stroma and in the tunics of the testis. There was calcium deposition on the basement membrane resulting in increased thickness. Calcification was also observed in an intraluminal part of the seminiferous tubules. Maurya *et al.* (1968) found that the tunica albuginea in the affected testicle was thickened and the glistening appearance was absent. Jubb *et al.* (1993) attached little significance to the occurrence of calcification of tubules in mature and old bulls and according to them necrosis and calcification of testis seen in majority of cases might be due to infectious process. Bargni *et al.* (1984) observed calcification of necrotic areas of seminiferous tubules in ten beef bulls infected with Besnoitia. Calcification of dystrophic type was due to deposition of crystalline calcium phosphate in dead or dying tissues (Vegad, 2004).

In the present study various lesions in pituitary, thyroid and adrenal glands were encountered along with different types of testicular disorders in bull. The testicular hypoplasia and degeneration was seen in the bulls affected by

pituitary gland cysts. This indicated that the cyst in the pituitary gland interfered in the gonadotrophic hormone secretion leading to degeneration of seminiferous epithelium. The mean pituitary gland weight was higher in the testicular hypoplasia cases due to pituitary cysts than the other conditions affecting testis. This observation is in accordance with the observation of Galloway *et al.* (1992) where in the mean pituitary gland weight of the rams were significantly higher in testicular hypoplasia than in normal rams. Ahmad *et al.* (1985) indicated the abnormalities in the hypothalamic-hypophyseal-gonadal control of postnatal reproductive development. Capen (1993) described that pituitary cyst may develop from remnants of the craniopharyngeal duct, which normally disappears by birth in most of the animal species. The cyst leads to hypofunction of adenohypophysis resulting in gonadal atrophy. In the present study, pituitary cysts were seen in three regions of pituitary gland. These cysts, which caused compression of the adjacent pituitary cells might have contributed to gonadal dysfunction. Jubb *et al.* (1993) reported that the structures adjacent to the cysts showed atrophy of varying degrees owing to compression and interference with the blood supply. Capen (1993) stated that the disruption of a large cyst with escape of the proteinaceous contents into the adjacent tissues might incite an intense, local inflammation with subsequent fibrosis that may interfere with pituitary function leading to gonadal atrophy. Due to functional abnormalities of the pituitary gland gonodotrophin secretion was decreased resulting in the gonadal dysfunction.

In the present study, haemorrhage, congestion and oedema of the pituitary gland was seen in the testicular hypoplasia, degeneration, fibrosis and calcification cases. In such cases hyperplasia of thyroid gland and adrenal gland were also noticed. This indicated that the pituitary haemorrhage might cause secondary lesions in the testis, thyroid and adrenal gland. The haemorrhagic lesions of pituitary gland might be due to inflammatory or infectious diseases affecting the animals. Schmidt and Wallace (1998) reported pituitary apoplexy following sudden enlargement of pituitary gland in human beings. Other factors

responsible for haemorrhages are pituitary adenomas, anticoagulants, arterial or intracranial hypertension as well as traumatic injury. Veldhuis and Hammond (1980) reported an incidence of Growth Hormone (GH) deficiency of 88 %, secondary adrenal deficiency of 66 %, secondary hypothyroidism of 42 % and hypogonadotrophic hypogonadism of 67 % as a result of pituitary apoplexy in human patients.

In majority of testicular disorders like hypoplasia, degeneration, varicosity, fibrosis and calcification various lesions in the thyroid gland could be detected. The hypoplasia, hyperplasia, cystic goitre, colloid and nodular goitre observed in the various testicular disorders clearly indicated that there was correlation between them (Sreekumaran, 1976, Reddy, 1982 and, Sharma and Ramkumar, 2001). Jubb *et al.* (1993) reported that hypofunction of thyroid gland, in long standing cases leads to marked atrophy of the spermatogenic epithelium of the testis. It may result in the lack of libido and reduction in sperm count. The primary function of the thyroid hormone was considered as regulation of cellular oxidation and stimulation of oxygen consumption for normal growth and development. Calderbank (1958) reported that the high producing animals and breeds would be susceptible to a deficiency of iodine most frequently evinced by reproductive disturbances. Fritz *et al.* (1976) reported a familial incidence of interstitial, lymphocytic orchitis associated with testicular atrophy and reduced fertility in inbred beagle dogs with lymphocytic thyroiditis. In the absence of sufficient amount of thyroid hormones in circulation, gonadotrophic hormones of pituitary probably could not carryout its normal functions effectively leading to degenerative changes of the testes (Gorbaman and Bern, 1974). Therefore the different types of goitre definitely influence the pituitary gonadotrophic hormones leading to testicular degeneration. Abraham *et al.* (1987) reported thyroid hyperplasia and changes in basophils of pituitary gland in experimental hypothyroidism and also observed degenerative changes in testis. In the present study, eight cases of hyperplastic conditions of thyroid glands revealed testicular degeneration with extensive affection. Sreekumaran (1976)

recorded hyperplasia of the thyroid gland in cases of testicular degeneration in induced hypothyroidism in goats. Reddy and Rajan (1985) also reported similar pathological findings in experimental hypothyroidism in goats. A similar atrophic change in the testis was observed in the present study with goitre. Abraham *et al.* (1987) reported an increase of relative weight of the adrenal glands in experimentally induced hypothyroidism in male calves.

In thyroid hypoplasia there was testicular aplasia, hypoplasia and degeneration. Jubb *et al.* (1993) reported that hypo function of thyroid gland, in long standing cases leading to marked atrophy of the spermatogenic epithelium of the testis. It resulted in the lack of libido and reduction in sperm count.

In majority of testicular degenerative changes, seminal vesicles showed hyperplastic changes with papillary projections with concomitant hyperplasia and fibrosis of prostate. The thyroid glands were hyperplastic in these cases. Jubb *et al.* (1993) reported hyperplasia of thyroid gland in cases of atrophy of both testes and accessory sex glands in goats.

In the present study, the lesions encountered in adrenal gland were cortical hypoplasia, cortical hyperplasia and medullary hyperplasia. Cortical hypoplasia and cortical hyperplasia of adrenal was encountered in testicular hypoplasia and degeneration. Proliferation and depletion of fat in the cells of zona fasciculata was recorded and a significant decrease in the relative weight of testes noticed. Medullary hyperplasia was evident in testicular degeneration, fibrosis and calcification. The cortical and medullary hyperplasia might be due to various stress factors concurrently affecting the adrenal. Adrenal cortices normally produce androgens, and adrenal androgens secretion might be greatly increased in a variety of pathological condition including adrenal hyperplasia (McDonald and Pineda, 1989). Mudge (1955) attributed adrenal hyperplasia to a metabolic block in the normal synthesis of adrenal steroids, with resultant accumulation of androgenic steroids resulting in the testicular disorders via pituitary-gonadal axis. Nair *et al.* (1981) recorded cortical hyperplasia of the

adrenal gland in five years old buck, but no changes were noticed in testes and pituitary gland.

A number of other environmental chemicals have been implicated as endocrine disruptors. The mechanisms of action of these compounds or substances are not well known but they may involve in antiandrogenic activity; modulatory effects on enzymes controlling sex hormone metabolism; or direct influence on the hormone-producing organs such as the thyroid gland, pituitary gland, and adrenal glands. These compounds may also affect estrogen levels through indirect feedback mechanisms. Research on possible reproductive effects in human and animal were limited (Toppari *et al.*, 1996).

Various chemicals, toxic plants, insecticides, pesticide and other environmental pollutants also might have played a vital role in the production of endocrine and testicular disorders of bull observed in the present study. The interrelationship of thyroid gland and testis due to fluorosis through water and forage crops might play a role in the pathology of both organs. The research indicated that fluoride exposure at lower doses in animals could cause toxic effects to Sertoli cells and gonadotrophs, reduction in circulating testosterone, and reductions in total fertility rate. In Kerala toxic levels of fluoride in ground water sources are high (Gopakumar *et al.*, 2003) and the consumption of water and forage crops might be important factor for the development of thyroid and testicular disorders in bull. Cheeran *et al.* (1987) found fluoride intoxication in cattle in most part of Kerala especially nearby industrial areas. They found more than permitted level of fluorine in the water sources. Permitted level of fluorine by the World Health Organisation (W.H.O) is 0.5 to 1 ppm. Gopakumar *et al.* (2003) found pasture contaminated with fluoride to be the potential source of fluoride toxicity in cattle in the study areas of Kerala. Cattle and sheep were most susceptible to fluorine toxicosis among farm animals. Ghosh (2002) concluded that fluoride treatment was associated with testicular disorders, which may be due to induction of oxidative stress in reproductive organs along with possible adverse effects of fluoride on pituitary-testicular axis. Mahmood (1996)

evaluated the effect of sodium fluoride on the thyroid glands and testis of guinea pigs. In thyroid gland there was depletion of colloid from the follicle, shrinkage of follicle, disruption of follicular basement membrane associated with oedema, degeneration of the follicular epithelial cells, increased follicular vascularity and fatty degeneration in the interfollicular connective tissue. Testicular section revealed dilated seminiferous tubules and few mature luminal spermatozoa. Mikhailets *et al.* (1996) found excess of fluorine in drinking water as a risk factor for the rapid development of thyroid pathology in human beings. They found elevated production of thyroid stimulating hormone (TSH) and a decrease in the concentration of T3 hormone due to elevated fluoride content. The anterior pituitary gland releases TSH to direct the thyroid gland to manufacture thyroid hormone, but if the thyroid is sluggish in its response, then the pituitary will release excess TSH and stimulate further thyroid activity. Primary functions of T3 are to regulate carbohydrate and protein metabolism in all cells. T3 also may interact with and modulate the action of other hormonal system such as growth hormone and steroids. Therefore, diminished T3 hormone secretion due to flourosis may inhibit metabolic and growth function of organs. Jannini *et al.* (1995) indicated Sertoli cells as the major targets of T3 binding in the testis. These cells, along with gonocytes, in the seminiferous epithelium of the testis are critical for normal sperm maturation. Pesticides applied to crops and livestock can remain on or in food and act as endocrine disruptors. Exposures to these contaminants result in endocrine disruption ultimately leading to higher incidence of pathological lesions in the related organs. Gopakumar *et al.* (2003) found application of higher levels of pesticides and insecticides in the forage crops in Kerala and reported the various reproductive disorders associated with the toxicosis. Jubb *et al.* (1993) reported that long-term perturbations of the pituitary-thyroid axis by various xenobiotics predispose the animal to a higher incidence of proliferative lesions in testis than in human beings. Research on wildlife has shown that endocrine disrupting chemicals profoundly impair animal reproduction and development (Toppari *et al.*1996). The overall high level of testicular disorders seen in this study also indicate that there is need for

evaluating the water quality and presence of noxious and toxic substances like excess fluorine for ruling out the involvement of this in the causation of testicular and endocrine pathology.

The present investigation undertaken has made it possible to identify the various endocrine lesions associated with testicular disorders in bulls. It was observed that in many cases there was a co-existence of lesions in the testis along with either or the pituitary, thyroid and adrenal glands. Future studies should be aimed at identifying causative factors which contribute to these disorders.

Summary

6. SUMMARY

An investigation was undertaken to assess the prevalence and pathology of pituitary, thyroid and adrenal glands in testicular disorders of bulls. The testicular lesions were classified based on gross and histopathological lesions and were correlated with the concurrent lesions of pituitary, thyroid, adrenal and accessory sex glands. Out of 300 animals screened for testicular disorders in this study, 261 cases of testes, pituitary, thyroid, adrenal and accessory sex glands were examined for any gross lesions. Forty-five samples revealed presence of testicular disorders in bulls to the tune of 17.24 per cent indicating that the disorders of testis in bulls are more than what is generally expected.

A higher incidence of testicular disorders in age group of five to seven years were observed in animals brought from the Government farms and whereas in the different places other than government farms the testicular disorders were more common in young animals (three to five years).

Among breeds, testicular disorders were found higher in ND and CBHF and the prevalence of the disorders were 36 % and 46 % respectively. These breeds found to be more susceptible to various testicular disorders.

History of the bulls from government farms revealed decreased libido and congenital anomalies. The semen of the bulls showed different abnormalities as watery in consistency, reduced concentration, and motility and defective sperms.

Relative weight of the testis in relation to age was studied and the relative weight was found decreased in hypoplasia and degeneration and increased in varicosity, fibrosis and calcification.

Gross pathological changes of testis revealed aplasia (1), hypoplasia (10) which was bilateral (8) or unilateral (2), degeneration (25), varicosity (2), fibrosis (3) and calcification (4). Pituitary gland revealed cyst and haemorrhage.

Hypoplasia, hyperplasia, cyst and nodular goitre were the thyroid lesions. Adrenal glands revealed hyperplasia and hypoplasia. Hyperplasia and hypoplasia of seminal vesicle, and hyperplasia and atrophy of prostate gland were also recorded.

Histopathological lesions were classified according to various changes observed in the testis. In testicular aplasia, total absence of seminiferous tubules with proliferation of fibrous tissue were the features. In hypoplasia, very small to medium sized seminiferous tubules with predominance of interstitial cells and basement membrane lined by Sertoli cells were observed. Testicular degenerative change was classified as mild, moderate and extensive degrees. In varicosity, dilatation of blood vessels in the tunica vaginalis with degeneration, desquamation of the seminiferous epithelium were noticed. Fibrosis was characterised by the loss of normal architecture of tubules with dense fibrous tissue proliferation. Calcification revealed thickening of basement membrane and intraluminal mineralisation with necrosis of seminiferous tubules. Degeneration and hypoplasia of testis were predominant findings in the present study.

Histological lesions of pituitary gland were cysts of either solitary or multiple cysts. Small or large cyst was observed in the pars distalis, pars intermedia and pars nervosa associated with degeneration, necrosis, hyalinisation and haemorrhage. The acidophils and basophils revealed diffuse hypertrophy.

The thyroid gland revealed hypoplasia, degeneration, cyst, colloid goitre and nodular goitre. Micro follicles without colloid and follicular epithelium lined with the flattened cells were characteristic lesions in hypoplasia of thyroid gland. Irregular follicles with focal degeneration and desquamation of follicular epithelium were noticed in degenerative changes of thyroid. In cystic condition of the thyroid gland, the follicles were irregular in outline with scanty colloid. Distended follicles lined by flattened cells and filled with pale staining colloid were recorded in colloid goitre. Varying sized follicles which coalesced to many nodular aggregates were characteristic of nodular goitre. Among the adrenal

lesions, cortical hypoplasia, cortical hyperplasia and medullary hyperplasia were noticed. There was reduction in various zones in cortical hypoplasia. Accessory cortical nodule separated by fibrous tissue widening of all the zones was noticed in cortical hyperplasia. In medullary hyperplasia, narrowing of cortical segment with vascular congestion and haemorrhage.

In the accessory sex glands, the lesions observed were hypoplasia and hyperplasia of seminal vesicle; and hyperplasia and atrophy of prostate gland. Proliferation of glandular cells of seminal vesicle appeared columnar with intraluminal projections seen in hyperplasia. In seminal vesicle hypoplastic lining cells were flat and reduction in size of vesicular lumen without secretory activity evident. In prostate gland, hyperplasia and atrophy were the lesions observed. Dilated acini of varying sizes lined by flattened epithelium were seen in prostatic hyperplasia. In atrophy of prostate there was collapsed acini with fibrous tissue proliferation in the interstitial tissue seen.

The correlation of testicular disorders with endocrine lesions revealed that in many cases there was co-existence of lesions in the testis as well as the endocrine glands studied. Cyst in the pituitary gland was recorded in cases of testicular hypoplasia and degeneration. Haemorrhage, congestion and oedema of pituitary gland were observed in cases of testicular hypoplasia, degeneration, fibrosis and calcification. In thyroid hypoplasia, there was testicular aplasia, hypoplasia and degeneration. Thyroid degeneration was seen in testicular degeneration, varicosity, fibrosis and calcification. Thyroid cyst were seen in cases with testicular hypoplasia and degeneration. Colloid goitre was seen in testicular degeneration. In nodular goitre there was testicular hypoplasia and degeneration encountered. In majority of pituitary and thyroid gland lesions the prominent testicular changes encountered were hypoplasia and degeneration. In adrenal cortical and medullary hyperplasia the testicular lesions were degenerative in nature. Fibrosis and calcification of testis were evident in medullary hyperplasia.

The seminal vesicle and prostate gland were comparatively less affected in testicular disorders.

The correlation study has brought into light the fact that the endocrine gland disorders has concurrent affections of the testes with different intensities in cases.

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ABSTRACT

The present investigation was undertaken to evaluate the prevalence and nature of various pathological conditions of the testis and correlate with the lesions of pituitary, thyroid, adrenal and accessory sex glands. A total of 300 bulls were screened for testicular disorders at the Corporation slaughterhouse, Thrissur and Meat technology unit, Kerala Agricultural University, Thrissur. A detailed systemic examination of 261 cases was made during the period of investigation and the gross and histopathological lesions were studied in detail, classified and documented. The study revealed a high prevalence (17.24 %) of testicular disorders in bulls. It was observed that in the Government farms, bulls of the age group above five to seven years were most commonly affected due to excessive usage for semen collection. In other than government farms, bulls of the age group three to five years were highly affected and attributed to severe nutritional deficiencies. Among the breeds, crossbred Holstein-Friesian (CBHF) and Nondescript (ND) was found to show the higher predisposition to testicular disorders. The relative weight of the testis, was decreased in hypoplasia and degeneration, increased in varicosity, fibrosis and calcification. Out of 45 testicular disorder cases, the conditions observed were aplasia (1), hypoplasia (10), degeneration (25), varicosity (2), fibrosis (3) and calcification (4) respectively.

Among the endocrine glands, lesions observed were pituitary cyst (9), pituitary haemorrhage (7), thyroid hypoplasia (7), thyroid degeneration (10), thyroid cyst (5), colloid goitre (3), nodular goitre (2), adrenal cortical hypoplasia (2), adrenal cortical hyperplasia (7) and adrenal medullary hyperplasia (12) respectively.

Among the accessory sex glands, lesions observed were hyperplasia of seminal vesicle (8), hypoplasia of seminal vesicle (6), prostatic hyperplasia (2) and prostatic atrophy (5) respectively.

Correlation study has highlighted that in many of the testicular disorders there were concurrent affections of endocrine glands particularly pituitary, thyroid and adrenal glands which is of major significance.