STUDIES ON
THE PATHOLOGY OF TESTIS AND EPIDIDYMIS OF BUCKS

BY

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THESIS

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DECLARATION

I hereby declare that the thesis entitled "STUDIES ON THE PATHOLOGY OF TESTIS AND EPIDIDYMIS OF BUCKS" is a bona fide record of research work done by me during the course of research and that the 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.

JOSEPH MATHEW

Mannuthy,

6-10-1976.

CERTIFICATE

Certified that this thesis, entitled "STUDIES ON THE PATHOLOGY OF TESTIS AND EPIDIDYMIS OF BUCKS" is a record of research work done independently by Sri. Joseph Mathew, 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

INTRODUCTION

Infertility or sterility in animals has been known to produce severe economic set backs in the livestock industry all over the world. Though Zschokke, as early as 1900, opined that the bull played an important role to cause infertility in cattle, there were not many to agree on this. Poulson (1906) and Albrechtsen (1908) reported that the problem of infertility was extremely uncommon in bulls. Richter (1926) attributed the failure of conception even with one normal service, solely to some defects on the female side. However, Stalfors (1912), Sand (1914), Hess (1920) and Wester (1921) expressed the view that males could also probably play a part, though insignificant, to bring about infertility in animals. It was Williams (1921) who stated catagorically that the bull was probably as great an offender as the cow to cause infertility in dairy herds. This seemed to have inspired the interest to study the problems of male infertility in U.S.A. and during the next few years some good work was published particularly concerning improved methods of investigation of seminal micropathology of bulls (Williams, 1922; Williams and Savage, 1925; 1927). The line of investigation till then was limited to semen evaluation with special reference to the presence of abnormal sperms, and no attempt was made to correlate the seminal changes with the histological changes

of the testis. In 1929, Lagerlof initiated research, linking the morphological changes of the sperm with structural alterations of the testicles of sterile and subfertile bulls. The series of devoted research work subsequently carried out by him, not only laid a strong foundation for the study of male infertility but also established unequivocally the role of males in producing subfertility in animals (Lagerlof, 1934; 1936; 1938).

With the ever increasing use of artificial insemination in all species, many more females are being exposed to the potentialities of individual male. Therefore, the degrees of fertility in male become correspondingly more important. Since cattle continue to occupy a pivotal position among livestock, most of the work relating to male infertility has been done in this species (Lagerlof, 1934; 1936; 1938; Blom & Christensen, 1947; 1956; 1960; Galloway, 1961; Lagerlof, 1963, Blom & Christensen, 1972; Ladds et al. 1973). Humphrey and Ladds (1975) have done a yeoman service in compiling the reports published over a period of ten years on the pathological conditions of testis and epididymis of bulls.

Several studies have also been carried out, especially in Australia on the problems of infertility in rams (Gunn, 1942;

Moule, 1950; Miller & Moule, 1954; Osborne, 1959; Smith, 1962; Murray, 1969; Watt, 1970; 1971; 1972; Galloway, 1973). In an exhaustive review on this subject, Watt (1972) revealed that the pathological conditions of testis and epididymis played an important role to produce infertility in rams.

The problem of infertility was reported to be very common and severe in bucks (Lagerlof, 1938; Williams, 1943; Blokhuis, 1962; McEntee, 1973). Still surprisingly enough, there are only very few reports concerning the incidence, nature and magnitude of prevalence of infertility of bucks, in English literature (Rollinson, 1950; Fraser & Wilson, 1966; Fraser, 1971). However, a good amount of work on this subject has been done in Germany and other neighbouring countries (Richter, 1919; Ott, 1937; Rosenberger, 1951; Schonherr, 1956; Widmaier, 1957; Loliger, 1957; Haugen, 1960; Weber, 1969).

Richter (1919) contributed a highly informative monograph on the problems of sterility in male goats, and showed clearly the need for maintaining the reproductive health of the bucks in goat husbandry. Ott (1937) reported infertility in 20-25 per cent of male goats. Of 501 bucks studied by Rosenberger (1951), 11.8 per cent were found to be sterile. Blokhuis (1966) stated that the incidence of infertility of bucks in the Netherlands was about 10 per cent.

The magnitude of prevalence of infertility in males of different breeds of goats in Germany was recorded by Loliger (1957). The incidence was 15.3 per cent in White German Improved, 18 per cent in Fawn German Improved, 24.3 per cent in Toggenburg and only 2.4 per cent in Swiss Verzasca goats. Out of 966 young bucks of three polled breeds, examined by Weber (1969), 16.8 per cent were sterile but he could not identify a single case of infertility among 175 bucks of the horned black Verzasca breed. It was believed that some linkage existed between sterility condition and polledness (Asdell, 1946; Koch, 1963; Weber, 1969).

Goat husbandry is an important livestock enterprise in Kerala. According to livestock census (1972), goat population in Kerala is 1.5 lakhs, which stands second to cattle population. The one and only breed of livestock that Kerala possess is a breed of goat viz., Malabari goat. Recently a lot of emphasis has been given for the improvement of this breed by cross-breeding with Saanen and Alphine.

Studies pertaining to reproduction of Malabari bucks were mostly confined to fix the norm on the physico-chemical characteristics of semen (Kurian & Raja, 1965; Patil, 1970; Patil & Raja, 1973a; 1973b) and to evolve a suitable diluent for its preservation (John & Raja, 1973). Recently

investigations on the post natal development of testis (Unnikrishnan, 1975) and epididymis (Harshan, 1975) were undertaken. No work has hitherto been conducted to study the problems of infertility in bucks.

The present work was therefore taken up with the object of assessing the incidence and nature of pathological conditions of testis and epididymis of bucks by gross and histopathological examination of the organs collected at random from the abattoir.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Prior to the work of Lagerlof (1934) studies on testicular pathology were generally confined to the more obvious and severe clinical lesions such as orchitis and fibrosis. A major break through in the studies pertaining to male infertility was achieved when Logerlof (1936) conclusively established the significant correlation between the semen characteristics and the conditions of the seminiferous tubules of the testis.

Lagerlof (1938) classified the causes of infertility in males as: Reduced to complete lack of sexual desire; Incapacity to copulate (Impotentia coeundi) and Incapacity to fertilise (Impotentia generandi). Of these impotentia generandi characterized by the insufficient number of functional spermatozoa passing off in the ejaculate was considered to be the most important from the point of view of breeding.

Of the various developmental anomalies that bring about impotentia generandi, testicular hypoplasia assumes paramount importance. Testicular hypoplasia has been observed in all species of domestic animals. However, most of the work pertaining to hypoplasia was done in bulls (Lagerlof, 1934; 1936; 1938; Blom & Christensen, 1947; Laing & Young, 1956; Kanagawa, 1961; Donaldson, 1963; Carrol et al. 1963; Kodagali, 1966; Kodagali & Kerur, 1968; Carrol & Ball, 1970;

Kodagali et al. 1971; Ladds et al. 1973). Lagerlof (1938) observed testicular hypoplasia in 23 per cent of total testicular affections in Swedish bulls. The incidence of testicular hypoplasia in bulls in other countries was reported to be only low and sporadic (Roberts, 1971).

Gunn et al. (1942) observed testicular hypoplasia/atrophy in about 3.4 per cent of 9,000 rams examined in Australia. The condition was noted in 0.7 per cent of 16,665 rams by Miller & Moule (1954). The incidence was reported to be 0.9 per cent by Smith (1962). Watt (1971c) estimated that 1.3 per cent of 2,281 Merino rams had testicular hypoplasia.

Wester (1915) reported testicular hypoplasia as an important cause of sterility in bucks. Richter (1919) observed that eight out of twenty sterile bucks studied by him had testicular hypoplasia. Of 501 males studied by Rosenberger (1951), 59 were sterile, 25 owing to hypoplasia of the testis. Schonherr (1956) stated that one third of sterility problems in young male goats was on account of testicular hypoplasia. Fraser (1971) described testicular hypoplasia as a principal factor to produce lowered fertility in goats.

There is general agreement among the workers that testicular hypoplasia is congenital and possibly hereditory

in origin. In Swedish Highland breed the condition has been reported to be due to a single recessive autosomal gene with incomplete penetrance (Eriksson, 1943) and with linkage to white coat colour (Settergren, 1963). A hereditory basis for the condition in cattle was also suggested by Laing & Young (1956) and Bishop (1972). The possibility of certain exogenous factors such as hormonal disturbances, vitamin deficiencies, toxic factors etc., operating intra-uterine or post natal life was suggested by Cohrs (1966). Bruere et al. (1969) observed testicular hypoplasia in rams in association with XXY sex chromosomes, a condition resembling Klinefelters syndrome in man. Richter (1919) attributed testicular hypoplasia in bucks largely to heredity.

The hypoplastic testis was smaller than normal. The consistency was normal or soft in mild and intermediate degrees of hypoplasia, but with extreme degrees it tended to be firmer than normal. The histological changes were found to vary depending on the extend of hypoplasia. Deficiency of few, most or all functional tubules were observed in mild, severe and extreme degrees of hypoplasia respectively (Jubb & Kennedy, 1970).

Hypoplasia of the epididymis accompanied testicular hypoplasia (Rollinson, 1950; Jubb & Kennedy, 1970; Fraser, 1971). Fraser (1971) described the morphological changes

in the hypoplastic epididymis of bucks as loss of detectable lobulation in the caput epididymis, which could be seen in normal ones. Histologically there was marked underdevelopment, narrow lumen and subnormal depth of epithelial sheets of the epididymal tubule (Rollinson, 1950).

Cryptorchidism or incomplete descend of the testis has been observed in all domestic animals, either unilaterally or bilaterally. It was seen most commonly in stallions, boars, dogs, less commonly in rams, bucks, and uncommonly in cattle (Roberts, 1971). The low incidence of cryptorchidism in bulls was reported by other workers also (Blom & Christensen, 1947, Carrol et al. 1963; Bishop, 1972; Ladds et al. 1973; Galloway, 1974).

The condition was observed in less than one per cent of rams (Gunn et al. 1942; Moule, 1950; Miller & Moule, 1954). Lush et al. (1930) recorded that 5.5 per cent of Angora goats were affected with cryptorchidism. It was further observed that the condition was usually unilateral affecting almost always the right testis. In an extensive survey covering about 8,000 bucks of Angora breed, Skinner et al. (1970) found an incidence of 1.98 per cent of the condition.

The etiology of cryptorchidism was attributed to a hereditary factor. In most of the animals except horses,

this was considered to be a recessive character (Roberts, 1971). Abnormal gubernacular development in site, direction or extend could also be attributed to several forms of cryptorchidism (Wensing, 1973). Defects of the inguinal ring, the spermatic cord, persistence of the processes vaginalis could also probably produce the condition (Farrington, 1967). The possibility of hereditary predisposition to cryptorchidism in goats was suggested by Lush (1930) and Couvreux (1943). Warwick (1961) was able to eleminate this defect in a flock of Angora goats by adopting scientific breeding and rigid culling of parents and close relatives of the affected animals.

The cryptorchid testis was usually small insize, flabby and darker in colour than normal (Jubb & Kennedy, 1970). The histologic picture of cryptorchid testes revealed varying degrees of structural alterations. Generally the seminiferous tubules were present only in traces and the testicular tissue consisted solely of firm connective tissue with scanty or possibly numerous interstitial cells. In few cases the seminiferous tubules were almost normally developed but the spermatogonia showed no signs of differentiation (Cohrs, 1966).

The changes in the epididymis associated with cryptorchidism has not been well studied in animals. Roberts (1971)

stated that the loosely attached epididymis in the cryptorchid horses often descended into the inguinal canal.

Ectopia of the testis with location in the femoral canal, perineal region or close to the penis was occasionally found (Cohrs, 1966). Gross and histopathological findings resembled those in cryptorchidism (Humphrey & Ladds, 1975).

A perusal of available literature does not reveal any report on testicular aplasia in bulls and rams. But the condition has been recorded in bucks, stallions and dogs (Rosenberger, 1951; Runnels et al. 1965; Cohrs, 1966).

Multiple heterotopic testis formation in the form of numerous smaller or larger nodules with characteristic appearance of testicular tissue, scattered over the entire peritonium was observed in pigs (Cohrs, 1966). There appeared to be no such reports in other species.

The sensitivity of testicular epithelium to any adverse influence is well recognized and, in part at least, explains the observation that testicular degeneration is the most frequent cause of reduced fertility in male animals (Jubb & Kennedy, 1970). On the basis of an extensive study conducted on male infertility, Lagerlof (1963) reported that the testicular degeneration accounted to about 50-60 per cent of all testicular disorders in domestic animals.

The causes of testicular degeneration are reported to be quite numerous. Testicular degeneration most commonly occurred as a side effect of more generalised disease processes making it difficult to locate the primary cause in certain cases (Roberts, 1971). A weak endocrine constitution which was generally hereditary might predispose for degeneration (Lagerlof, 1963). Thermal degeneration due to high external heat (Lagerlof, 1938; Gunn et al. 1942; Smith, 1962; Rao, 1963; Waites & Ortavant, 1968); scrotal frost bite (Faulkner et al. 1967); testicular inflammations (Lagerlof, 1938; Gassner & Hill, 1955; Turnbull & Mckay, 1973); post vaccination pyrexia (Radhakrishnan et al. 1975) etc., were often reported in the literature. Nutritional disturbances including under-and-over feeding and deficiency of vitamins and minerals were at times cited as etiological factors to cause degeneration (Haq, 1949; Ghannam et al. 1966; Underwood, 1966; Moustgard, 1969). Old age might be another factor as degeneration was more frequently observed in aged animals (Gunn et al. 1942; Moule, 1950; Miller & Moule, 1954; Knudsen, 1954; McEntee; 1958; Galloway, 1961; Carrol et al. 1963; Bishop, 1970; Ladds et al. 1973). Other miscellaneous factors which were reported to cause testicular degeneration were, obstructive leisons of excretory ducts (Blom & Christensen, 1947; Ashdown, 1958); irradiation (Erickson, 1965; Sexena & Mathur, 1975); autoimmunization or isoimmunization

with seminal or testicular materials (Losos et al. 1968; Menge & Christian, 1971; Parsonson et al. 1971); experimental administration of chlorinated naphthalene, cadmium chloride and estrogen (McEntee & Olafson, 1953; Powel et al. 1964; Cupps and Briggs, 1965; Pate et al. 1970).

In degeneration the testicles become soft and flabby, sometimes firm in cases where the size of the testicle becomes considerably reduced. The cut surface does not bulge and lacks normal turgor (Jubb & Kennedy, 1970). The commonly reported histological changes in degeneration include, cytoplasmic vacuolation, germinal desquamation, pyknosis of the spermatic nuclei, giant cell formation, spermiostasis, calcification of the inspissated tubular contents, tubular atrophy, fibrosis, wave-like thickening of the basement membrane, fibrosis and interstitial cell hyperplasia (Lagerlof, 1938; 1963; Bishop, 1970; Jubb & Kennedy, 1970; Humphrey & Ladds, 1975). Konig (1964) recognized the basement membrane changes as the most helpful sign in differentiating testicular hypoplasia from degeneration.

Orchitis or inflammation of the testis, occurs in all species of animals. The reported incidence in bulls ranged from 0.2 to 13.3 per cent (Blom & Christensen, 1947; Van Der Sluis, 1953; Kanagawa, 1960; 1961; Galloway, 1961; Bellenger,

1971; Ladds et al. 1973). The incidence of orchitis in rams was reported to be 0.6 and 1.6 per cent by Moule (1950) and Miller & Moule (1954) respectively. The condition was recorded in bucks also (Richter, 1919; Loliger, 1956; Helmy, 1966).

According to Runnells et al. (1965) traumatic injuries and infectious agents were mainly responsible to produce orchitis in animals. Lagerlof (1938) reported that testicular infection especially that caused by Brucella species, formed the most common cause of testicular inflammation. Out of 6 cases of orchitis in goats studied by Helmy (1966), 4 were due to corynebacterium pyogenes, one each due to Brucella melitensis and staphylococci.

Two types of orchitis viz., interstitial and necrotic were recognised by Konig (1964). There might be difficulty to identify interstitial orchitis macroscopically. However, histologically the condition was characterized by mononuclear and giant cell infilteration of intertubular stroma with subsequent fibrosis. The structure of the tubular outline was obliterated and replaced centrally by neutrophils and detritus. Necrotic orchitis was characteristic of brucellosis but might result from other infections also. Total necrosis of the testis with thickening and adherence of the tunics was the common outcome of brucella infection. Microscopically

there was total coagulation necrosis bordered by fibrosis and mononuclear cell infilteration (Konig, 1964; Cohrs, 1966).

Periorchitis or inflammation of the tunica vaginalis was observed to an extent of 0.5 - 2.1 per cent in bulls (Galloway, 1961; Ladds et al. 1973). The nature and extend of the condition in rams and bucks was not well studied. Brucellosis was reported to be the most frequent cause to produce the condition. Tuberculous periorchitis was also observed at times (Cohrs, 1966). Infection due to Brucella organism was found to produce a serous, serofibrinous, fibrinous, haemorrhagic or purulent inflammation leading to adhesion of the tunics. In tuberculous periorchitis the tunica vaginalis would be studded with pearl like structures, resembling the tubercle nodule (Cohrs, 1966).

Adhesion between parietal and visceral layers of the tunica vaginalis was reported to be very common in bulls (Donham & Sims, 1931; Kanagawa, 1960; 1961; Galloway, 1961; Ladds et al. 1973). Increased occurrence of the condition in bulls with advancing age was observed by Galloway (1961) and Ladds et al. (1973). Watt (1971c) recorded the condition in 20.6 per cent of 2281 Merino rams examined. The adhesion was believed to be due to infectious or traumatic inflammations (Gunn et al. 1942; Miller & Moule, 1954; Galloway, 1961).

Testicular atrophy has been occasionally recorded in bulls (Webster, 1932; Lagerlof, 1938; Hooker, 1944; Blom & Christensen, 1947; Barker, 1956; Lancaster, 1956; Willet & Ohms, 1957; Knudson, 1958; McEntee, 1958; Galloway, 1961), in rams (Gunn et al. 1942; Miller & Moule, 1954; Watt, 1971c) and bucks (Richter, 1919; Loliger, 1966). The condition occurred as a result of testicular degeneration (Runnells et al. 1965).

Atrophied testes were firmer than normal with decreased size, weight and turgidity. (Watt, 1972). Microscopically, the tubular epithelium was single layered or entirely absent. There was thickening of the membrana propria with increased intertubular connective tissue. The interstitial cells of Leydig might be normal or increased in number (Runnells et al. 1965).

Testicular calcification usually occurred as a result of degeneration and fibrosis. This could also occur insidiously (Humphrey & Ladds, 1975). The reported incidence of calcification in bulls ranged from 1.7 to 31 per cent (Webster, 1932; Barker, 1956; Galloway, 1961; Ladds et al. 1973). Maurya et al. 1968 reported 9.5 per cent of calcification in buffaloe bulls. The incidence of calcification was reported to be only 3 per cent in rams (Fraser & Wilson, 1966). Richter (1919) described

the frequent occurrence of calcareous degeneration of the seminiferous tubules of sterile bucks. In goats, Fraser & Wilson (1966) reported an incidence of 40% calcification which appeared to be very high when compared to other ruminants. Fraser & Wilson (1966) considered that calcification was established by way of saponification with granule formation in the sperm masses. Venkataswami & Pattabiraman (1970) described four stages in the development of calcinosis viz., desquamation, coalescence of desquamate, hyalinization and subsequent calcification of the hyalinized mass.

Testicular haemorrhage occurring in association with certain infectious diseases, due to disturbances in clotting mechanisms, or in mechanical injuries of the testicle has been occasionally recorded in animals (Runnells et al. 1965).

There seems to be no report on melanosis of the testis proper in domestic animals. But the condition was reported to be common in wild birds, chickens and Turkeys. The pigments were seen in the interstitial connective tissue. Spermatogenesis in the affected testis was reported to be badly affected (Runnells et al. 1965; Cohrs, 1966; Valsala, 1973).

Testicular neoplasms were unusual in most domestic

species except the dog and possibly old bulls (Roberts, 1971). In bulls, interstitial cell tumour of a benign type has been reported frequently (Innes, 1942; McEntee, 1958; 1959; Cupps et al. 1964; Dabholkar et al. 1967). Sertoli cell tumour and seminoma were only rarely recorded in bulls (McEntee, 1959; Cotchin, 1960; Dabholkar et al. 1967; Bhagwat et al. 1972; Ladds et al. 1973; Nigam, 1975). In rams, isolated cases of sertoli cell tumours (Shortridge, 1962) and seminomas (Jensen & Flint, 1963; Shortridge & Cordes, 1969; Watt, 1971b) have been recorded. No reference could be traced on the occurrence of testicular neoplasms in bucks.

Innes (1942) stated that benign interstitial cell tumour seen in old animals might be related to hormonal imbalance. Dunn & McEntee (1964) observed distinct breed differences in the frequency of occurrence of testicular neoplasms in cattle. The experimental studies on mice conducted by Steves (1964) showed that, testicular tumours were largely genitically mediated. Moreover the action of genes appeared to be confined to testis, the genotype of the somatic tissues playing only a minor role. The gross and microscopic features of different types of testicular neoplasms were well described by Cohrs (1966) and Jubb & Kennedy (1970).

Hermaphroditism or intersexuality occurs most commonly in goats and pigs (Roberts, 1971). True hermaphroditism was only rarely reported but compared to other species its incidence was high in goats (Asdell, 1946; Divekar, 1953; Jamdar, 1955; Kaikini & Puranik, 1964; Raja, 1965). Male pseudohermaphroditism has been very often seen in hornless breeds of goats; its incidence being about 5-15 per cent (Eaton & Simmons, 1939; Eaton, 1943; Paget, 1943; Asdell, 1944; Buechi, 1957; Brandsch, 1959; Laor et al. 1962; Koch, 1963; Soller, 1963). The reported incidence of the condition in horned breeds of goats was less than 0.1 per cent. (Asdell, 1942; Paget, 1943; Divekar, 1953; Kaikini & Puranik, 1964).

Studies by Eaton (1939), Asdell (1942) and Buechi (1957) have led them to suppose that hemaphroditism in goat might be due to a recessive factor linked with the gene for hornlessness. Kondo (1955) also held the view that higher incidence of intersexuality in Japanese goats was due to selection of polled animals for breeding.

Detailed study of the anatomy of the genitalia in pseudohermaphrodite goats was made by Crew (1924), Eaton (1943) and Widmaier (1958). Affected animals showed a wide range of abnormalities from phenotypically normal

males through many intermediate gradations to almost normal females. Despite the variability in the morphology of external genitalia and the accessory organs, the intersexes possessed only testicular tissue (Biggers & McFeeley, 1966). The majority of male pseudohermaphrodites were also cryptorchids (Koch, 1963).

Asdell (1944) was the first to make the suggestion that in goats the genetic sex of Male pseudohermaphrodite was female. His argument was based on the fact that an approximately normal sex ratio was obtained in goats, with high incidence of intersexuality, if the intersexes were counted as females. Some preliminary work on this line, using direct cytogenetic techniques was in support of this view (Luers & Struck, 1959; Bielanaka & Osushowaka, 1960; McFeeley, et al. 1966).

Among the various pathological conditions affecting the epididymis, spermatocele and spermatic granuloma formed an important entity especially in bucks (McEntee, 1973). Blom & Christensen (1960) reported an incidence of 3 per cent spermatoceles in Danish bulls. Spermatoceles in the testicular tissue and epididymes have been observed in rams (Watts, 1972). The condition was reported to be very frequent in bucks. Richter (1919) observed the condition

in many young sterile male goats. Ott (1937) reported that 20-25 per cent of all male goats born in breeding herds in Germany later became sterile due to the development of sperm granuloma. Of 501 males studied by Rosenberger (1951), 59 were sterile, 27 as a result of abnormal stasis of semen, Schonherr (1956) stated that two third of a large number of sterile young male goats had sperm granulomas. Koch (1963) stated that, sperm retention as a cause of sterility occurred in 10-30 per cent goats of hornless breeds, whereas in horned goats it was very uncommon.

Rosenberger (1951) believed that an unduly high protein content in the ration of young animals, insufficient grazing and deficiency of Vitamin A and B predisposed. Loliger (1957) suggested that spermatic granuloma in goats was caused by local infection with endemic bacteria. But many regarded it as an inherited congenital malformation (Richter, 1919; Blokhuis, 1962). Spermiostasis in one or more aberrant ductules, mesonephric tubules closed at one end but retaining communication with rete testis or epididymal duct, causing subsequent sperm granuloma was still a very attractive theory as to the etiology of the condition in bucks (McEntee, 1973). Evidence has been accummulated in recent years to indicate that sperm granuloma

might be due to a variety of factors which damage the wall of efferent ducts thereby releasing the sperm from the duct (Eriksson, 1970; P[illegible]son et al. 1971; McEntee, 1973).

The striking macroscopic feature of spermatic granuloma was the enlargement of the head of the epididymis, less frequently the body and the tail of the epididymis (Cohrs, 1966). Spermatoceles and spermatic granulomas were macroscopieally similar (Watt, 1972). The histologic features of spermiostasis was the distention of lumen of efferent tubules with darkly staining masses of spermatozoa. Large lymphocytic foci in the adjoining connective tissue might also be noticed (McEntee, 1973). The histology of the sperm granuloma closely resembled that of a tuberculous granuloma (Jubb & Kennedy, 1970).

In spermatoceles or spermatic granuloma, the testis was generally not affected but occasionally it might become small and fibrotic with the presence of smaller or larger areas of caseation and calcification (Cohrs, 1966).

The segmental aplasia of the Wolffian duct was first described by Blom & Christensen (1947) in bulls. The condition was subsequently reported by several others (Ashdown, 1958; McEntee, 1958; Galloway, 1961; Kanagawa et al. 1961). Blom (1972) summarised that the overall

incidence of the condition came up to 0.56 per cent in bulls. Segmental aplasia of the Wolffian duct was also reported in rams (Bruere, 1970). But there seems to be no report of this condition in bucks.

Segmental aplasia of Wolffian duct was reported to be a hereditary condition (Blom, 1972). Konig et al. (1972) considered an autosomal recessive gene to be involved. Unilateral and bilateral involvements were described, with spermiostasis and increased pressure resulting in spermatocele formation and degenerative tubular changes, the severity of which was proportional to proximity of the apalstic segment to the testis (Kenney, 1970).

The epididymal melanosis was described by various workers in bulls (Blom & Christensen, 1956; Galloway, 1961; Ladds et al. 1973). McEntee (1973) stated that the epididymis of black faced ram contained melanin giving it a black colour. There seemed to be no report of epididymal melanosis in bucks. The pigmentation might be single or double sided and might comprise the whole epididymis. Most often, however, only part of the organ was pigmented. The intensity of pigmentation varied from Indian ink black to lead grey. The pigment occurred in melanophores in the connective tissue as well as in the epithelial cell (Blom & Christensen, 1960).

Two congenital cystic conditions of bovine epididymis, viz., paradidymis and cystic persistent mesonephric tubules, have been observed by Blom & Christensen (1956). Cystic persistent mesonephric tubules was reported in rams (Quinlivan, 1970; Watt, 1971a) and Boars also (Thomas & Raja, 1974). But perusal of available literature does not reveal any report on the occurrence of the condition in bucks.

Epididymitis was occasionally observed as an acquired lesion in all species of animals (Roberts, 1971). The incidence of the condition in bulls ranged from 0.1 to 2.4 per cent (Blom & Christensen, 1947; Van Der Sluis, 1953; Galloway, 1961; Carrol et al. 1963; Ladds et al. 1973). The condition was extensively studied in rams (Gunn et al. 1942; Moule, 1950; Simmons & Hall, 1953; McGowan & Shultz, 1956; Buddle, 1956; Kennedy et al. 1956; Biberstein & McGowan, 1958; McGowan & Devine, 1960; Biberstein et al. 1962; Biberstein et al. 1964; Biberstein et al. 1966; Swift & Maki, 1968; Murray, 1969; Swift & Weyerts, 1970; Quinlivan & Lindsay, 1971). The incidence of epididymitis in rams ranged from 3.9 to 18.6 per cent. The epididymitis was occasionally reported in bucks also (Williams, 1946; Loliger, 1957; Cohrs, 1966).

Epididymitis arises chiefly by spread of infection in the genitourinary passages, less frequently by haematogenous metastasis, seldom by trauma (Konig, 1964). Infection of Brucella species was the most important cause of epididymitis in all species of animals (Roberts, 1971). Other organisms like corynebacterium, Pasteurella, streptococcus, staphylococcus, Actinobacillus species were also found to cause the condition (Gunn et al. 1942; Simmons & Hall, 1953; Edgar, 1959; Shortridge, 1962; Galloway, 1966; Helmy, 1966; Watt et al. 1970).

The gross lesion of epididymitis was enlargement of tail of epididymis accompanied by varying degrees of adhesion of the layers of tunica vaginalis. Fibrosis and spermatic granuloma were also present. The characteristic histological findings were papillary hyperplasia and focal hydropic degeneration with the formation of intra epithelial cysts in the lining of epididymal ducts. Leucocytic infiltration at the site of inflammation might also be present. (Kennedy et al. 1956; Biberstein et al. 1964; Jubb & Kennedy, 1970; Swift & Weyerts, 1970).

Primary tumors of the epididymis were extremely rare in domestic animals (McEntee, 1973). Testicular tumours might invade the epididymis and spread through the epididymis

to the vas deferens and cord. Occasionally metastatic tumours might develop in the epididymis (Roberts, 1971).

In bulls several workers have reported the occurrence of spermatozoan tail abnormalities as a result of pathological composition of epididymal plasma consequent to primary functional disturbances of the epididymal epithelium (Swanson & Boyd, 1962; Hopwood et al. 1963; Cupps & Briggs, 1965; Gustafsson, 1965; 1966; Gustafsson et al. 1972; Raja et al. 1974; Rao, 1976). But the condition has not been reported in other species.

MATERIALS AND METHODS

MATERIALS AND METHODS

The materials for the study comprised of 1000 pairs of testicles with the appendanges, of goats, collected at random from the Municipal slaughter house, Trichur.

The animals from which the organs were collected were approximately in the age group of 6 to 18 months. The materials from grossly undersized and weak bucks were not used for the present study.

The testicle with epididymis was removed from the scrotal sac and transported to the laboratory in separate polythene bags within an hour of slaughter. In case of cryptorchid animals, the retained testis were removed after opening the body.

Gross Examination.

The testis and epididymis were exposed by incising the parietal layer of tunica vaginalis. Presence of any adhesion between tunics, epididymis and testis was noted. After trimming the epididymis from the testis proper both the organs were separately subjected to detailed examination.

The testis was examined for any deviation in size, shape and consistency. It was then cut into two halves by a midsagittal incision along the long axis. The degree of

bulging, the colour and gross lesions of the cut surface were noted. The testis was then cut into several transverse sections of 1 cm. thickness and each piece was carefully examined for any lesions.

Vas deferens was separated from epididymis at the junction with the tail of epididymis. The epididymis was then carefully examined for any abnormality in size, shape and consistency.

Histopathological examination.

Representative samples of tissues, 0.5-1 cm. thickness from the dorsal, middle and ventral regions of the grossly affected testes and from those suspected to be affected were removed and transferred to Bouin's fluid. Similarly pieces of tissues from caput, corpus and cauda of epididymis were also transferred to the fixative fluid.

Bouin's fluid as modified by McEntee (1973) was prepared by mixing 750 cc. saturated aqueous picric acid, 250 cc. commercial formalin and 25 cc. of glacial acetic acid and used as fixative.

The tissue pieces were fixed in Bouin's fluid for 36 hours, dehydrated, cleaned and embedded in paraffin.

Sections of 5-7 micron thickness were cut and stained by Haematoxylin-Eosin (regressive method) and Van Gieson's stain (Humason, 1971).

Special staining procedures were adopted for identification of calcium deposits (Von Kossa's Method), Hemosiderin (Pearls stain) and Melanin (Lillie Nile Blue Method, Ferric Ferri cyanide Method) as described by Humason (1971).

R E S U L T S

R E S U L T S

The various pathological conditions of testes and epididymis of bucks, observed during the course of the present study are listed in Tables 1 and 2.

Hypoplasia

Incidence.

There were 58 cases (5.8%) of hypoplasia, of which 56 (96.6%) were bilateral and 2 (3.4%) unilateral. In both the unilateral cases the right testis was affected.

Macroscopic Pathology.

In bilateral hypoplasia (Fig. 1) the size and weight of the testicle ranged from 2.2 x 1 x 1.4 cm. to 3 x 2 x 2.2 cm. and 4 to 7.5 g respectively. The consistency appeared to be slightly softer than that of the normal testis. The degree of bulging of the cut surface was poor. The colour of the parenchyma varied from dark brown to reddish brown.

The size of the hypoplastic and normal testicles in one case of unilateral hypoplasia was 3.5 x 2.3 x 2.5 cm. and 5.3 x 3.2 x 3.82 cm. respectively. The corresponding weights of the organs were noted to be 14 g and 48 g. In

the other case, the affected testicle had the size and weight of 3.8 x 2.6 x 2.8 cm. and 20g. The corresponding measurements of the normal testicles were 5.5 x 3.6 x 4.4 cm. and 52 g.

The weight of the epididymis in bilateral hypoplasia ranged from 0.9 to 1.8 g. The weight of the epididymis in unilateral hypoplasia ranged from 4 g and 8.1 g.

Microscopic Pathology.

Under-development of the seminiferous tubules of varying degrees and magnitude was the characteristic lesion observed in bilateral hypoplasia. In most cases the seminiferous tubules were lined by a single layer of supporting cells. Towards the centre of the luman, normal or degenerating gonocytes could be seen (Fig. 2 and 3). Certain degree of spermatogenic activity as revealed by the presence of spermatocytes was observed in some cases. But many of the spermatocytes showed pyknotic nuclei (Fig.4). Mild fibroplasia of the interstitial connective tissue and proliferation of Leydig cells were noticed.

In unilateral hypoplasia varying degrees of spermatogenic activities were evident. In some tubules even spermatozoa could be seen. In the majority of the tubules,

however, differentiation proceeded only upto the formation of spermatocytes (Fig. 5). Completely hypoplastic tubules with a single layer of supporting cells, which were very common in bilateral hypoplasia were only rarely seen. Some tubules revealed slight desquamation of epithelial cells. Intertubular giant cell formation was absent. Histologic picture of the normal testicles showed progressive spermatogenic activity.

In bilateral hypoplasia the section of the epididymis showed small sized tubules with narrow and empty lumen. Structural integrity was always maintained. But there were varying degrees of intertubular fibrosis, this being pronounced in certain cases. The structure of the epididymis in unilateral hypoplasia appeared to be normal except for the absence of sperms in many of the tubules. Some tubules in the caput contained large number of seminiferous epithelial cells intermingled with giant cells having 4-12 nuclei (Fig. 6). In some tubules spermatozoa were also present (Fig. 7). The cauda epididymis contained mostly eosinophilic granular materials with occasional spermatozoa and degenerated seminiferous epithelial cells. The section of the normal epididymis in cases of unilateral hypoplasia revealed normal picture.

Cryptorchidism

Incidence.

Out of 1000 pairs of genitalia examined 29 (2.9%) showed cryptorchidism. The unilateral and bilateral occurrence of the condition was noted in 23 (79.3%) and 6 (20.7%) cases respectively. In unilateral condition the right testis was always involved. The testis was retained inside the abdominal cavity in all except one in which the organ was located in the inguinal canal. In 12 cases, the epididymis had its attachment with the testes at the region of the caput only. In seven of these cases, the detached part of the epididymis alone was descended down to a distance of about 3-8 cm. from the external inguinal ring.

Macroscopic Pathology.

The size and weight of the retained testes ranged from 1.7 x 1.3 x 1.1 cm. to 4.4 x 2.8 x 3.5 cm. and from 1.5 to 25.2 g respectively. The testes had a flabby consistency. Bulging of the cut surface was poor. The colour of the cut surface ranged from chocolate colour through various shades of red and brown to almost normal fleshy white. The size (4.5 x 3 x 3.2 cm. to 5.6 x 4.1 x 4.2 cm.) weight (30.3 g to 62 g) and consistency of the descended testes were within

the normal range (Fig. 8).

The weight of the epididymis ranged from 0.95 g to 3.8 g. In cases where epididymis alone was descended, the detached part of the epididymis was stretched to a length of 5-12 cm. (Fig. 9).

Microscopic Pathology.

Microscopic picture of the testicles showed varying degrees of degenerative changes. The basement membrane was thickened and wavy. In most cases, the seminiferous tubules were lined by a single layer of supporting cells only (Fig. 10). Normal or degenerating gonocytes were occasionally met with at the basement membrane and the centre of tubule. Many tubules were distorted and few even destroyed and partially replaced by fibrous tissue (Fig. 11). Varying degrees of intertubular fibrosis and Leydig cell proliferation were also observed (Fig. 12).

Except for an increase in intertubular connective tissue no structural alteration was present in the epididymis. The intertubular fibrosis was very marked in certain cases (Fig. 13 and 14). The lumen of the epididymis was empty in all the cases.

Ectopic Testis

Incidence.

There were 4 cases (0.4%) of unilateral ectopic testis of the right side. Of these one was seen in association with Male pseudohermaphroditism.

Macroscopic Pathology.

In all cases the testis were located below the skin, slightly posterior to the external inguinal ring, close to the penis. In one case the testis was rotated on its long axis (Fig. 15). The size and weight of the affected testes ranged from 2.7 x 1.7 x 2.2 cm. to 3.3 x 2.1 x 2.4 cm. and 4.6 to 11 g respectively. The consistency was noted to be softer than that of a normal testis. The bulging of the cut surface was not pronounced. The weight of the epididymis ranged from 1.1 to 4 g.

Microscopic Pathology.

The seminiferous tubules were lined by a single layer of supporting cells, with few gonocytes scattered towards the centre. Degenerative changes characterised by vacuolation and hyperchromatosis of the lining cells were also noticed. An apparent increase in the interstitial cells was present (Fig. 16).

The epididymis of the ectopically placed testes revealed increased intertubular connective tissue. The one which was associated with Male pseudohermaphroditism in addition, showed vacuolation, desquamation and haemorrhage of the epididymal tubules at the region of the caput. The normally descended testis and epididymis did not reveal any structural alteration.

Degeneration

Incidence.

Extensive degenerative changes of the seminiferous tubules were observed in 4 cases (0.4%).

Macroscopic Pathology.

The size and weight of the testicles were within the normal range. The consistency was very soft with practically no bulging of the cut surface. The cut surface appeared somewhat granular and more reddish in colour.

Microscopic Pathology.

The microscopic changes showed wide variation not only between the organ but also between the tubules. Extreme degeneration characterized by very prominent

desquamation (Fig. 17) and vacuolation (Fig. 18) of the seminiferous epithelium were observed in all the four testicles. In many places the tubules were lined by a single layer of epithelium (Fig. 19). The basement membrane were thickened and hyalinized. Marked increase of intertubular connective tissue and apparent increase in the number of Leydig cells were also observed.

The epididymis revealed varying degrees of pyknosis, vacuolation and desquamation of lining cells together with intertubular fibrosis (Fig. 20).

Adhesion

Incidence.

There were 18 cases (1.8%) of adhesions. Of these, 13 (87%) were found to be bilateral and diffuse and the remaining (13%) unilateral and localised. Three cases of unilateral adhesions were seen in the right testes and two in the left. In bilateral cases, the adhesion with epididymis was also present in all except one.

Macroscopic Pathology.

In diffuse adhesion, the testicles were noted to be hard and atrophied. The tunics were firmly adhered to the

testis and epididymis. In localised adhesion the testicles appeared to be normal in size and consistency. The area of adhesion ranged from 0.5-1.5 sq. cm.

Microscopic Pathology.

Pronounced fibrous tissue proliferation at the site of adhesion was the characteristic change presently observed (Fig. 21). It was difficult to differentiate the tunica albugenia from the layer of tunica vaginalis. In 2 cases of diffuse adhesion and in one case of localised adhesion, infilteration of mononuclear cells especially lymphocytes was noticed (Fig. 22). The testicular tissue below the region of adhesion revealed varying degrees of degenerative changes. In diffuse adhesion the entire testis showed atrophic changes.

In one case of localised adhesion, metaplasia of the flat, mesothelial cells of the visceral layer of tunica vaginalis was observed. The cells appeared cuboidal or round in shape with large round nucleus. At some places these cells got organised to form small groups of tubules and attached to the external surface of the tunica albugenia (Fig. 23). These tubules resembled normal seminiferous tubules except for the absence of spermatid and spermatozoa. In some other areas unorganised clumps of seminiferous epithelial cells were observed near the visceral layer of tunica vaginalis.

Atrophy

Incidence.

The incidence of testicular atrophy was observed to be 1.3% (13 cases). All the cases were bilateral in occurrence.

Macroscopic Pathology.

The size (Fig. 24) and weight of the testicle ranged from 2.2 x 1 x 1.4 cm. to 3.7 x 2.1 x 2.5 cm. and 4.2 to 14 g respectively. There was diffuse adhesion with tunics. The testicles appeared to be hard in consistency. The cut surface was brownish yellow in colour. 3 cases revealed grittinous due to calcification. The calcified area appeared yellowish white. The epididymis was harder in consistency and weighed from 1.2 to 3 g.

Microscopic Pathology.

Eleven cases of testicular atrophy were characterized by complete necrosis and hyalinization of the parenchyma with pronounced fibrous tissue replacement in the periphery of the testes (Fig. 25). The interstitial cells were almost always hyalinized. Two cases however showed proliferative changes. Prominent mononuclear cell infilteration especially with lymphocytes, in the tunica albugenia and periphery of

the testicles was observed in two other cases (Fig. 26). The blood vessels in the tunica albugenia appeared to be sclerotic. Three cases revealed calcification of the seminiferous tubules. Calcification occurred mostly on the desquamated contents of the seminiferous tubules (Fig. 27), but in some tubules calcification of the epithelium occurred in situ also (Fig. 28).

The microscopic picture of the testicles in two cases showed diffuse fibrosis of the entire testis. The seminiferous tubules were found to have thick, hyalinized basement membranes lined internally by a single layer of degenerated cells. Marked proliferation of the interstitial cells was noticed in one case (Fig. 29).

Microscopic picture of the epididymis revealed varying degrees of pyknosis, vacuolation and desquamation of the lining epithelium and prominent intertubular fibrosis (Fig. 30).

Testicular Haemorrhage

Incidence.

Haemorrhage was observed in the right testis in one case (0.1%).

Macroscopic Pathology.

The affected testis appeared to be normal in size, shape and consistency. On the medial aspect of the testis a dark area of about 4 sq. cm. was observed.

Microscopic Pathology.

The section of the testicle revealed a thick band of tissue - Orange with H & E stain and Yellow with Van Gieson's stain - underneath the tunica albugenia (Fig. 31). It was composed of closely packed, degenerated and hyalinized red blood cells. At some regions the blood was seen penetrated into the testicular parenchyma (Fig. 32). In these, RBCs were present in the intertubular and intratubular spaces. The seminiferous epithelium below the region of haemorrhage showed marked degenerative changes, characterized by desquamation, vacuolation and hyperchromatosis with practically no gamatogenic activities.

Male Pseudo Hermaphroditism

Incidence.

One case of Male pseudohermaphroditism (0.1%) was noticed in the present study.

Macroscopic Pathology.

The affected animal had external genitalia resembling that of a female with prominent teats and mammary glands. A greately enlarged clitoris with an imperforate vulvar configuration was present. The internal genitalia also resembled that of female (Fig. 33). But the gonads located on either side of the uterine horns appeared to be like cryptorchid testes. The right gonad was located ectopically under the skin slightly posterior to the external inguinal ring and the left, in the abdominal cavity. At the region of the right caput epididymis, there was a cyst of about 1 cm. diameter.

The uterus, cervix and vagina formed a single destended cavity containing about 300 ml of clear mucous fluid. The destended uterine on either side was seen originated from the respective cauda together with a structure resembling vasa defferentia. The vas deferens initially passing along the lateral border of the respective uterine horn, turned ventrally at about the middle and travelling further down, it became thickened and enlarged like ampulla at the region of the anterior vagina. Beyond this, the structure could not be palpated. The uterine horn and vasa defferentia on either side was observed to be bound together by connective tissue.

Microscopic Pathology.

Sections of the gonads revealed densely packed seminiferous tubules, lined by a single layer of supporting cells. Mild degeneration of the lining cells was present in both the testes. The interstitial cells showed proliferative changes. Seminiferous tubules and interstitial cells were present inside the wall of tunica albuginea at different locations (Fig. 34).

The epididymis revealed a marked increase in the intertubular connective tissue. The structural integrity of the lining cells was maintained in the left epididymis. But the right epididymis especially at the region of efferent ducts showed vacuolation and desquamation of the lining cells. At some regions haemorrhage was also noticed between or within the tubules.

The microscopic characteristics of the vas deferens, ampulla, uterus and vagina appeared normal (Figs. 35 & 36).

Spermiostasis

Incidence.

Spermiostasis in the efferent duct was observed in 11 cases (1.1%).

Macroscopic Pathology.

The epididymis was normal in all respects except for the stasis of sperms in one or two efferent ducts. Macroscopically the affected ducts appeared like thick yellowish-white coiled lines.

Microscopic Pathology.

The efferent tubules were distended with closely packed sperm masses. The lining of the tubule was intact (Fig. 37). In two cases large groups of lymphocytes were observed in the intertubular connective tissue near the distended tubules (Fig. 38). The sections of testes and epididymis were found to be normal.

Spermatic Granuloma

Incidence.

One case (0.1%) of spermatic granuloma was observed.

Macroscopic Pathology.

A large number of yellowish-white nodules of varying sizes (some upto 2 cm. in diameter) were noted at the region of efferent ducts on both sides (Fig. 39). The weight of the right and left epididymis were 16.5 g and 16 g respectively. Both the testicles were also found to be larger in

size and softer in consistency. The right testis had a size and weight of 7.1 x 4 x 4.7 cm. and 93.2 g respectively, while the left testicle measured 6.5 x 3.6 x 4.1 cm. and weighed 72 g.

The nodules when incised revealed thick yellowish-white caseated material resembling inspissated pus. Clear watery fluid oozed out from the cut surface of the testicle.

Microscopic Pathology.

The tubular lining of the affected ducts was completely destroyed resulting in the extravasation of sperms into the interstitium of the epididymis. Around this accummulated mass of sperms, a typical foreign body granulomatous reaction characterized by the presence of a large number of macrophages and few giant cells was seen. The zone of granulomatous reaction was walled off by a thick band of fibro-collagenous tissue. The lumen of the destroyed tubules showed the presence of thickly packed sperm masses together with cellular debris, macrophages and giant cells. Most of the giant cells revealed ingested sperms in their cytoplasm (Fig. 40). Isolated groups of lymphoid cells amidst the fibrous tissue were also observed at many places. Some of the efferent tubules were densely packed with sperms, but the lining epithelium was intact.

The seminiferous tubules were separated from the surrounding connective tissue, resulting in the formation of a clear vacant space all around the tubules. Most of the tubules were shrunken and completely packed with seminiferous epithelial cells intermingled with spermatozoa. Pronounced desquamation of the seminiferous epithelium was also present.

Epididymal Haemorrhage

Incidence.

There were 4 cases (0.4%) of unilateral epididymal haemorrhage. Two cases were observed in the right epididymis and 2 in the left epididymis.

Macroscopic Pathology.

The haemorrhage characterized by brownish black discolouration was confined at the region of efferent ducts. The affected epididymis appeared to be normal.

Microscopic Pathology.

Haemorrhage was observed mostly in the epithelial lining of the efferent ducts (Fig. 41). Red blood cells could be located in the lumen in certain areas. Haemorrhagic

tubules were very much dilated. The integrity of the epithelial lining was lost at some places. There was no pathological lesions in the testicles or epididymis.

Epididymal Melanosis

Incidence.

43 cases (4.3%) of epididymal melanosis was observed, of which 37 (86%) were unilateral and the remaining bilateral (34%). Among the unilateral cases, 22 (58.5%) were seen in the right epididymis. Localised and diffuse type of melanosis was observed in 21 (46.5%) and 22 (53.5%) cases respectively.

Macroscopic Pathology.

Localised melanosis characterized by a brownish black discolouration was confined mostly at the region of efferent ducts. The diffused type showed varying degrees of black pigmentation at different regions of epididymis. Sometimes the whole epididymis was involved.

Microscopic Pathology.

In localised melanosis the pigments appeared as dark brown round masses within the epithelial cells of the efferent ducts (Fig. 42). No structural alteration was

generally observed. But in some cases, the epithelium of the efferent ducts was destroyed at certain places. In diffuse type, the brownish black elongated or spindle shaped pigment masses were seen scattered in the smooth muscles and connective tissue layers of epididymis (Fig.43). Structural integrity of the epididymis was maintained in all cases.

Cystic Persistent Mesonephric Tubule

Incidence.

89 cases (8.9%) of cystic persistent mesonephric tubules were observed, of which 41 (46.1%) were seen on the right side, 35 (39.3%) on the left and 13 (14.6%) on both sides.

Macroscopic appearance.

Majority of the cysts appeared as round or oval structures measuring about 0.2-0.5 mm. in diameter, located at the anterior aspect of caput epididymis near its ventral border (Fig. 44). Very rarely cysts upto 1 cm. in diameter were observed. Infrequently 2 or 3 cysts were observed in the same location. A few (4 cases) were observed within the tunica albugenia about 1-2 cm. below the usual location.

The cysts contained a clear watery fluid. The testis and epididymis did not reveal any macroscopic changes in any of the cases.

Microscopic appearance.

Histologically the cysts were found to be lined by low columnar ciliated epithelial cells (Fig. 45). In all cases except one, the testes and epididymis did not reveal any inflammatory or pathological changes. In one case in which the cyst was located within the tunica albuginea (Fig. 46), testicular parenchyma beneath the cyst, showed marked degeneration of the seminiferous tubules. In this area dilatation and rupture of lymphatic channel were also observed.

Appendix Epididymis

Incidence.

In 1000 pairs of organs examined, 147 (14.7%) showed appendix epididymis. Of these 89 (61.3%) were right sided, 32 (21.8%) left sided and 26 (16.9%) bilateral. 115 (78.2%) cases appeared haemorrhagic in type.

Macroscopic appearance.

The appendix epididymis was a small, flat, disc-shaped

structure attached to the anterior aspect of caput epididymis near its ventral border. Its diameter ranged from 0.2-0.6 mm. Two types were observed one reddish and the other, whitish in colour.

Microscopic appearance.

These bodies were found to be lined by a layer of tall columnar cells on the exterior. The lining cells blended with the visceral layer of tunica vaginalis. The body contained tubule similar to the epididymal tubule in structure, surrounded by connective tissue. The serial sections revealed that the tubules ended blindly, with no mechanical connection with the epididymal tubule (Fig.47). The appendices which appeared haemorrhagic grossly, were found to be closely packed with RBCs masking the tubular structure within (Fig. 48).

Cystic Remnants of Mullerian Duct

Incidence.

22 cases (2.2%) were observed. The frequency of occurrence of the condition in the right and left organ was found to be 9 and 8 cases respectively. In 5 cases, both the organs were involved.

Macroscopic appearance.

The size of the cyst varied from 0.3-1.2 cm. in diameter. The cysts were located either in the cauda near the origin of vas deferens (Fig. 49) or anywhere in the vas deferens (Fig. 50).

Microscopic appearance.

These cysts, which were embedded in the connective tissue consisted of a layer of smooth muscles lined interiorly with a layer of columnar cells. The section of the testis and epididymis did not reveal any pathological alterations.

DISCUSSION

DISCUSSION

Out of the total 1000 pairs of testicles and epididymis examined, 174 (17.4%) revealed various pathological lesions. The percentage of pathological conditions observed during the course of the present study was comparable to that reported in the literature (Ott, 1937; Rosenberger, 1951; Blokhuis, 1966; Loliger, 1957; Weber, 1969). In addition 258 organs (25.8%) showed minor developmental aberrations of little significance.

Of the total 174 pathological conditions, 147 were congenital or possibly hereditary in origin. The incidence of acquired conditions was only in 28. The low incidence of acquired conditions might be due to the comparatively low age group of the experimental animals. A positive correlation between age of the animal and the occurrence of acquired pathological conditions of the gonads has been observed by several workers (Gunn et al. 1942; Miller & Moule, 1954; McEntee, 1958; Murray, 1969; Bishop, 1970).

Testicular hypoplasia, seen in 58 genitalia (5.8%) formed the most commonly encountered pathological condition of the testicles in bucks. The frequency of occurrence of

the condition presently observed is comparable to that reported in goats by the earlier workers (Richter, 1919; Rosenberger, 1951; Schonherr, 1956; Fraser, 1971). In the present study hypoplasia was noted to be mostly (96.6%) bilateral in nature. In bulls and rams, on the other hand, unilateral hypoplasia was of more frequent occurrence (Lagerlof, 1938; Bruere, 1970). The bilateral cases revealed the gross and microscopic characteristics similar to those of total gonadal hypoplasia described in bulls by Lagerlof (1938). The affected testicles were strikingly small in size andweight and showed total absence of gamatogenic activity.

Unilateral hypoplasia was observed only in 2 cases (3.4%) and in both the right testes were involved. In contrast Lagerlof (1938) reported that the left testicles (25%) were more prone to be affected with hypoplasia than the right (1%) in bulls. The hypoplastic testicle was undersized and histologically resembled a developing testicle. Majority of the seminiferous tubules revealed varying degrees of spermatogenic activities. Section of the caput epididymis showed spermatozoa intermingled with seminiferous epithelial cells and giant cells - a picture often seen in association with a developing testis (Harshan, 1975; Settergren, 1975). Temporary unilateral

hypoplasia due to late descend of testis has been reported in rams (Gunn et al. 1942; Bruere, 1970). It is probable that the two unilateral conditions presently observed might not have been true hypoplasia but only cases of delayed onset of spermatogenic activity resulting from the late descend of testicles.

Cryptorchidism was noticed in 2.9% (29 cases) of the organs examined. The incidence of the conditions presently observed is in keeping with that reported in bucks (Lush et al. 1930 and Skinner et al. 1970). The unilateral and bilateral occurrence of the condition was noted to be 79.3% (23) and 20.7% (6) respectively. In unilateral cases, only the right testis was involved. In Angora goats Lush et al. (1930) also observed that cryptorchidism was usually unilateral affecting almost always the right testis. According to Brodey and Martin (1958), unilateral cryptorchidism more often affects the right testis, possibly because the right testis develops in the embryo a greater distance away from the scrotum.

Microscopic picture of the cryptorchid testis showed varying degrees of degenerative changes. But extensive degeneration characterized by fibrosis and interstitial cell proliferation (Cohrs, 1966; Jubb & Kennedy, 1970) was

not observed in any of the cases. Since the animals in the present study were in the low age group, the organs probably might have been collected much before the terminal stage of degeneration.

In 12 cases, attachment of the epididymis with the corresponding testis was confined only at the caput region. Further, there was partial descend of the detached part of the epididymis in seven cases. The condition similar to this has been reported in horses (Roberts, 1971). Except for an increased intertubular connective tissue no other structural alteration was observed in the epididymis. The lumen of the epididymis was empty.

In four cases, the right testis was found ectopically located below the skin, slightly posterior to the external inguinal ring, close to the penis. Ectopia of the testis with location in the femoral canal, perineal region or close to the penis has been occasionally reported in domestic animals (Cohrs, 1966). Gross and histopathological changes resembled those of cryptorchid testes. This finding is essentially in keeping with that reported by Cohrs (1966). According to Roberts (1971) the condition was seen more frequently in male pseudo hermaphrodites and attributed this to the absence of scrotum. Out of the four cases

presently observed, one was seen in association with male pseudo hermaphroditism.

Extensive degeneration of seminiferous tubules characterized by severe desquamation, vacuolation, pyknosis and fibrous tissue replacement was observed in four (0.4%) testicles. The incidence of testicular degeneration presently recorded is much lower than that reported in domestic animals (Lagerlof, 1963). Jubb & Kennedy (1970) opined that the sensitivity of testicular epithelium to any adverse influence might be the reason for observing high proportion of testicular degeneration in animals. In the present study, only organs which were grossly affected and those suspected to be affected were included. Since it is not possible to detect the first two degree of degeneration clinically (Lagerlof, 1963), some cases of mild degeneration might have passed unnoticed and left out from the study. Further, there are several reports to indicate the incidence of testicular degeneration increases with increasing age of the animals (Miller & Moule, 1954; Galloway, 1961; Ladds et al. 1975). The comparatively low age group of the animals might also have contributed for the present low incidence of the condition.

There were 18 cases (1.8%) of adhesions. Thirteen (72.2%) were bilateral and diffuse and seen in association with testicular atrophy. The five unilateral adhesion was localised and confined to an area of about 0.5-1.5 sq. cm. The incidence of adhesion presently observed was much lower than that reported in rams (Watt, 1972) and bulls (Webster, 1932; Kanagawa, 1960; 1961; Galloway, 1961). Increased occurrence of the condition with advancing age has been reported in bulls (Galloway, 1961; Ladds et al. 1973). The low incidence of adhesion presently encountered probably could be attributed to the comparatively low age group of the animals. The microscopic characteristics of the adhesion observed in the present study are comparable to those reported in the literature (Cohrs, 1966; Jubb & Kennedy, 1970). The testicular tissue below the region of adhesion revealed varying degrees of degenerative changes. In diffuse adhesions, the entire testis showed atrophic changes.

Metaplasia of the epithelium of the visceral layer of tunica vaginalis and organization of the metaplastic cells into structures resembling normal seminiferous tubules at different regions, observed in one case of localised adhesion seems to be a new finding. The metaplasia of the epithelial cells of tunica vaginalis into stratified

squamous epithelium has been reported in the literature (King, 1954). The epithelial cells lining the visceral layer of tunica vaginalis are known to be the remnants of the germinal epithelium that covers the primordium of the gonad in the embryo giving rise to the glandular tissue (Bloom & Fawcett, 1968). The formation of seminiferous tubule like structures from the metaplastic visceral layer as observed in the present case could thus be explained.

There were 13 cases (1.3%) of testicular atrophy. The incidence of gonadal atrophy presently observed is comparable to that reported in rams (Miller & Moule, 1954; Smith, 1962). In contrast, Richter (1919) and Fraser (1971) expressed the view that testicular atrophy was the most important and frequently encountered condition in bucks. However, it is to be pointed that under "atrophy" they included cases of testicular hypoplasia also. Hence, it is probable that some of the cases recorded by them might not have been "atrophic testicles" in the true sense, as pointed out by Rollinson (1950).

In eleven cases of testicular atrophy there were complete necrosis and hyalinization of parenchyma with pronounced fibrous tissue replacement at the periphery of

the testis. Interstitial cells were also hyalinized in most of the cases. Two of the cases showed in addition cellular infilteration. The lesions in these resembled those resulting from Brucella orchitis described by Cohrs (1966). Since the prevalence of Brucellosis in goats has been reported from different parts of India (Nelakhantan and Pande, 1948; Mathur, 1968; Panda & Pat, 1969; Mathur, 1972; Kumar et al. 1976), it is possible that the two cases under report might have developed as the after effect of Brucella infection. Three of the atrophied testicles (23%) revealed calcification also. The frequency of occurrence of calcification of caprine testis has been reported to be high (Richter, 1919; and Fraser & Wilson, 1966). Two cases revealed diffuse fibrosis with prominent interstitial cell proliferation.

Testicular haemorrhage was observed in one case (0.1%). Haemorrhagic area was dark in colour and was located in the medial aspect of the right testis. The affected organ was normal in size, shape and consistency. Microscopically a thick band of closely packed degenerated and hyalinized red blood cells was noticed just below the tunica albuginea. At some regions, the red blood cells were present inside the testicular parenchyma. The seminiferous epithelium below the region of haemorrhage showed marked degeneration.

Testicular haemorrhage occurring in association with certain infectious diseases, due to disturbances in clothing mechanisms or mechanical injuries of the testicle has been occasionally recorded in animals (Runnells et al. 1965).

One case (0.1%) of male pseudo hermaphroditism was observed. The condition has been reported to be very common in hornless breeds of goats (Eaton & Simmons, 1939; Eaton, 1943; Paget, 1943; Asdell, 1944; Beuchi, 1957; Laor et al. 1962; Koch, 1963). In horned breeds, on the other hand, the reported incidence of male pseudo hermaphroditism was only less than 0.1% (Asdell, 1942; Paget, 1943). Malabari goat is considered as a horned breed (Kaura, 1957) and this probably may explain for the low incidence of the condition presently observed. The morphological and histological characters of the gonads resembled those of cryptorchid testes. One peculiarity observed was the presence of testicular tissue within the tunica albuginea. Except for the absence of oviduct, the tubular genitalia of the female type was well developed. The absence of oviduct might be due to regression of the cranial portion of the mullerian duct. No attempt has been made to identify the genetic sex of the animal under study. However, there

are reports to indicate that in goats the genetic sex of male pseudo hermaphrodite is always female (Luers & Struck, 1969; Bielanska-osushowska, 1960; McFeeley et al. 1966).

There were 11 cases (1.1%) of spermiostasis, affecting 1 or 2 efferent ducts. The histologic picture was similar to that described by McEntee (1973), and was characterized by distention of efferent tubules with closely packed sperm masses. The structural integrity of the lining cells was not affected. In two cases there were large lymphocytic foci in the interstitial connective tissue near the distended tubule. The presence of lymphocytic foci in the interstitium of the epididymis has been observed even in normal animals (Rao, 1971 and Thomas, 1973) and according to McEntee (1973) this is of no significance. Spermiostasis in one or two aberrant tubules was not considered important unless it produced spermatic granuloma (McEntee, 1973).

There was only a single case (0.1%) of spermatic granuloma. The frequency of occurrence of the condition was very low when compared to that reported in polled breeds of goats (Richter, 1919; Ott, 1937; Rosenberger, 1951; Schonherr, 1956; Koch, 1963). However, in horned breeds, spermatic granuloma was observed only rarely (Koch, 1963; Weber, 1969). The low incidence of the condition presently

observed might probably due to the breed difference. The microscopic picture of the spermatic granuloma was characterized by distruction of the lining epithelium, extravasation of sperms into the interstitium and granulomatous reaction around the sperm masses. This is essentially in keeping with the findings of earlier workers (Cohrs, 1966 and Jubb & Kennedy, 1970). The testicles appeared large in size and soft in consistency. On incision, clear watery fluid oozed out from the testicles. The seminiferous tubules at certain regions were found to be separated from the surrounding connective tissue. A clear vacant space all around the tubules was also present. Most of the tubules revealed degenerative changes. The peculiar testicular changes could be attributed to the retention of fluid within the testicles, probably due to blockage of the efferent ducts.

Four cases (0.4%) of haemorrhage of the efferent ducts were observed. The condition was not seen reported in domestic animals. Haemorrhage was mostly confined within the epithelial lining of the efferent ducts. There was no pathological lesions in the testicles or epididymis associated with this condition. The efferent ducts are known to be very sensitive to inflammation and injury (Parsonson et al. 1971; McEntee, 1973). It may be possible

that epididymal haemorrhage presently encountered might have been due to some traumatic injury.

Epididymal melanosis was observed in 43 cases (4.3%). Blom & Christensen (1956) reported an incidence of 5.3% epididymal melanosis in bulls. Two morphologically and histologically different forms of melanosis viz., Localised and diffuse were presently recognised. The localised melanosis was noticed only at the region of efferent ducts and characterized by the presence of melanin pigments within the epithelial cells. On the other hand, the diffuse melanosis was observed in the epididymis at different regions. In this the pigmentation was confined almost always to the smooth muscles or connective tissue layers of the epididymal tubule. The present findings are akin to those reported in bulls by Blom & Christensen (1956). The importance of epididymal melanosis has been reported to be insignificant (McEntee, 1973). But in the present study few cases of localised melanosis showed destruction of epithelial layers of efferent ducts at certain areas. It is difficult to say with certainity whether these changes are of any significance. It may however be worthwhile to remember that melanosis could also sometimes be caused by adrenal diseases or hormonal imbalance (Runnells, 1965).

Eightynine cases (8.9%) of cystic persistent mesonephric tubules were observed in the present study. The condition does not seem to have been reported in goats. But in sheep and boar, this has been frequently observed (Watt, 1971a and Thomas, 1974). The cysts appeared as round or oval structures measuring about 0.2-0.5 mm. in diameter, located mostly in the anterior aspect of caput epididymis near its ventral border. In four cases, the cysts were located within the tunica albuginea, 1-3 cm. below the usual location. The macroscopic and microscopic characteristics of the cyst closely resembled those described by Watt (1971a). Section of the testicles and epididymis did not reveal any lesion in most of the cases. However, in one case where the cyst was present in tunica albuginea, the lymphatic channel below it was found to be very much distended resulting its rupture and effusion of fluid into the testicular parenchyma. This might have been due to the pressure exerted by the cyst in the lymph vessel. Moderate degeneration of seminiferous tubules was also present at the region.

During the course of the present study, 147 organs showed appendix epididymis. Of these, 89 were right sided, 32 left sided and the remaining 26 bilateral. The condition has been described in man (Bloom & Fawcett, 1970) but not

in domestic animals. The appendix epididymis appeared as a small, flat, disc-shaped structure; attached to the anterior aspect of the caput epididymis near its ventral border by a stalk. Most of them were haemorrhagic (reddish) and the others non-haemorrhagic (whitish). Microscopically this body was lined exteriorly by a layer of tall columnar epithelial cells. Within this, there was a tubule having a structure similar to the epididymal tubule. However, this tubule remained as a separate entity without having any connection with the epididymal tubule proper. The haemorrhagic type showed closely packed red blood cells masking the tubular structure within. The tubule which was present within the appendix epididymis was believed to be the remnant of a portion of mesonephric body (Bloom & Fawcett, 1970). No pathological lesion of testis or epididymis was noticed in association with the condition.

Cystic remnants of Mullerian duct were observed in 22 cases (2.2%). Of these, 9 were right sided, 8 left sided and the remaining bilateral. There seems to be no comparable reports in bucks or rams. The condition has been reported to be very high (24%) in bulls (Blom & Christensen, 1956). The cysts were located either in the cauda near the origin of vas deferens or anywhere in the vas deferens. The size of the cyst varied from 0.3-1.2 cm.

in diameter. Histologically the cysts were embedded in the connective tissue. The cyst wall consisted of smooth musculature, interiorly lined with a layer of columnar cells. The morphological and histological features resembled those reported in bulls (Blom & Christensen, 1956). No pathological changes of testis and epididymis was noticed in association with the condition. Hence it is considered as an anatomical variation of not much significance.

S U M M A R Y

SUMMARY

A study on the incidence and nature of testicular and epididymal pathology in bucks was undertaken as an aid to understand the problem of male infertility in goats. The study was based on one thousand pairs of testicles and epididymis of bucks, aged 6-18 months, collected from the Municipal Slaughter House, Trichur. After noting the macroscopic changes, the organs which were grossly affected and those which were suspected to be affected were subjected to detailed histological examinations.

Out of the 1000 pairs of organs examined, 174 (17.4%) revealed various pathological lesions. In addition, 258 organs (25.8%) showed minor developmental aberrations of little significance.

Of the total 174 conditions, 147 were congenital or possibly hereditary in nature. The various pathological conditions encountered under this catagory were: Hypoplasia, Cryptorchidism, Ectopic testis, Male pseudo hermaphroditism, Spermiostasis, Spermatic granuloma and Epididymal melanosis. The incidence of acquired conditions such as Testicular degeneration, Haemorrhage, Adhesions, Atrophy and Epididymal haemorrhage, was observed to be only 2.8% (28 cases). The developmental aberrations recorded were: Cystic persistent

mesonephric tubule, appendix epididymis and cystic remnants of the Mullerian duct.

Testicular hypoplasia was the most frequently encountered pathological condition. There were 58 cases (5.8%) of hypoplasia, of which 56 (96.6%) were bilateral and 2 (3.4%) unilateral affecting the right testis. All the bilateral cases revealed the gross and microscopic characteristics similar to those of total hypoplasia. Histologically the unilateral hypoplasia appeared more like a developing testicle. Hence the possibility of this being a condition of late descend of testicle was suggested.

Cryptorchidism was observed in 29 animals (2.9%) - 23 (79.3%) unilateral and 6 (20.7%) bilateral. In unilateral cases, always the right testis was involved. Detachment of corpus and cauda from the testis proper was observed in 12 cases, and in seven of these the detached part of the epididymis alone was partially descended. Microscopically the affected testicles revealed varying degrees of degeneration and interstitial cell proliferation.

Ectopia of the testis was observed in 4 cases (0.4%). In all these, right testis was involved. One case was

observed in association with Male pseudo hermaphroditism. The gross and microscopic picture of the gonads resembled the cryptorchid testes.

Extensive degeneration of seminiferous tubules characterized by severe desquamation, vacuolation, pyknosis and fibrous tissue replacement was observed in four (0.4%) testicles.

Adhesion of the tunics with epididymis and testis were observed in 18 cases (1.8%). Thirteen of these showed diffuse adhesion and were seen in association with testicular atrophy. In five cases the adhesion was localised to an area of 0.5-1.5 sq. cm. only. One case of localised adhesion revealed metaplasia of the epithelial cells lining the visceral layer of tunica vaginalis. These metaplastic cells at some places became organised to form structures resembling seminiferous tubules. This appears to be a new finding.

There were 13 cases (1.3%) testicular atrophy. In eleven cases there were complete necrosis and hyalinization of the parenchyma with pronounced fibrous tissue replacement at the periphery of the testis. Interstitial cells were also hyalinized in most of the cases. Two of these cases

showed in addition, cellular infilteration and the lesions in these resembled those resulting from Brucella orchitis. Three of the atrophied testicles revealed calcification also. Two cases revealed diffuse fibrosis with prominent interstitial cell proliferation.

Testicular haemorrhage was observed in one case (0.1%). The haemorrhagic area was dark in colour and located in the medial aspect of the right testis. The affected organ was normal in size, shape and consistency. Microscopically a thick band of closely packed, degenerated and hyalinized red blood cells was seen just below the tunica albuginea. At some regions red blood cells were present inside the testicular parenchyma. The seminiferous epithelium below the region of haemorrhage showed marked degenerative changes.

One case of male pseudo hermaphroditism was observed in the present study. The animal appeared like a female, except for the presence of a large penis like clitoris below an imperforate vulva. The tubular genitalia also was that of a female. Histologically both the gonads appeared like testes.

Spermiostasis in one or two efferent ducts of epididymis was observed in 11 cases. The testicles and epididymis did

not show any lesions. Lymphocytic foci near the obstructed tubules was observed in two cases.

There was one case (0.1%) of spermatic granuloma. The condition was characterized by the presence of a large number of granulomatous nodules at the region of the efferent ducts on both sides. Destruction of the lining epithelium of the duct, extravasation of sperms into the interstitium and granulomatous reaction around the sperm masses were observed microscopically at different places. The cauda epididymis was empty. The testes were enlarged and there was accummulation of fluid within the organs. The seminiferous tubules were found to be separated from the surrounding connective tissue resulting in the formation of a clear vacant space all around the tubules. Most of the tubules revealed pronounced desquamation.

Four cases of epididymal haemorrhage was noticed. The haemorrhage was mostly confined to the lining epithelium of the efferent ducts.

There were 43 cases (4.3%) of epididymal melanosis. The condition mostly occurred unilaterally (37 cases). In 6 cases bilateral involvement was seen. Two morphologically and histologically different forms of melanosis viz., localised and diffuse, were presently recognized. The

localised melanosis was noticed only at the region of the efferent ducts and characterized by the presence of melanin pigments within the epithelial cells. On the other hand, diffuse melanosis was observed in the epididymis at different regions. In this the pigmentation was confined almost always to smooth muscles or connective tissue layers of the epididymal tubule.

Eighty nine cases (8.9%) of cystic persistent mesonephric tubules were observed, of which 41 were on right side, 35 on the left side and 13 on both sides. Except in one case no pathological alteration in the testis or epididymis was observed. In one case dilation and rupture of the lymphatic channel together with moderate degeneration of testicular parenchyma was noticed below the region of the cyst.

147 cases (14.7%) of appendix epididymis were observed in the present study. Of these, 89 were right sided, 32 left sided and 26 bilateral. The appendix epididymis appeared as a small flat disc-shaped structure attached to the anterior aspect of the caput epididymis near its ventral border by a stalk. Two types viz., haemorrhagic (78.2%) and non-haemorrhagic (31.8%) were noted. This body was exteriorly lined by a layer of tall columnar cells. Within this there was a tubule having a structure similar

to the epididymal tubule. This tubule was found to have no connection with the epididymal tubule proper. The haemorrhagic type was closely packed with red blood cells masking the tubular structure within. Appendix epididymis has not been seen reported in domestic animals.

There were 22 cases (2.2%) of cystic remnants of Mullerian ducts located either in the cauda near the origin of vas deferens or anywhere in the vas deferens. No pathological lesion of the testis or epididymis was noticed in association with the condition.

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TABLES

Table-1

Pathological conditions of the testis and epididymis.

Sl. No.	Conditions	Total genitalia examined	Incidence	
			No.	%
1.	Hypoplasia	1000	58	5.8
2.	Cryptorchidism	"	29	2.9
3.	Ectopic testis	"	4	0.4
4.	Degeneration (severe)	"	4	0.4
5.	Adhesion (localised)	"	5	0.5
6.	*Atrophy	"	13	1.3
7.	Testicular haemorrhage	"	1	0.1
8.	Male pseudo hermaphroditism	"	1	0.1
9.	Spermiostasis	"	11	1.1
10.	Spermatic granuloma	"	1	0.1
11.	Epididymal haemorrhage	"	4	0.4
12.	Epididymal melanosis	"	43	4.3
	Total	1000	174	17.4

* This includes 13 cases of diffuse adhesion.

Table-2

Developmental aberrations of the testis and epididymis

Sl. No.	Conditions	Total genitalia examined	Incidence	
			No.	%
1.	Cystic persistent mesonephric tubules	1000	89	8.9
2.	Appendix epididymis	"	147	14.7
3.	Cystic remnants of Mullerian duct	"	22	2.2
	Total	1000	258	25.8

P L A T E S

PLATE - I

Fig - 1.

Fig - 2.

Fig 3.

PLATE - II.

Fig - 4.

Fig. 4 Bilaterally hypoplastic testis showing dividing spermatogonial cells with pyknotic nuclei. H & E. stain; x 400.

Fig - 5.

Fig. 5 Unilaterally hypoplastic testis showing cellular differentiation upto the formation of spermatocytes. H & E. stain, x 400.

PLATE-IV.

Fig-8.

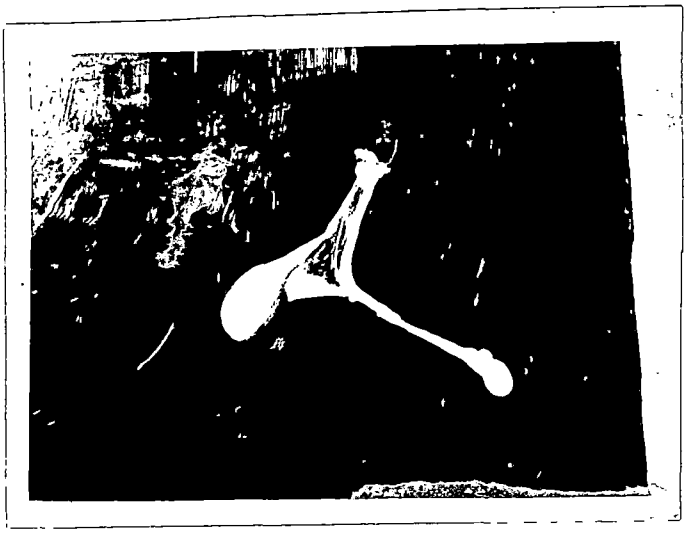

Fig-9.

Fig-10.

PLATE - V.

Fig. 11.

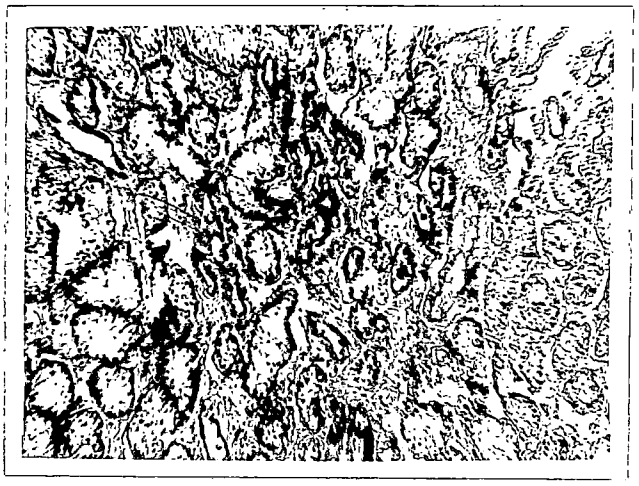

Fig. 12.

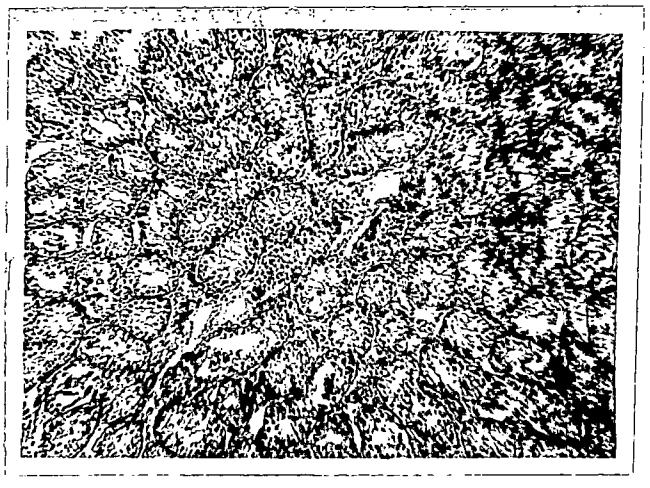

PLATE - VI.

Fig - 13.

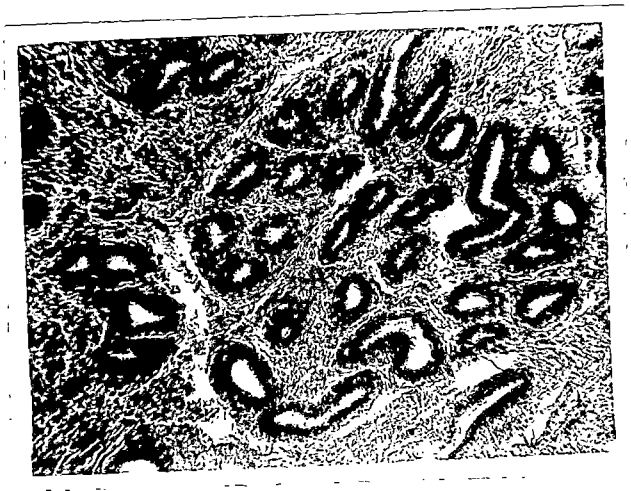

Fig - 14.

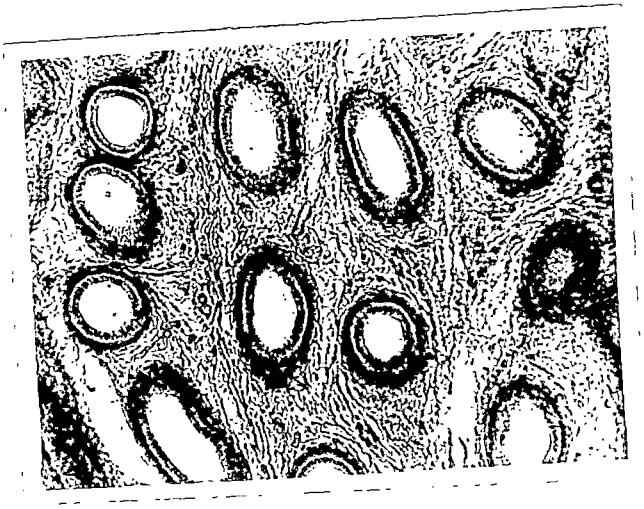

PLATE. VII.

Fig. 15.

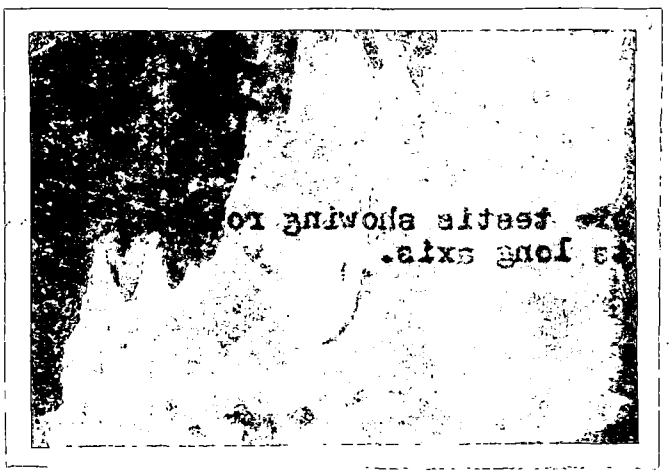

Fig. 16.

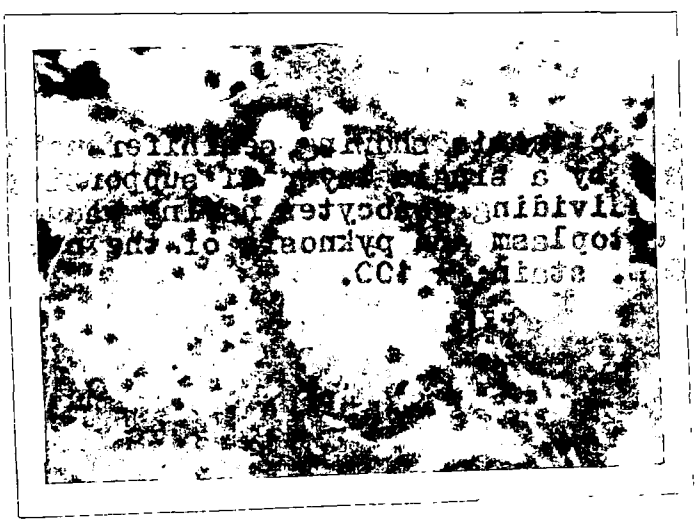

PLATE - VIII

Fig - 17.

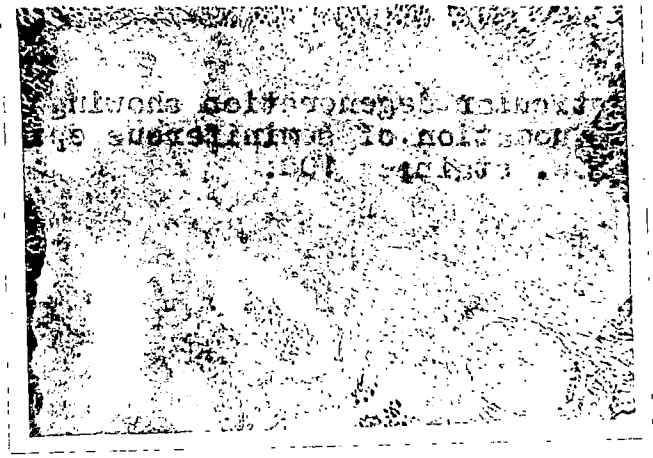

Fig - 18.

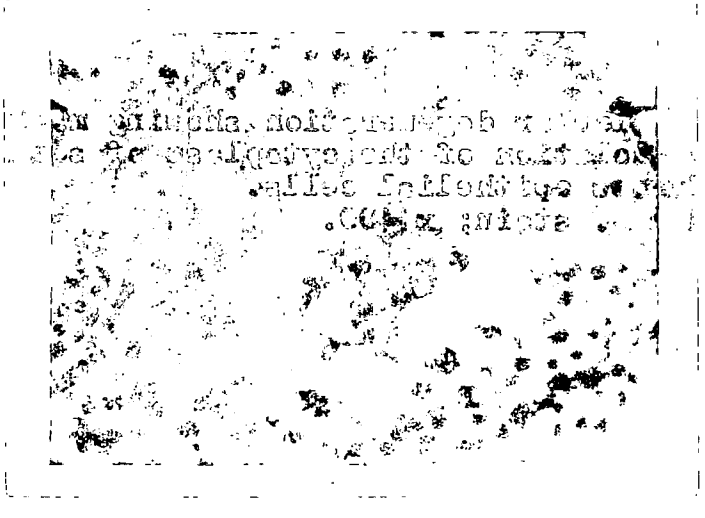

PLATE - IX.

Fig - 19.

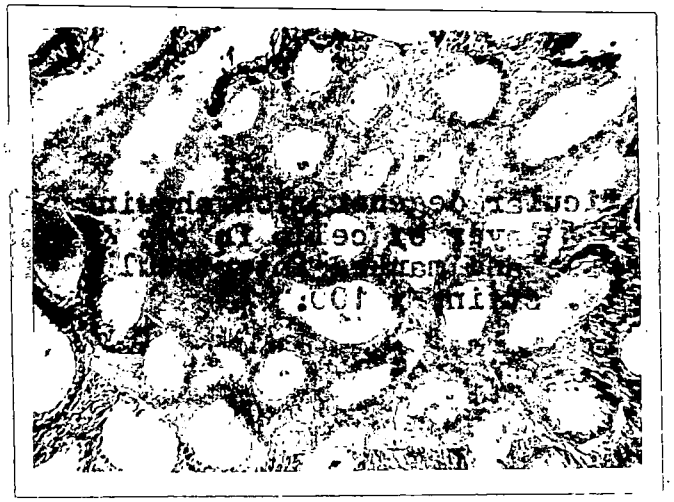

Fig - 20.

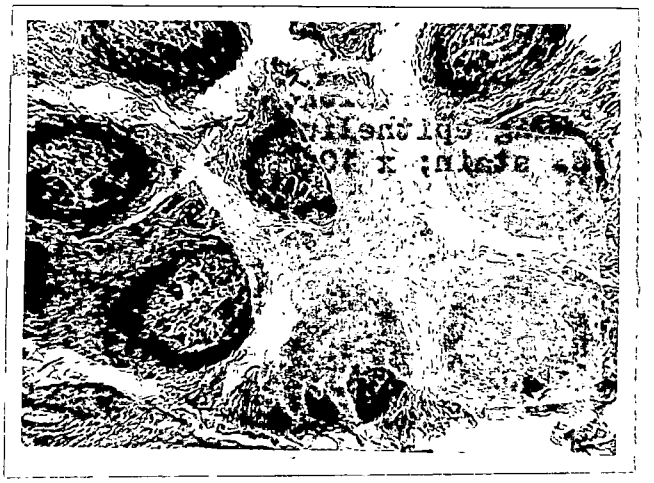

PLATE - X.

Fig - 21.

Fig - 23.

PLATE - XI.

Fig. 24.

Fig. 25.

Fig. 26.

PLATE. XII.

fig - 27.

FIG. 27. [illegible] showing calcification of the [illegible] the seminiferous [illegible] x 130.

fig. 28.

FIG. 28. [illegible] double showing calcification of the [illegible] epithelium [illegible] x 130

PLATE - XIII.

Fig - 29.

Fig. 30.

PLATE - XIV.

fig - 31.

Fig. 31. Testicular haemorrhage showing a thick band of [illegible] red blood cells.
[illegible] x [illegible]

fig - 32.

Fig. 32. Testicular haemorrhage showing the penetration of blood into the testicular parenchyma.
[illegible] x 130.

PLATE - XV.

Fig - 33.

Fig. 33. Inter[illegible] pseudo
[illegible]ded
[illegible]me with
[illegible]

Fig. 34. Testis of male pseudo hermaphrodite showing part of testicular tissue within the tunica albuginea.
X 105. Stain: [illegible]

Fig - 34.

PLATE - XVI.

Fig - 35.

Fig. 36. Vertical section of male pseudo hermaphrodite animal
showing normal structure.
Magnified x 105.

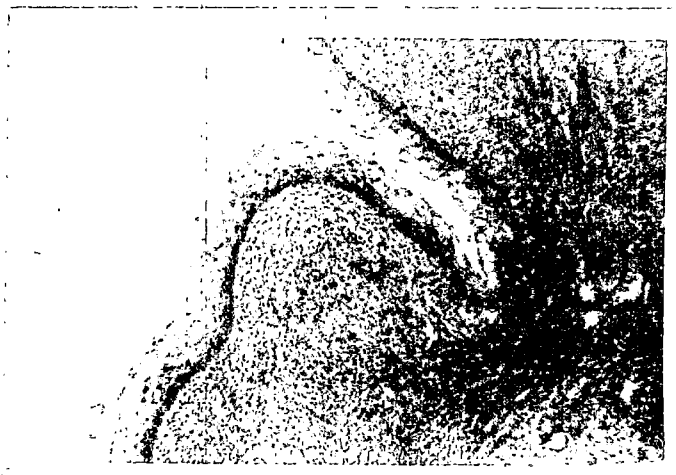

PLATE - XVII.

fig - 37.

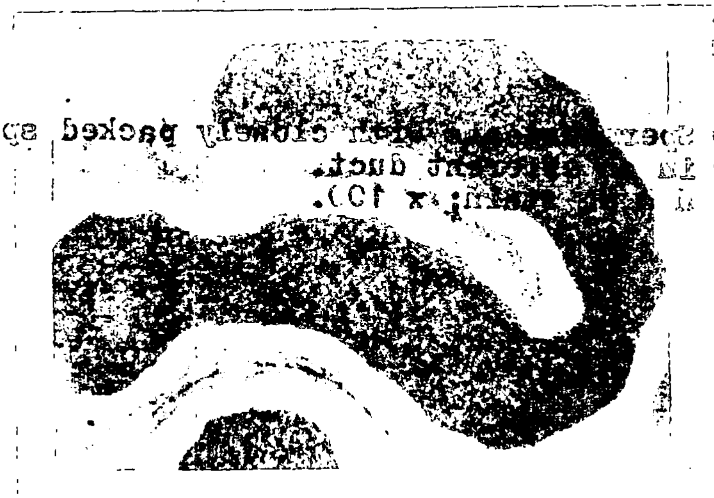

fig - 38.

PLATE - XVIII.

Fig -39.

Fig. 39. Bilateral [illegible] granuloma showing nodules at the [illegible] different stages.

Fig. 40. [illegible] granuloma showing destruction of [illegible] epithelium of epididymis, penetration of sperms into the interstitium and a typical

PLATE - XIX.

fig - 41.

fig - 42.

fig - 43.

PLATE - XX.

fig - 44.

fig - 45.

fig - 46.

PLATE - XXI.

Fig - 47.

Fig. 47. Appendix epididymis exteriorly lined by a [illegible] of tall columnar cells resting [illegible] similar to epi[illegible]didymis.

Fig-48.

Fig. 48. Appendix epididymis (haemorrhagic type) showing closely packed red blood cells, masking the [illegible] structure within.
[illegible]; x 45.

PLATE - XXII.

fig - 49.

Fig. 49. Cyst
epi
cau
H

Fig. 50. Cystic remnant of Müllerian duct
embedded in the connective tissue
layer of vas deferens.
H & E. stain; x 40.

STUDIES ON

THE PATHOLOGY OF TESTIS AND EPIDIDYMIS OF BUCKS

By

JOSEPH MATHEW

ABSTRACT OF A THESIS

Submitted in Partial fulfilment of the requirement for the degree

MASTER OF VETERINARY SCIENCE

Faculty of Veterinary and Animal Sciences

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ABSTRACT

Infertility or sterility in bucks are almost always caused by testicular or epididymal affections. Hence a study on the pathological conditions of these organs might give an approximate idea of the nature and prevalence of sterility conditions in male goats. The present study was taken up with this object in view.

Thousand pairs of testicles and epididymis of bucks, aged 6-18 months were collected at random from the abattoir and examined. The organs which revealed gross lesions and those which were suspected to be affected were subjected to detailed histopathological studies.

Out of the thousand pairs of organs examined, 174 (17.4%) revealed various pathological lesions. The following pathological conditions were observed during the course of the present study: Testicular hypoplasia (5.8%), Cryptorchidism (2.9%), Ectopic testis (0.4%), Testicular degeneration (0.4%), Adhesion with tunics (1.8%), Testicular atrophy (1.3%), Testicular haemorrhage (0.1%), Male pseudo hermaphroditism (0.1%), Spermiostasis (1.1%), Spermatic granuloma (0.1%), Epididymal haemorrhage (0.4%), Epididymal melanosis (4.3%).

In addition, 258 (25.8%) organs showed minor developmental aberrations such as cystic persistent mesonephric tubules (8.9%), appendix epididymis (14.7%) and cystic remnants of Mullerian duct (2.2%).