INFERTILITY AND GENITAL PATHOL0GY OF BOLLS INDIGENOUS TO NIGERIA

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This thesis is an original work carried out by myself and no part of it has been presented elsewhere for a degree or diploma.

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SECTION I

Introduction

Animals play a major role as a source of protein in the world. Kriesberg (1968) reported that the most important and widespread nutritional deficiency is protein. With the world population still on the increase, food shortage will inevitably become more critical and the protein deficiency more acute. This is more likely to be the case in the developing countries like Nigeria, and other African countries. The daily intake per capita of animal protein in Nigeria has been estimated at 5 to 8 grams (F.A.O. 1971; Idusogie, 1971; Olayide et al., 1972). This is already, far below the recommended average of 5 to 8 grams (F.A.O. 1971).

The number of livestock in Nigeria is adequate and matches that of most developed countries (Kumi-Diaka, 1976) and yet protein supply is at its lowest. Among the causes of inadequate supply are low reproductive performance, poor and inadequate nutrition for the livestock, and poor nomadic husbandry practices (Kumi-Diaka, 1976). Over 90% of the reported 8.5 million cattle in Nigeria are in the hands of the nomadic Fulani (Nuru, 1974). They are excellent cattle-men but their traditional nomadic husbandry practices are not production-oriented. They roam about with their cattle all year round in search of food. Livestock under-nutrition is their major problem. Undernutrition and nutritional deficiencies in livestock are problems that fluctuate with the rainy season and the dry seasons in Nigeria. The number of animals in a herd determines one's richness and to some extent, one's social status.

For this and other reasons, the Fulani keeps all the animals - sick and old; and excess bulls in the herd.

Other problems facing the livestock industry in Nigeria are management, diseases and their interactions. Tick infestations, cutaneous streptotrichosis and tsetse flies are serious problems, especially to the cattle industry. Internal parasitism, especially helminthiasis, poses a serious threat to the livestock owners. This generally runs a chronic course in the affected animals leading to emaciation, poor food utilization, and poor reproductive performance. Heavy annual losses are sustained by livestock owners through diseases and mismanagement.

The Nigerian Government is making every avenue available, to enhance the livestock industry. Exotic cows/bulls have been imported in an attempt to upgrade the reproductive and productive performance of the indigenous cattle in Nigeria. However, many research workers believe that the indigenous animals have all the potentials; and that rather than importing exotic animals, the adaptation of the indigenous food-producing animals within their environment, must be investigated.

Several factors, such as nutrition, seasons and diseases such as corynebacterial infections influence the reproductive performance of animals. The literature is full of informations on the female animal (cows, in this case). But there is very little information on the reproductive performance of the bull and the role it plays in the fertility and infertility problems of the cows, in the tropics, especially Nigeria. The objectives of this investigation, therefore were:

1. To study spermatogenesis of healthy and poor-condition bulls indigenous to Nigeria.

2. To study the gross and histopathological conditions of the genitalia of bulls indigenous to Nigeria.

3. To study the effect of experimental infection of Corynebacterium pyogenes organisms on the reproductive organs of bulls indigenous to Nigeria.

Literature Review

A. Reproductive performance

a. Fertility indices in bulls:

Reproductive soundness of indigenous bulls in the tropics and especially in Nigeria has not been extensively studied. Common parameters used to estimate breeding performance in bulls include semen characteristics, scrotal circumference and other scrotal dimensions, and the physical conformation of the bull. In the past, emphasis was mainly laid on semen characteristics such as: motility, concentration, morphology, volume, appearance (this gives an indication of the concentration of sperms or total count), and the percentage of live cells.

Tests for semen quality are usually performed under field conditions on samples collected by electro-ejaculation. However, in a study conducted by Ball et al., (1974) all of these tests became suspect because they lack repeatability. A semen sample that grossly deviates from the normal is easily "spotted", whereas a sample that may show a low percentage of sperm cells having changes in morphology may be responsible for a high degree of infertility. The findings that make these criteria less precise than expected, are summarised below.

(1) Appearance: (table 1)

Concentration was not a valid biological instrument of fertility in beef bulls (Wiltbank et al., 1965); Sexual inactivity might result in first ejaculates showing high concentration even though daily sperm output of the testes is low. Bulls with extensive sexual activity could produce a semen sample that is thin even though daily sperm production is high (Ball et al., 1974). Ball et al. (1974) recommended that estimation of concentration should be abandoned as one of the criteria for semen evaluation.

(2) Motility:

Ball et al. (1974) classified sperm motility as follows:-

Motile	Classification
80 - 90%	(5) Very good
60 - 80%	(4) Good
40 - 60%	(3) Fair
20 - 40%	(2) Poor
0 - 20%	(1) Very poor

Repeatability of gross motility estimates is not very good under field conditions. Gross and individual evaluations are subjective. Such evaluations are therefore influenced by the person making the observations, and differ with time and person. Physiological factors in the bull may affect motility levels and cause day-to-day variations, i.e. response to ejaculator or

a drop or two of urine (Wiltbank et al., 1965).

(3) Morphology:

Semen morphology, especially primary sperm cell abnormalities and percentage normal sperm cells are highly repeatable (Ball et al., 1974). There is considerable evidence that increased sperm cell abnormalities are associated with poor conception rates in all species (Rao, 1971). Some types of sperm defects (primary) occur when spermatogenic epithelium misfunctions, others when the excurrent duct system is not functioning properly. Rao (1971) reported that primary abnormalities are the most significant and should not exceed 18 to 20 percent or reduction of fertility is likely. He also noticed that, secondary sperm abnormalities fluctuate, but excess of these is also associated with poor reproductive performance. Rao (1971) and Cupps and Briggs (1965) stated that if more than 35% of sperm cells were abnormal, conception rate was likely to be lower than 30% on one service if it is obvious that semen morphology reflects the functional condition of the testes and the excurrent duct system.

(4) Percentage of live spermatozoa:

Ball et al. (1974) concluded, from studying several data, that repeatability of estimates of live cells made by the live - dead staining method are not dependable. Results of estimates were not only difficult to duplicate, but also differed from two individuals counting the cells on the same slide, and also from different ends of the slide, as illustrated by the data in table 2.

(5) Scrotal circumference:

Several investigators (Hahn, et al., 1969; Willet and Ohms, 1957; Ball et al., 1974) recommended the use of the scrotal circumference in the evaluation of breeding soundness in the bull. According to them, this is a more accurate index of semen producing capacity and also it is highly repeatable. It has been known that there is a correlation between scrotal circumference, testes size and semen production (Willet and Ohms, 1957), especially in young bulls (tables 3 and 4). The scrotal circumference is probably the best measurement available for field evaluation of semen-producing capability. Hahn et al. (1969) suggested that most yearling beef bulls should have a scrotal circumference of at least 30 cm. They observed that young bulls with relatively small testes still had small testes at maturity. Willet et al. (1957) suggested that, whereas it might not be of any use in older bulls, any young bull (bull calf) with scrotal circumference below the 80% confidence limits for bull calves of its age group should be viewed with suspicion that it may be subnormal in development. Accurate prognosis of potential high semen output could probably be made at an early age.

According to the investigators cited above, the only accurate and therefore good parameters for the evaluation of breeding soundness in the bull, under field conditions are: (1) semen morphology, (2) semen motility, and (3) scrotal circumference.

Table 1 : Evaluation of semen motility*

Grade	Appearance
Very good	Vigorous swirls and eddies
Good	Slow swirls and eddies
Fair	No waves - some cells moving
Poor	Slow, laborious progression of a few spermatozoa

* Ball et al., (1974).

Table 2*: Cell counts of different ends of slides stained with eosin - nigrosin stain.

Mean - %: Unstained on front end	Mean - %: Unstained on back end
25.75	39.25
45.25	51.75
25.00	45.50
38.25	50.00
32.25	42.00
27.00	44.50
40.50	50.50
28.50	43.50
23.50	39.50
45.74	66.75
63.25	70.25
47.74	53.25
50.25	54.25
26.25	57.50
41.50	52.00
$\bar{X}$ 37.38**	49.17**

1. * Ball et al., (1974)
2. ** Means are different ($P < 0.01$).

Table 3**: Age, scrotal circumference, and sperm output

Age (yr)	Circumference (cm)	Weekly sperm output		*Correlation coefficients
		Total ($X 10^9$)	Per gram ($X10^6$)	
1.6	35.1	28.4	61.2	0.81
3.2	40.0	41.5	41.5	0.72
4.0	40.0	38.0	53.6	0.64
5.2	42.0	42.7	52.8	0.40
Aged (6+)	42.6	28.2	34.4	0.22

* Correlation of scrotal circumference to sperm output/gm testis.

** Hahn, Foote and Seidel, Jr. (1969).

Table 4: Age and scrotal circumference

Breed of bulls	Number of bulls	Average age (mos)	Average scrotal circumference (cm)
English beef breeds[2]	37	10.5	30.6
	36	12.0	32.7
	20	13.5	34.5
Angus[4]	28	mature	38.3
Hereford[4]	12	mature	37.9
Shorthorn[4]	13	mature	40.3
Santa Gertrudis[1]	54	14.00	34.9
	11.3	19.2	35.8
	21	29.5	38.6
Charolais[1]	30	16.5	34.3
	41	18.0	35.0
	71	20.5	35.0
	9	29.0	37.9
Dairy[3]	59	13-18	34.9

[1]Ball et al., (1974):

[2]Carroll et al. (1963)

[3]Hahn et al. 1969:

[4]Willet and Ohms (1957)

b. Fertility indices in cows:

It is accepted that the best and most reliable indicator of reproductive performance of bulls is fertility of the serviced cows. It is therefore obvious that some of the parameters used in evaluating reproductive performance in cows, can be a reflection of reproductive capabilities of bulls. Obviously a low reproductive performance may be influenced by factors in the female as well as in the males. Parameters commonly used to estimate breeding performance in cows include: age at first calving, calving interval, 60 and 90 - day non-return rate, first service conception rate and generation interval. Those parameters can be used, at least indirectly, to evaluate the breeding soundness of bulls, especially where artificial insemination is extensively practiced.

Reproductive performance of indigenous cattle is low (Wheat and Broadhurst, 1968; Lamorde and Weinman, 1972; Akpokodje, 1974). The role of indigenous bulls in reproductive performance has not been extensively investigated in Nigeria. Some of the available information is summarised in tables 5 and 6.

c. Seasonal reproductive pattern:

Seasonal reproductive pattern of indigenous bulls in Nigeria has not been extensively studied. There is very little information on reproductive pattern of indigenous bulls in Africa and other tropical, countries especially, developing countries.

In a study of spermatogenesis in indigenous bulls in Nigeria, it was found that, although spermatogenesis was active throughout the year, the spermatogenic epithelium indicated increased activity during the rainy and pre-dry seasons (Kumi-Diaka, 1976). Those seasonal peaks coincided with the seasonal breeding activity of the indigenous cows (Osori, 1976).

Table 5*: Reproductive performance of indigenous cattle in Nigeria

Parameter	Nomadic cattle	LIBC** Govt. herds	Optimum performance
Age at first calving	60 mos	40 mos	26 - 28 mos
Calving interval	17-24 mos	16-18 mos	13 mos
Annual calf crop	40%	60%	85-90%
Calves produced in life-time	2½	4	8

* Zemjanis (1974): ** Livestock Improvement and Breeding Centre

Table 6*: Temperate zone standards for comparing reproductive efficiency of indigenous cattle of Nigeria

Parameter	Average	Good
90 - day non-return rate	65 - 70%	Above 75%
Services per conception	1.8 to 2.2.	Below 1.5
Calving interval	13.5 to 15 mos	12 to 13 mos
Annual calf crop	70 to 80%	Above 90%
Days open	120	100 or less

* Hafs et al. (1973)

B. Normal spermatogenesis

The testes of bulls, like the males of all animal species, perform a dual function - a gametogenic and endocrine function. The endocrine function is the production of testosterone from the interstitial cells - Leydig cells and the gametogenic function is the production of spermatozoa from the seminiferous tubules.

Spermatogenesis is a synchronised process whereby spermatozoa, the final product, containing a haploid number of chromosomes are formed from the precursor diploid spermatogonia (Knudsen, 1954; Ortavant, 1958). The process of spermatogenesis comprises spermatocytogenesis and spermiogenesis. The former is the formation of primary and secondary spermatocytes from spermatogonia Type A. Spermiogenesis is the formation of mature spermatozoa from immature spermatids (Roosen-Runge, 1955; Ortavant, 1958).

According to Ortavant (1959), spermatocytogenesis is the proliferative phase in which the spermatogonia are multiplied by a series of mitotic divisions, ending with a final number of 16 (in the bull) primary spermatocytes from one spermatogonia Type B. The next event is a meiotic division of the primary spermatocytes in which the chromosome number is halved but the total number of the secondary spermatocytes is doubled (32 in the bull). The secondary spermatocytes undergo a further meiotic division, an equatorial one as far as the chromosomes are concerned, but ending up with 64 spermatids as the final product, each having the haploid number of chromosomes (Roosen-Runge, 1955; Ortavant, 1958; 1959).

The formation of spermatids signifies the end of spermatocytogenesis and the beginning of spermiogenesis.

Spermiogenesis begins with the formation of the spermatids in the seminiferous tubules. It begins in the seminiferous tubules and completes in the epididymis (Ortavant, 1956, 1958). The series of complex structural reorganizations which occur during the process of spermiogenesis is summarised (Leblond and Clermont, 1952; Knudsen, 1954):

1. The newly formed spermatid is a large rounded cell with spherical nucleus.
2. Golgi zone soon forms and gives rise to the acrosome of the spermatozoon.
3. Centrioles are formed and later give rise to the flagellum and still later on, to the body and tail of the spermatozoon.
4. Mitochondria are arranged around the flagellum and form the midpiece.
5. At completion of spermiogenesis when the spermatozoa are in the head of the epididymis, unused cytoplasm and golgi material are pressed out and appear on the tail as the cytoplasmic droplet.

The germinal cells.

a. Spermatogonia:

Ortavant (1958) defined the spermatogonia as "the combination of germ cells contained in the parietal layer of the seminiferous epithelium before the primary spermatocytes are formed". Morphologically, three classes of spermatogonia have been identified in the bull (Ortavant, 1958, Leblond and Clermont, 1952; Kundsen, 1954).

These are:

(1) Spermatogonia type A;

Also termed spore cells, the spermatogonia type A is poor in cytoplasm, with a nucleus sprinkled with fine chromatin granules. This gives it a "dustlike" appearance. It is found on the basement membrane of the seminiferous tubules, with the axis of the chromosome spindle nearly always parallel to the membrane of the tubule (Regaud, 1901; Schoenfeld, 1901; Ortavant, 1958; Abdel-Raouf, 1960).

(2) Spermatogonia type B.

The nucleus which tends to be spherical is smaller in size, reaching 8-9 μ in diameter, and exhibits "crustlike" chromatin granules which tend to adhere to the nuclear membrane giving the former a thickened "crust-like" appearance (Regaud, 1901; Ortavant, 1958, Abdel-Raouf, 1960).

(3) The intermediate spermatogonia:

This is seen with difficulties under the light microscope.

The appearance of the nucleus varies with the stage of division. The chromosomes are elongated during the prophase of the type A spermatogonia, and shorter and contracted during the prophase of the type B spermatogonia. Spermatogonia cells have also been observed in the metaphase, anaphase and telophase with the spindle axis parallel to the basement membrane (Ortavant, 1958; Abdel-Raouf, 1960; Roosen-Runge, 1955; Venge 1952).

b. Primary spermatocytes:

These are large spherical cells mostly observed in prophase and metaphase. In metaphase the spindle has the shape of a double acute-angled cone with spindle fibers attached to the edges of the chromosome plate. These cells form different layers central to the spermatogonia, towards the lumen of the tubule (Knudsen, 1958; Abdel-Raouf, 1960).

c. Secondary spermatocytes.

These are spherical cells of smaller size than the primary spermatocytes and slightly larger than the spermatids when they are in the prophase stage. The nucleus is rounded and contains several karyosomes. In the metaphase, these cells are characterized by their narrow long spindle, the fibers of which are not attached to the edges of the chromosome plate. These cells are not often seen in the testes, even in fully mature animals with active spermatogenesis. This is probably due to the very rapid division of these cells (Abdel-Raouf, 1960).

d. Spermatids:

The shape and position of these cells vary with the stage of seminiferous epithelium cycle. In the early stages the cells are located centrally to the spermatocytes in two and sometimes up to five layers. They are spheroids with a round nucleus which show up to four dark granules. These cells are in the golgi or cap phase (Leblond and Clermont, 1952). In the acrosome phase, the cells wander towards the sertoli cells and form groups or clusters. The cells are elongated

and the nucleus more oval. The nucleus is stained brickred. The cells are deeply situated and some are found to be close to the spermatogonia or even the basement membrane. Others are found at the tip of the sertoli cells (Leblond and Clermont, 1952; Knudsen, 1954; Ortavant, 1956, 1958; Abdel-Raouf, 1960).

Clermont and Leblond (1955) described and classified spermiogenesis in mammals into the following phases:

Golgi phase: during this phase, the cytoplasm of the newly formed spermatid, the idiosome - a poorly stained and irregularly spherical zone can be observed. Proacrosomic granules - several PAS reactive particles - are seen in the idiosome. These granules eventually coalesce to form a single particle, the acrosomic granule.

Cap phase: the main event is the formation of the head cap. The acrosomic granule increases in size, flattens slightly and expands over the nuclear surface to form the head cap. It eventually covers half of the nuclear surface.

Acrosome phase: the spermatid becomes oriented. The acrosomic granule and head cap bulge on the cell surface facing the outside of the tubule, while the cytoplasm accumulates on the lumen side. Finally the nucleus elongates and flattens, while there is a transformation of the acrosomic granule into acrosome.

Maturation phase: the spermatid completes its transformation/evolution into a spermatozoon. The acrosome flattens at the apex of the head cap, and the reactivity of the head cap and acrosome decreases markedly. (Clermont and Leblond, 1955).

Major events in spermatogenesis can be schematically represented as:

Spermatogenesis ⟶ I Spermatocytogenesis
Spermatogenesis ⟶ II Spermiogenesis

I Spermatocytogenesis: (Begins with resting Spg A and ends with Spermatid)

Resting Spermatogonium A
↓
Active Spg A --> Resting Spg A
↓
Intermediate Spg
↓
Spg B (1)
↓
Prim. Spcyte (16)
↓
Sec. Spcyte (32)
↓
Spermatid (64)

II Spermiogenesis (Begins with spermatid and ends with mature spermatozoon. Begins in the seminiferous tubules and is completed in the epididymis).

Events:

Newly formed spermatid

↓

Idiosome

↓

Proacrosomic granule

↓

Acrosomic granule

-> I head cap (fits tightly on nucleus)

-> II acrosome (continuous with head cap, flattened at its apex).

--> Mature spermatozoon (final product 64)

e. Spermatozoon

The fully formed, mature sperm cells form a regular ring with the heads directed towards the basement membrane and the tails projecting into the lumen of the tubule (Abdel-Raouf; 1960; Kumi-Diaka, 1976). The dorsoventrally flattened, oval shaped head of the bull spermatozoa consists almost entirely of the nucleus. It is clear, homogenous and light blue-voilet while the tail is rose-red when stained with hematoxylin and eosin (Blom and Birch-Andersen, 1960, Clermont, 1972, Kumi-Diaka, 1976). The proximal part of the nucleus is protected by the acrosomic cap (Clermont, 1972). The neck, which connects the head of the spermatozoon to the midpiece, is composed of basal granules and bundles of fibrils. The tail, which is about

40μ - 50μ long, is differentiated into three parts; midpiece, mainpiece and endpiece (White, 1972). The midpiece, the thickened region between the head and the mainpiece, is about 10μ to 15μ long. The mainpiece is the longest part of the tail (30μ) and provides most of the propellant mechanism. The endpiece is the short terminal portion (3μ) not surrounded by a sheath (Blom and Birch-Andersen, 1960; Fawcett, 1961; Wu and Newstead, 1966). Residual bodies, which are large cytoplasmic structures are observed at the time of completion of the maturation phase of the spermatids and before the sperms are completely released into the lumen. They are oval or round, granular, and stain pale red with hematoxylin and eosin. They are situated around the tails of the newly formed sperm as well as free in the lumen of the seminiferous tubules. In the tubules, when all the produced sperm had left, these bodies are absent (Abdel-Raouf, 1960).

Non-germinal cells

a. Sertoli cells are large, elongated cells located at the basement membrane of the seminiferous tubule. The cell boundaries are not distinct but the cells are identified by their characteristic nuclei. The irregularly shaped nucleus is large, light, either pyramidal or oval with variable situations in the cell cytoplasm. The nuclear membrane has deep indentations, the nucleolus is prominent and the cytoplasm is scanty. The cells have multiple long processes which surround spermatocytes and spermatids. Sertoli cells are the only non-germinal cells in the seminiferous epithelium and they are fundamental to normal spermatogenesis. They are rich in glycogen, glycoproteins, lipids and some steroids. Sertoli cells remain in contact with the

vascular supply, hence the term "nurse cells". They are also believed to offer protection and support to the developing elongated spermatids, until the latter become transformed into mature spermatozoa. The precise role played by the sertoli cells is still not clearly understood. In addition to furnishing nutrition for the germ cells, they undergo cyclic transformation which is coordinated with the cycle of the seminiferous tubules. Sertoli cells have also been called foot cells, sustentacular cells and trophocytes (Ortavant, 1959; Cole and Cupps, 1959; Abdel-Raouf, 1960).

The possibility, still unproven, that these cells may also be the source of endocrine secretions or of influences more subtle than nutrient communication which are concerned in germ cell maturation, exists (Eiftman, 1963).

Seminiferous epithelial cycle

During the process of spermatogenesis specific cellular associations arise from the basement membrane and are directed towards the tubular lumen. At each stage of spermatogenesis there are several distinct groups of cells present when a cross-section of the tubule is examined (Kumi-Diaka, 1976). Thus any section of a tubule shows several generations of germ cells. They develop in close relation to each other so that in any given area of the seminiferous tubule, there is a constant development of different cells occurring with cyclic regularity. These cellular associations are called stages of the cycle of the seminiferous epithelium (Faulkner, 1969). The seminiferous epithelium cycle is defined as the series of changes occurring between two successive appearances of the same cellular associations

(Ortavant, 1959; Faulkner, 1969). The spermatogenic cycle represents the time interval required from the first division of resting spermatogonia to the release of spermatozoa into the lumen. The length of the cycle in the bull is estimated to be 55 days (Dawson, 1958; Orgebin et al., 1958). Maturation of spermatozoa in the bovine epididymis is estimated to require 10 days (Orgebin et al., 1958). The entire process of spermatogenesis has been studied and, labelled sperm with radio-active phosphorus showed up in about 90 days in the bull (Cole and Cupps, 1959).

C. Infertility and sterility

I. Non-pathological factors:

"Infertility" can be defined as temporary inability to produce offsprings. Both words are frequently used indiscriminately to describe disturbances associated with reproduction.

Asdell (1968) stated that sterility is not a disease in itself, but may result from the action of several diseases and conditions. Among such conditions are mismanagement and malnutrition.

a. Nutrition and reproduction:

Underfeeding and/or malnutrition plays a role in infertility and sterility. Asdell (1968) and Kalkus (1920) showed that when reproduction suffers as a result of malnutrition, the animal itself nearly always exhibits definite signs of the disorder before the sexual functions are disturbed. This may be indicated by stunted growth, thinness, roughness of coat, depraved appetite or by definite symptoms of a specific deficiency.

The usual form of nutritional deficiency is general underfeeding with rations of poor quality. This is the case in Nigeria where nomadism is widely practised by the Fulani herdsmen. Calves reared under such conditions tend to be retarded in growth/development, and the sexual organs share in this retardation.

In feeding trial experiments in Cornell University, Asdell (1968) and other investigators proved that under-feeding delays the onset of the first heat in heifers. In a similar experiment with Holstein-Friesian bull calves, it was found that the average age at which semen

contained motile spermatozoa was 51 weeks at low feeding level, 43 weeks at the medium feeding level, and 37 weeks at the high feeding level. Delay in spermatogenesis in the retarded calves and reduction in the numbers of spermatozoa produced have been the rule on the lowest energy intake level (Hignett, 1952).

Mineral deficiencies have been implicated in certain forms of infertility in cattle (Eckles et al., 1935; Hignett, 1952; Asdell, 1968). It seems, from the works of Tuff (1923) and Hignett (1952), that phosphorus deficiency interferes with ovarian function, causing, in the milder cases, failure of heat but not failure of ovulation. Although there is no evidence that calcium deficiency causes reproductive problems, many investigators have demonstrated the importance of the calcium-posphorus ratio (Eckles et al., 1935). Iodine deficiency has been blamed for lack of sexual desire and poor quality semen in some bulls (Kalkus, 1920). This may be related to a possible lowered general metabolism due to failure of the thyroid gland to secrete enough thyroxine.

The literature stresses the importance of vitamin deficiencies. Vitamin A deficiency has been reported in the field among range cattle that have been on dry feed or on dried - up range for some time (Asdell, 1968). Asdell (1968) stated that, in extreme cases of vitamin A deficiency, the number of services per conception increases, but the difficulty may be one of implantation. In some species at least, vitamin A deficiency causes cornification of the mucous membrane (of the uterus) and this may interfere with effective implantation, or passage of the spermatozoa to

reach the egg (Madsen and Davis, 1949).

The effect of vitamin A deficiency on reproduction of the bull calf has been reported. Madsen and Davis (1949) and Asdell (1968) reported that bull calves kept on a low carotene diet show a degeneration of the germinal epithelium in the testis and absence of spermatozoa. They stated that the adverse effect, in mature bulls, seems to be not upon spermatogenesis, but upon the ability to mount a cow. Muscular incoordination arises, and may become sufficiently severe to bring about this inability. Thus, vitamin A deficiency may, therefore, not be a major factor in adult male sterility due to defective spermatogenesis. There is little or no evidence that marginal deficiencies of vitamins B and C affect reproduction.

b. Inheritance and reproduction:

Inheritance plays a role in the general infertility problem in cattle and other domestic animals. Most of the cases of inherited infertility that have been found belong to the class with readily recognizable anatomical defects. Such inherited structural anomalies include: white heifer disease (cows); "gonadless" (cows and bulls); inherited hernia (bulls); impotence due to failure of sigmoid flexure to straighten (bull); intersexuality in which apparently normal bulls are infertile (Yapp, 1947). The most clear-cut case of inherited infertility in cattle is hypoplasia of testes or ovaries.

According to Asdell (1968) gene or genes that prevent fertility (ability to carry a calf during gestation and to deliver it at term in a viable condition) are called "lethals". They are simple

autosomal recessives and tend to express themselves when line breeding or inbreeding is practiced. These genes cause developmental faults of a nature that prevents the calf from living. Such conditions include achondroplasia, cerebral hernia, short spine, atresia ani, congenital dropsy, congenital spasm, fused nostrils and muscular contracture (Mead et al., 1946; Pou et al., 1953).

In view of the existence of such deleterious genes, it is clear that extensive use of one bull for artificial insemination might spread a lethal gene far and wide among its offspring, thus increasing the chance that two such genes may meet in subsequent breeding operations.

2. Disease factors

Along with faulty managements, disease factors account for the greatest amount of infertility and/or sterility in cattle (Asdell, 1968). These factors may take the form of specific diseases (vibriosis, brucellosis, trichomoniasis, epi-vag etc); that affect the reproductive organs directly, or non-specific diseases that affect reproduction by reducing the status of the animal as a whole. Lagerlof, (1936, 1938, 1957) indicated that any disease that has a severe effect upon the health may affect reproduction. The reproductive organ and tract are susceptible to many deleterious agents and many of the bacteria and viruses that attack cattle. These may produce low-grade chronic infections that impair the effeciency of the genital organ in general.

Abnormal spermatogenesis:

In the female animal, the ovaries are well protected inside the animal's body, and are therefore little affected by outside influence, hence breakdowns in oogenesis seldom occur, except in cases of severe systemic stress. The male domestic animal has his gonads suspended in a vulnerable pouch and therefore is subjected to the stress of trauma and temperature changes, both systemic and ambient. These stresses have an effect upon spermatogenesis, whether of a temporary or permanent nature.

Spermatozoa are produced in the seminiferous tubules which make up over 80-90% of the testicular mass (Jubb and Kennedy, 1970). Inability of the spermatogenic epithelium to differentiate normally may either be congenital (primary) or acquired (secondary). Secondary disturbances are usually associated with testicular pathological conditions, such as degeneration, atrophy, inflammation (Lagerlof, 1939), in which the intrinsic capability of the spermatogenic epithelium to differentiate normally, is prevented from doing so by some external damaging factors. If any intrinsic or extrinsic factor causes injury to the spermatogonia, this will cause permanent damage to the testes, but if only differentiating cells are damaged, repair or regeneration is possible (Faulkner, 1969).

A. Congenital:

a. Testicular hypoplasia

Testicular hypoplasia has been defined as defective or incomplete development of the seminiferous tubules, resulting in varying degrees of abnormal spermatogenesis (Knudsen, 1961; Konig, 1964). Testicular hypoplasia is a congenital under-development of the spermatogenic

epithelium, which however, does not affect the hormone-producing interstitial Leydig cells (Lagerlof, 1934). In total testicular bilateral hypoplasia practically no sperms are produced and no spermatozoa enter the epididymis from the testis. In moderate or unilateral testicular hypoplasia a very small quantity of spermatozoa and/or varying amounts of degenerated cells emanate from the germinal epithelium.

The clinical signs and manifestations of infertility are not spectacular and vary greatly (Konig, 1964; Humphrey and Ladds, 1975). In most cases sexual desire is excellent and coitus is prompt (Jubb and Kennedy, 1970). Lowered conception rates are often evident in bilaterally affected, but incomplete hypoplastic testes. Jubb and Kennedy (1970) reported a conception rate of 20 to 40 percent. Grossly affected testes vary from being smaller and softer than normal to near normal in size and consistency, and the cut surfaces tend to bulge (Lagerlof, 1938; Konig, 1964; Humphrey and Ladds, 1975). Konig, (1964) reported that the hypoplastic testis, is often not observed until after puberty, and may be as small as a quarter to a third of normal size. Histologically, hypoplastic tubules are characterised by partial to complete lack of germinal elements, predominance of sertoli cells, or failure of spermatogenesis depending upon severity (Lagerlof, 1938; Kodagali et al., 1971; Humphrey and Ladds, 1975). Gustavsson (1969) observed aspermia in bilateral hypoplasia, and reported that the seminiferous tubules of the affected testes contained sertoli cells but no spermatogenic epithelium. In testes of other bulls he observed, that the differentiation of the spermatogenic epithelium proceeded up to and including the spermatid stage, but without formation of mature sperms. Pyknotic nuclei were abundant

and cells with pyknotic nuclei and vacuolated cytoplasm sloughed off into the tubular lumen. In other sections of the same testis, arrest of spermatogenesis appeared at the spermatocyte stage. In certain cases of hypoplasia, seminiferous tubules showed more or less reduced number of spermatogenic cells intermingled with completely normal or completely hypoplastic ones (Kumi-Diaka, 1976). Other investigators reported the presence of giant cells in the affected tubules and also hyperplasia of the interstitial cells (Lagerlof, 1938; Laing and Young, 1956). Lagerlof (1939) reported a reduction in size of the seminiferous tubules in hypoplastic testis. On the basis of numerous tubular measurements, Konig (1959) prepared a scale showing expected tubular diameters (in microns) for hypoplasia and other congenital conditions of the testis. Lagerlof's classification of testicular hypoplasia is summarised (1939):

1. A small proportion of tubules hypoplastic; germinative cells several layers thick; some spermatocytes with hydropic degeneration; spermatid giant cells present; number of mature spermatozoa small; tubular diameter varying from 130 to 310 microns.
2. Approximately two-thirds of tubules hypoplastic with only one layer of cells, resembling sertoli cells; some tubules have spermatogonia, others spermatocytes in various stages of development, and others no cells at all; numerous giant cells present; tubular diameter varying from 110 to 250 microns and;
3. Only a single layer of cells (sertoli cells) present; no germinal cells; basement membrane of normal thickness; tubular diameter varies from 100 to 250 microns.

Many investigators agree that differentiation between hypoplasia and degeneration is difficult because of the variable histological manifestation of hypoplasia (Bishop, 1970; McEntee, 1970; Humphrey and Ladds, 1975). Grossly, diagnosis especially of bilateral hypoplasia, is often erroneous especially in young bulls. Histological assessment of the testis is needed for a tentative diagnosis. This could be achieved through testicular biopsy. But incidentally, biopsy techniques have been known to cause deleterious effects on the testis and testicular function. The sequelae of testicular biopsy include orchitis and degeneration (Lagerlof, 1936; Jubb and Kennedy; 1970).

The incidence and frequency of testicular hypoplasia in bulls have been reported by several investigators. Lagerlof (1934) described testicular hypoplasia as a cause of 23 percent of testicular pathology in Swedish Highland breeds. Bloom and Christensen (1951), Haq (1949) and Vander-Sluis (1953) reported an incidence of 12 to 17 percent. Settergren (1961) observed that gonadal development in Swedish Highland breed, a polled breed, nearly all, 90 percent or more were white. In these bulls hypoplasia of the left testis occurred in 25% of all the bulls, of the right testes in 1% and of both testes in 4 to 5%. Carroll et al. (1963) reported an incidence of 20% of offsprings with hypoplasia and poor semen quality.

Etiology of testicular hypoplasia:

Testicular hypoplasia, according to most investigators, is congenital, and possibly hereditary in origin, and is caused by a marked lack of or reduction in spermatogonia in the gonads during fetal life (Lagerlof, 1934;

Kenney, 1971; Humphrey et al., 1975; Konig, 1964; 1959; Kodagali et al., 1971). Eriksson (1943) suggested a single recessive autosomal gene with incomplete penetrance as a cause of testicular hypoplasia, and actually demonstrated this hereditary basis in Swedish Highland cattle. Hypoplasia may be the result of exogenous factors acting during gestation (Cohrs, 1967). Jubb and Kennedy (1970) described hypoplasia with excessive scrotal fat, probably thermal degeneration was also involved. The defectiveness of the spermatogenic epithelium in certain testicular hypoplasia was found by Knudsen (1961) to be related to sticky chromosomes or multipolar spindle formation. Kenney (1971) suggested that sertoli cell factors might be responsible for the death of the germ cells. Up to date, the concept of testicular hypoplasia being congenital and possibly hereditary in origin has not been disproved, and no specific etiological agent has been known to account for the condition.

Cytogenetic disturbances:

Concomittant with the morphological changes of spermateliosis (spermiogenesis) there is, in several species of animals, an alteration of the typical somatic type of histone into a more basic and arginine-rich histone (Gledhill, 1966). According to classical genetic principles, the number of chromosomes and therefore the amount of deoxyribonucleic acid (DNA) is not altered during spermiogenesis. Each spermatozoa should contain a haploid amount of DNA. However, there are some reports which seem to challenge this concept of a constant haploid amount of DNA - within a species. Leuchtenberger et al. (1953) reported that, one form of human male

infertility might be correlated with a deficient and grossly fluctuating amount of DNA in spermatozoa. Giedhill, (1966) found, with Feulgen microspectrophotometric techniques, that spermatozoa from infertile bulls contained significantly more DNA than did spermatozoa from normal bulls. Although another study (Welch and Resch, 1960) found a statistically non-significant reduction in mean Feulgen-DNA content of ejaculated spermatozoa from infertile bulls. Parez et al. (1960) found a striking fluctuation between normal and abnormal DNA amounts with repeated sampling from the same fertile and infertile bulls.

Other investigators (Knudsen, 1954; Gustavsson, 1969) reported two abnormalities in the primary spermatocytes:

(a) "Stickness" of chromosomes in which they failed to separate at anaphase, and (b) a pyknotic nucleus and multiple spindle formation due to a dysfunction of the mechanism of cell division characterized by extra centrosome division resulting in the formation of giant cells. The authors stipulated that the cause might be a recessive gene as all the bulls investigated belonged to the same family. Gustavsson (1969) described a chromosomal translocation in Swedish cattle that resulted in a degree of infertility characterised by early embryonic deaths.

Sperm cell defects, possibly heritable, have been described in bulls, in which large numbers of spermatozoa produced are similarly affected (Roberts, 1971). These are summarised as:-

1. Abnormal acrosomes or knobbed spermatozoa (Donald and Hancock, 1953; Blom and Birch-Andersen, 1965).
2. Coiled or returned tails and narrow heads (Swanson and Boyd, 1962).
3. Dag defect or strongly coiled, folded or split tails (Blom, 1966).
4. Cockscrew defect (Blom, 1959).
5. Pseudo-droplets defect or rounded or elongated thickened areas in the midpiece (Blom, 1968).
6. Lack of intact cells (Hancock and Rollinson, 1949; Jones, 1962).
7. Eversion of the galea capitis and crater-shaped depressions in the nucleus (Aamdel, 1951).
8. Abaxial tails and swollen midpieces (Aughey and Renton, 1968).

Acrosomal defects are related to defective spermiogenesis involving the Golgi apparatus. Defective acrosomes render the spermatozoa incapable of penetrating and fertilizing ova. The defect may be seen in spermatids (Donald and Hancock, 1953; Saacke et al., 1968; Roberts, 1971). Cytogenetic disturbances in spermatogenesis cause abnormalities in primary spermatocytes by chromosomes failing to separate at anaphase, and extra-centrosomal division resulting in giant cell formation (Knudsen, 1958). Apparently normal semen picture may accompany these defects.

c. Cryptorchidism:

Cryptorchidism can be defined as incomplete descent of the testis or retention of the testis. This is the best known bovine genital defect (Blom and Christensen, 1972). The undescended testis may be located anywhere from just caudal to the kidney to within the inguinal canal, with inguinal cryptorchidism being more common in bulls (Humphrey and Ladds, 1975), and involvement of the left testis seemingly predominating (Wheat, 1961; Gibbons, 1957). Occasionally the testis may be located ectopically under the skin of the ventral caudal abdomen; and this is more commonly the case in male pseudohermaphrodites where no scrotum is present (Roberts, 1971).

Grossly, a cryptorchid testis is small, soft and flaccid (Roberts, 1971). Histologically, there is a tendency towards extreme hypoplasia (Humphrey and Ladds, 1975). No spermatozoa are produced by the retained testis but well-developed or degenerate seminiferous tubules may be present (McEntee, 1970). Spermatogenesis is completely inhibited by the elevated temperature of the affected testis. Leydig cells are not affected so sexual activity is normal or even exaggerated in bilateral cryptorchids.

Cryptorchidism in bull is uncommon (Bishop, 1972), but this condition has been well documented in stallions and boars (Lowe and Higginbotham, 1969).

Bovine cryptorchidism is considered to be inherited but the mode of inheritance is uncertain (Wheat 1961; Bishop, 1972). Wheat (1961) suggested the condition was due to a dominant gene with variable expressivity. Cryptorchidism in swine is a monogenic sex-linked recessive.

d. Segmental aplasia:

Segmental aplasia of the mesonephric duct involving the epididymis, in which distinct portion/segment is absent, has been documented (Carroll et al., 1963; Gibbons, 1957). Uni-and bilateral involvement are described, in which a segment or even entire epididymis or even portion of the vas deferens, may be missing. The corresponding seminal vesicles on the same side may be hypoplastic or missing. Spermiostasis may develop proximal to the missing segment, with the increased pressure resulting in spermatocele formation and degenerative testicular changes, the severity of which is proportional to the proximity of the aplastic segment to the testis (Kenney, 1971). Spermiostasis involves the head of the epididymis and rarely other portions. The associated spermatocele and spermatic granulomas develop slowly. Spermiophagy and polypous epithelial proliferations in the efferent ducts and the rete testis have been observed (Blom and Christensen, 1956; Ashdown, 1963). Konig et al., (1972) considered an autosomal recessive gene to be involved.

e. Miscellaneous anomalies:

Other testicular anomalies possibly due to congenital or hereditary causes include:

1. Scrotal or inguinal hernia: If large, the hernia may depress fertility by markedly interferring with the normal thermoregulatory function of scrotum and testes (Roberts, 1971).

Roberts (1971) reported that the condition is a common hereditary defect in horses and pigs and less common in bulls.

2. Cystic conditions

Two cystic conditions of the bovine epididymis, paradidymis and aberrant ductules were reported (Blom and Christensen, 1956). Although the cysts often cause no harm, spermiostasis and subsequent spermatic granuloma may result (Blom and Christensen, 1960).

B. Acquired:

a. Testicular degeneration

Lagerlof (1934) and many other investigators agree that testicular pathology due to acquired causes is more common than congenital or hereditary causes and include testicular degeneration. Lagerlof (1934) estimated that 75 to 80 percent of all testicular pathology is related to degeneration, including fibrosis. The epithelium of the seminiferous tubule is highly sensitive to any adverse influences with resulting marked effects on spermatogenesis. Testicular degeneration is usually bilateral (Roberts, 1971) as it is most commonly due to generalized disease processes. Unilateral degeneration can occur secondary to local testicular lesions. Combining the semen picture and testicular changes, Lagerlof (1938) clinically classified bovine testicular degeneration into three categories:

1. Bull with considerably reduced fertility but with no morphologically demonstrable change in the genitalia;
2. Bulls of low fertility but with normal appearing genitalia; and
3. Sterile bulls with small atrophic testes.

The pattern and signs of testicular degeneration are the same despite the species and the etiology, variation occurring only in extent and degree (Roberts, 1971; Knudsen, 1954; Bishop; 1970). Grossly the size of the testes

is usually reduced from normal to about half or two-thirds normal size, depending on the duration and degree of atrophy of the seminiferous epithelium. Amann (1960) concluded that since 49.9 to 77.1 percent of the testis volume (depending on age of the bull) is comprised of seminiferous tubules, the close association of the degree of testicular degeneration and testicular size is apparent. The consistency of the testicular parenchyma is altered; soft and flabby in acute, moderate and severe degeneration. The testis does not bulge on sectioning, and eventually there is fibrosis and calcification of the parenchyma, and thickening of the tunica vaginalis and albuginea (Jubbs and Kennedy, 1970; Humphrey and Ladds, 1975). In mild degeneration, consistency differs only slightly from an elastic, tonic, slightly tense, fluctuating normal consistency. The use of tonometer has proven valuable in the objective measurement of testicular consistency (Hahn et al., 1969). Hahn et al., (1969) reported that bulls with soft testes tended to produce fewer spermatozoa.

The histopathology of degenerated testes has been extensively studied (Konig, 1964; Knudsen, 1954; 1958; Roberts, 1971; Kenney 1971; Humphrey and Ladds, 1975). Knudsen (1954, 1958) described the histological and cytogenetic changes in testicular degeneration. He reported three cellular reactions: mild change, with cytoplasmic vacuolation and centrosomal degeneration; moderate change, with complete centrosomal and nuclear spindle fiber degeneration; and severe changes with lack of spindle formation and appearance of restitution nuclei. Some of the commonly observed histopathological manifestations associated with testicular degeneration (and

with increasing severity) were: cytoplasmic vacuolation of the primary spermatocytes, germinal cell desquamation, hydropic degeneration, pyknosis of spermatocytic nuclei, karyolysis, giant cell formation, spermiostasis, calcification of inspissated tubular contents, tubular atrophy, wave-like thickening of the basement membrane, fibrosis, and interstitial cell hyperplasia (Konig, 1964; Bishop, 1970; Jubb and Kennedy, 1970; Kenney, 1971). It has been observed that in extreme degeneration, the seminiferous tubules could become completely denuded with only sertoli cells remaining (Lagerlof, 1938; Knudsen, 1954; Barker, 1956; McEntee, 1970). However, spermatogonia - B cells appear quite resistant and not affected even in severe case of degeneration. The very sensitive primary spermatocytes often exhibit the first degenerative change, which is cytoplasmic vacuolation (Knudsen, 1954). They exhibit contracted bivalents which spread throughout the cell, in grape-like formations (Konig, 1964). The most commonly observed changes in the secondary spermatocytes were changes in size and structure.

If the basal layers of the germinal epithelium, including the spermatogonia and sertoli cells are destroyed, regeneration of the germinal epithelium is not possible, and the bull is sterile (Roberts, 1971). Kenney (1971) and Knudsen (1954) observed that renewal of the seminiferous epithelium depended largely on persistence of spermatogonia and the elimination of the pathologic agent. They considered that once the spermatogonia disappeared or the basement membrane became hyalinized, regeneration was no longer possible.

The fertility of an affected bull will depend on the severity of degeneration and the stage of the process.

Etiology:

Causes of testicular degeneration and other factors affecting spermatogenesis are variable. Some of the causes may be summarised (Lagerlof, 1934; 1936; Roberts, 1971; Hooker, 1944; Galloway, 1961; Carroll et al., 1963; Konig 1964):

1. Thermal - thermal influence associated with elevation of testicular temperature include: cryptorchid and ectopic testes; hernias; scrotal dermatitis due to irritants, mange, localized skin infections or wounds, contusions and hematoma of the scrotum and testes, elevated pyrexia as in certain infectious diseases; frostbite (Roberts, 1971). Donaldson (1963) claims testicular degeneration is most common in temperate breed of bulls, within tropical environment or climate. Lagerlof (1934; 1936; 1938) induced testicular degeneration in bulls by scrotal insulation. Scrotal insulation interfers with the intrascrotal temperature Heat primarily affects the spermatids, the spermatozoa and the spermatocytes, but does not usually affect the spermatogonia (Steinberger and Dixon, 1959). Heat has no effects on the Leydig cells.
2. Vascular lesions - Degenerative changes can be caused by any interference with testicular circulation such as torsion, clamping with an emasculatome, biopsy, and vascular hyaline degeneration in older bulls (Faulkner, 1959; McEntee, 1970). Testicular biopsy in bulls often results

in focal areas of necrosis because of vessel damage when a desirable size of testicular material is taken (Roberts, 1971).

3. Nutritional - Malnutrition constitutes a greater stress to spermatogenesis in prepuberal males than in postpuberal males. Mature males underfed to the point of inanition (loss of 25 to 35 percent body weight) suffer damages to the seminiferous epithelium (Faulkner, 1969). Hypovitaminosis - A accompanied by protein-carbohydrate deficiency affects the germinal and Leydig cells, resulting in testicular atrophy, hypoplasia of the accessory sex glands and delayed puberty in young bulls (Faulkner, 1969; Davies et al., 1957). Moustgaard (1959) believes that vitamin E deficiency plays an insignificant role in infertility among domestic animals, though it causes severe testicular damage in rats. There is considerable evidence that energy dificient diets adversely affect gonadotropin secretion (McDonald, 1969).

   Spermatogenesis can be adversely affected by chronic diseases associated with inanition and debility including severe parasitism, worn teeth or any disease interfering with the intake or passage of food through the intestines (Davies et al., 1957).

4. Irradiation - causes degeneration by injuring the germinal cells - spermatogonia, spermatocytes and spermatids. Irradiation produces its greatest destructive effect in divisional stages, and spermatogonia are more sensitive than spermatocytes (Gillette et al., 1964), the post-divisional spermatids being radioresistant. However, Roberts (1971) reported that spermatocytes are the most sensitive to irradiation while Leydig and sertoli cells are quite resistant.

The type of change in semen quality is related to the type of damage produced, i.e. death of cells causing decreased sperm number, abnormal spermateliosis resulting in morphological defects, or damage to the genetic apparatus resulting in embryonal and fetal death or teratogeny (Faulkner, 1969; Murphree and Parish, 1956; Welch and Murphree, 1965). Spermatids are quite sensitive to X-irradiation, spermatozoa are less sensitive, and spermatogonia are the least sensitive.

5. Hormonal - Moore (1948) considered endocrine failure to be a basic cause of testicular degeneration. Excessive estrogenic hormone produced by sertoli cell tumors, and testosterone, and rarely estrogens produced by the Leydig cells may suppress F.S.H. (follicle stimulating hormone) production and cause testicular degeneration. This condition - dystrophia adiposo-genitalis syndrome - occurs commonly in dogs and rarely, in other animals (Roberts, 1971).

6. Neoplasm - Seldom observed except in old bulls and are usually interstitial cell tumors (Dunn and McEntee, 1964). Dunn and McEntee (1964) reported that degeneration may be due to compressing effect of tumor on the adjacent seminiferous tubules. They also indicated that the degeneration may result from excess steroids produced by the tumor cells.

7. Auto-immunization - Significance under natural conditions is unknown (Roberts, 1971). Degeneration has been experimentally produced in bulls by the subcutaneous injection of autologous testicular material, with Freund's adjuvant. Faulkner (1970) reported that anti-LH (Luteinising hormone) serum will induce testicular degeneration and sterility in male animals.

8. Toxins - A number has been incriminated (McEntee, 1970). McEntee and Olafson (1953) reported testicular degeneration in bulls and rams with hyperkeratosis caused by chlorinated napthalenes. McEntee (1970) cited alkylating agents, cadmium chloride and amphoterin-B as chemicals or metals capable of producing degeneration of the seminiferous epithelium. There is no evidence that systemic organic phosphate insecticides affect semen quality in bulls.

9. Infectious systemic diseases - Pneumonia, shipping fever, I.B.R. - IPV (infectious bovine rhinotracheitis-infectious pustular vulvo-vaginitis), equine infectious anemia, actino-bacillosis, strangles, blue tongue, tick-borne fever in rams, can cause testicular degeneration and lower fertility due to the accompanying fever (DeBoom, 1962; Watson, 1964; Bouters, 1964; Bruner and Gillespie 1966). Other infectious diseases causing gradual debilitation, and testicular atrophy include: foot and mouth disease, actinomycosis, Johne's disease, pyelonephritis and lymphomatosis (Roberts, 1956).

10. Trauma, stress or localised infections - Fatigue, fractured ribs and contusions from fighting, severe arthritis and liver abscesses. Orchitis and epididymitis are common cause of testicular degeneration and the etiological agents include: Brucella abortus, Corynebacterium pyogenes, Actinomyces bovis, "epi-vag" (IBR-IPV), G-up viruses (Bouters, 1964), Nocardia sp., Chlamydia sp. (Storz et al., 1968) and Besnoitia besnoiti (Pols, 1960).

11. Age - Permanent, progressive testicular degeneration occurs frequently with age without any indication of its pathogenesis (Collins et al., 1962; Hahn et al., 1969; McEntee 1970). Hahn et al., (1969) reported that aged bulls, 7 to 13 years old have lower sperm cell output per ejaculate than young bulls, 2 to 6 years old. Many diseases, genetic and management factors influence this age effect.

Conditions associated with advancing age include: increasing prevalence of tunic adhesions (Galloway, 1961; Bishop, 1970); intertubular fibrosis (Hooker, 1944); atrophic tubular changes (Hooker 1944; Knudsen, 1954); calcification (McEntee, 1958); reduction in the relative volume of sertoli cells (Humphrey and Ladds, 1975); vascular lesions and inflammatory cell infiltration (McEntee, 1958; Galloway, 1961) increased lipofuscin and decreased epididymal melanosis (Konig, 1964; Blom and Christensen, 1956).

Miscellaneous

(i) Testes of males of some seasonally breeding species regress after the breeding season and undergo recrudescence near the onset of the next breeding season. This physiological atrophy of the testis is a normal physiological process (Faulkner, 1969).

(ii) Genetic factors have been found to influence acquired testicular degeneration in bulls. The question of increased resistence to degeneration remains to be clarified (Bane, 1954; McEntee, 1970).

b. Orchitis:

Inflammation of the testis may raise the temperature of the testis to the point at which spermatogenesis becomes impossible.

Gibbons (1957) reported that orchitis, in bulls, is usually unilateral and is uncommon, if the results of trauma and degenerative changes are excluded. Orchitis causes morbid changes which result in secondary degeneration of the seminiferous epithelium and often destruction of the tubules (Konig, 1964). Mostly, it is considered to arise from hematogenous metastasis of infection; spread of infection from neighbouring organs through the genito-urinary passages is of lesser importance (Konig, 1964; Jubb and Kennedy, 1970). Orchitis can be classified as acute or chronic inflammation of the testes.

Acute orchitis is characterised by a tense swelling and enlargement of the testes accompanied by pain and heat. It is either an infection via the vas deferens and epididymis, a combined epididimo-orchitis or metastatic lymphogenous suppressed as a result of the intense pain (Roberts, 1971).

Chronic orchitis may be an extension of acute orchitis. Acute orchitis may completely resolve or continue in a chronic form. It may be focal or diffuse, uni-or bilateral. Fibrosis may be seen grossly in some cases. There are varying degrees of degeneration and disappearance of the tubular cells, and the walls of the tubule become

thickened and hyalinised. It is generally characterised by interstitial infiltration of lymphocytes, plasma cells and sometimes eosinophils together with atrophy of the germinal epithelium (Kenney, 1971; Konig, 1964).

Another classification of orchitis was by Konig (1964). He regarded orchitis as: (1) interstitial (intertubular) (2) intratubular or (3) necrotic. Interstitial orchitis is characterised by mononuclear infiltration of intertubular stroma with subsequent fibrosis. Infiltrates can be perivascular and sometimes mononuclear infiltrates are observed adjacent to normal seminiferous tubules. Kenney (1971) stated that such foci may be infectious or immune in origin, the latter being in response to antigen "leaking" from a damaged tubule. Intratubular orchitis is characterised by obliterated seminiferous epithelium and replacement, centrally, by neutrophils and detritus. Mononuclear and giant cells are present (Konig, 1964; Kenney, 1971).

Necrotic orchitis is characteristic of brucellosis, but may result from other infections (Konig, 1964). In brucellosis, total necrosis of the testis with thickening and adherence of the tunics is a common outcome (Cohrs, 1967). On sectioning, necrotic areas are dry, yellow, and calcified. The histologic picture is one of coagulation necrosis bordered by fibrosis and mononuclear cell infiltration. It has been observed that abscessation, periorchitis, and fistula through the rectum may accompany this form of orchitis (Konig, 1964; Cohrs, 1967; Humphrey and Ladds, 1975).

Etiology:

Many investigators reported trauma and infections as the major causes of bovine orchitis (Lagerlof 1938; McEntee, 1970; Humphrey and Ladds, 1975). Many infective agents have been isolated from inflammed testes and or from their semen: Br. abortus, C. pyogenes, A.bovis, Nocardia sp,, Diplococcus sp., Mycobacterium bovis, Chlamydia sp., and Streptococcus sp. (Helmy et al., 1966; Konig, 1964; Boryczko, 1972; Bieberdorf, 1953; Awad, 1960). Pathological changes in bacterial orchitis are mostly non-specific. Lesions in brucellosis are characteristic (Lambert et al., 1963; Helmy et al., 1966).

A cytopathogenic enterovirus and other infective viruses have been isolated from the testes and seminal vesicles of bulls with orchitis (Florent et al., 1962; Bouters, 1964; Storz and Bates, 1973; Mare and Van Rensburg, 1961). These included: epi-vag, (IBR-IPV), G-UP VIRUS, and parainfluenza-3 virus (Deas et al., 1966). Logue (1974) reported that the testicular degeneration observed in a bull with MCF (malignant catarrhal fever) was due to pyrexia rather than a specific pathological effect of the virus on germinal tissues. Likewise, Kenney (1971) has emphasized the need to determine whether testicular degeneration in chlamydial infection is due to hyperthermia, epididymal lesions or other factors.

c. Pathology of epididymis

Pathological or functional disturbances of the epididymis include: infection of the epididymal duct and epididymitis, anomalies often causing sperm granulomas, tumors and functional or hormonal disturbances (Roberts, 1971; Konig, 1964; Humphrey and Ladds, 1975; Blom, 1959).

1. Epididymitis:

Inflammation of the epididymis is often seen as an acquired lesion caused by or may be secondary to the same agents causing orchitis. Pulsford et al., (1967) reported traumatic injuries as a possible cause of epididymitis in rams. According to Konig, (1964) epididymitis arises mainly by spread of infection in the genito-urinary passages, less frequently by hematogenous metastasis, and seldom by trauma.

The course of epididymitis is variable. The acute stage with edematous swelling may be followed by abscess formation, sometimes with perforation, periorchitis and peritonitis (Humphrey and Ladds, 1975). Konig (1964) reported spermatic granuloma after extravasation of spermatozoa. The histological picture has been summarised by Konig (1964), and Brno and Blom (1972). According to them the affected ducts contain fibrin, neutrophils, damaged spermatozoa, damaged epithelium, macrophages and giant cells, some of which contain spermatozoa ("spermiophagy"). The duct walls are infiltrated by neutrophiles and lymphocytes and a variable mononuclear interstitial reaction is apparent.

Konig (1964) and Ladds et al., (1973) reported on chronic epididymitis, which they said may not involve the duct and may follow the acute stage or arise from an insidious onset. Excessive interstitial fibrosis may result in nodular swellings of the epididymis and spermiostasis.

Infectious causes of epididymitis in the bull are the same as

those for orchitis. These include: Br. abortus, C. pyogenes, Chlamydia sp., C. pseudotuberculosis, M. bovigenitalium, and epivag (IBR-IPV). Of special interest is epididymitis-vaginitis (epi-vag), first described in Kenya - East Africa (Daubney et al., 1938) and later in South Africa (Van Rensburg, 1953; Mare and Van Rensburg, 1961). Lesions in this condition were most pronounced in the epididymis and manifested by initial soft swelling followed by marked fibrosis and enlargement. Associated lesions were abscess formation, tunic adhesions, ampulitis, seminal vesiculitis and testicular degeneration (Anderson et al., 1951).

2. Anomalies of epididymis:

These may either be congnital or hereditary and include:

a. Spermiostasis:- due to distension of aberrant efferent or epididymal duct/tubules with secondary formation of spermatic granulomas in the region of the epididymal head (Roberts, 1971; Blom and Christensen, 1960). The resulting spermatoceles, in time, may lead to the back pressure effecting testicular degeneration, though the size and consistency of the testis may remain fairly normal due to the accumulation of testicular fluids (McEntee, 1970).

b. Segmental aplasia of the Wolfian duct: Already discussed under congenital lesions.

There is evidence that a change in the environment of spermatozoa in the epididymis due to probable changes in the epididymal epithelium may result in reduction or absence of motility or akinesia or necrospermia associated with bent or looped tails (Roberts, 1971).

Gustafsson, (1965) found a changed composition of the epididymal plasma in a bull in which sperm akinesia and certain sperm tail abnormalities and detachment of sperm heads appeared to be of epididymal origin, but the mechanism, underlying the presumed epididymal dysfunctions has not been fully elucidated (Gustafsson, 1966).

c. Diseases of the accessory glands

(1) Seminal vesiculitis:

Seminal vesiculitis is the most common condition found affecting the seminal vesicles of bulls (Roberts, 1971). Many investigators (Roberts, 1971; Ball et al., 1968) observed that the disease affects males of all ages, but it is more common in young and old beef bulls than in mature, middle-aged bulls. The reported incidence of bovine seminal vesiculitis is variable. In a study of slaughtered bulls in Denmark, the incidence was 0.85%, but among 7359 western beef bulls, 2.5% were affected (Carroll et al., 1963). In New York State, 4.7% of 343 bulls were affected (McEntee, 1962). Blom and Christensen (1956) reported the disease in 17 of 2,000 slaughtered bulls. Ball et al., (1968) reported the disease in 181 of 7359 bulls examined for breeding soundness.

Seminal vesiculitis is clinically diagnosed by rectal palpation of the vesicular glands and concurrent examination of the semen. On rectal examination seminal vesiculitis may be found to be characterised by: irregular enlargement of the gland, fibrosis, induration, loss of lobulations, or lack of well-defined lobulations, peritoneal

adhesions, fluctuation and abscessation (Ball et al., 1968; Roberts, 1971). Semen from affected bulls usually contain leukocytes which must be differentiated from spermatogenic cells from the germinal epithelium (Galloway, 1964).

The histopathologic lesions have been described by many authors (Galloway, 1964; Roberts, 1971). They observed the occurrence of both chronic interstitial inflammatory reaction and degenerative reaction with mild interstitial inflammation. Both focal or diffuse suppurative and nonsuppurative interstitial inflammation with fibrosis of the interstitium and subepithelial mononuclear infiltrations and proteinaceous fluid in the alveolar lumen have been observed (McEntee, 1962; Galloway, 1964).

Many bacterial pathogenes have been isolated from affected bovine seminal vesicles. C. pyogenes (most commonly) and others like Br. abortus, Actinobacillus actinoides, E. coli, have been isolated (Galloway, 1964). Viruses have been incriminated as a cause of bovine seminal vesiculitis (Bouters, 1964). Roberts (1971) observed that bulls with a seminal vesiculitis will, not infrequently, have another foci of infection in the testis, epididymis and/or ampulla.

In young bulls, infection may spread by homosexuality. Many young bulls with seminal vesiculitis syndrome (catarrhal or degenerative) overcome the infection spontaneously in a few months (Roberts, 1971).

The role of IBR-IPV (infectious bovine rhinotracheitis - infectious pustular vulvovaginitis) or epi-vag is controversial (Humphrey and Ladds, 1975). The difference may be one of strain virulence.

(2) Prostatitis:

Clinical prostatitis in the bull is rare and may be caused by Brucella abortus. Subclinical infection may be more common and due to a variety of bacteria and viruses as described under seminal vesiculitis.

d. Conditions of the tunica vaginalis:

It has been observed that adhesions between the parietal and visceral layers of the tunica vaginalis are common. Galloway (1961) observed that fine, fibrous adhesions confined to the cauda epididymal region and found mostly in young bulls were of no pathological significance. Severe extensive fibrous adhesions resulting from trauma may interfere with testicular function by interfering with testicular motility in the scrotum. Ladds et al., (1973) and Galloway (1961) found increased occurrence of adhesions with advancing age.

Non-specific periorchitis, initially fibrinous and later fibrous, with adhesions, may be responsible for thermal testicular degeneration (Galloway, 1961). Konig (1964) cited some causes of periorchitis: extension from orchitis, epididymitis, or from exogenous trauma. Hydrocele, chylocele, and hematocele involving the tunica vaginalis may occur (Jubb and Kennedy, 1970). Blom and Christensen (1972) reported testicular herniation as a sequel to rupture of the tunica vaginalis.

e. The scrotum:

The scrotum serves in part as a necessary thermoregulatory mechanism so that the temperature of the testes and epididymis is lower than that of the body. It accomplishes this by having a dartos and cremaster muscle which contracts and draws the testes next to the abdominal wall for warmth and then relaxes letting the testes descend for cooling (Roberts, 1971). According to Faulkner (1969) this mechanism applies only to temperate breeds of bulls within temperate environment. A second mechanism is the so-called pampiniform plexus; a system of looped veins and arteries that lies on the surface of the epididymis. This cools the blood entering the testes about 4^{o}.C, and warms venous blood as it approaches the central circulation (Faulkner, 1969; Roberts, 1971). It is believed that in those species that have less pendulous testes located high against the abdominal wall such as stallion and boar the pampiniform plexus plays a major role in cooling the testes (Eagleman, 1976).

Any deleterious effect on the scrotum may tend to affect the testis and hence spermatogenesis. Ectoparasitic infestation of the scrotum has been reported as a cause of severe scrotal dermatitis (Watson, 1964). In Nigeria, severe tick infestation and streptotrichosis of the scrotum have been observed to cause orchitis and testicular degeneration (Kumi-Diaka, 1976.) Severe parasitism is believed to cause dermatitis associated with thickening of the scrotal skin and resultant testicular degeneration (Pols, 1960 ;

Humphrey and Ladds, 1975). Traumatic injuries of the scrotum from thorns and spikes are important in Nigeria and other tropical countries where nomadic husbandry practice predominates.

f. Miscellaneous factors:

Miscellaneous male factors resulting in failure of fertilization, conception or reduced fertility are complex. Lacerations of the ventral portion of glans penis with a fistulous opening may result in semen being deposited in the middle portion of vagina and not sprayed into the cranial portion or over the cervix at ejaculation (Roberts, 1971).

Roberts (1971) is of the opinion that environment is probably the most important factor in conception rates of bulls. Some bulls used artificially have up to 90 percent conception in some herds, and as low as 20 percent conceptions in others. Lanman (1968) reported that spermatozoa, aged in the epididymis for weeks, aged by storing in liquid semen at 4^{o} to $5^{o}C$ for days or in the frozen state in bulls for many months, caused decreased fertilization rates and increased early embryonic or fetal deaths.

A. Diseases of penis and prepuce are common causes of inability or difficulty in copulating and frequently result in reduced libido.

1. Phallocampsis - or deviation of the penis is a common cause of copulatory difficulties and reduced libido in bulls (Roberts, 1971). The spiral or corkscrew type of deviation of the penis is most commonly observed in young bulls.

According to Roberts (1971), penile deviation in bulls may be due to congenital persistence of the ventral frenulum. Ashdown and Combs (1967) suggested that contraction of the retractor muscles of the sheath during erection might be a causative factor in spiralling of the penis. They reported also that up to 30% of Hereford bulls have a normal straight penis on intromission but have a curled or twisted penis on dismounting. They observed that in affected bulls, premature full erection occurs prior to intromission resulting in "corkscrewing" that prevents the completion of coitus.

Other much less common types of penile deviations are the ventral or "rainbow" and the mild S-shaped curvatures. The latter is of little importance while the former prevents normal copulation as the more erect the penis becomes the greater the ventral curvature (Roberts, 1971; Ashdown and Combs, 1967;).

2. Adhesions of penis and prepuce

Adhesions in the region of the sigmoid flexure often prevents obliteration of the S-curve and protrusion of the penis during erection (Roberts, 1971; Farquharson, 1952). Adhesions of the caudal portion of the prepuce in the region of the fornix results in a more severe phimosis. Adhesions may be secondary to lacerations of the prepuce especially in young bulls and in older, vigorous bulls. Any possible infections following the injury may produce abscesses and/or adhesions of the surrounding structures preventing the free movement of the prepuce and penis at erection (Farquharson, 1952; Roberts, 1971). A ruptured penis with secondary hematoma is usually observed in bulls. It occurs most commonly in bulls, with a strong sex drive breeding cows on pasture (Farquharson, 1952; Gibbons, 1956). According to Gibbons (1956) penile hematomas are due to a sudden angulation or bending of the erect penis at service and not only due to high blood pressure in the corpus cavernosum.

Other common causes of preputial and penile adhesions are horn injuries in bulls and from injections of local anaesthetics to block the dorsal nerve of the penis (Gibbons, 1952; Farquharson, 1956).

3. Tumors of penis and prepuce

Transmissible fibropapilloma is the only significant tumor of the penis in the bull (McEntee 1970). Grossly, these are single or multiple, firm, cauliflower-like growths. Young beef bulls are most

commonly affected as they, when housed together, mount each other and injure the prepuce affording an invasion site for the virus (McEntee, 1958; Roberts, 1971).

There is definite evidence that this tumor and cutaneous papillomas, or warts, in cattle, are similar and caused by the same agent (McEntee, 1958). McEntee (1958) considered the etiologic agent to be a virus. He reported that the tumor may cause phimosis or paraphimosis or prevent normal intromission.

4. Phimosis or stenosis

Stenosis of the preputial orifice may prevent normal protrusion of the penis. The condition is usually acquired due to injuries, wounds and infections. In cattle with pendulous sheaths, the preputial orifice may be stepped on causing severe contusion and swelling (Cardwell, 1961).

Chronic prolapse of the prepuce is a very common cause of posthitis and phimosis. It is common in Bos indicus cattle and less so in Bos taurus (Donaldson and Aubrey 1960). Roberts (1971) reported that, chronic preputial prolapse is probably an inherited trait associated with a pendulous sheath, a large preputial orifice and a relaxed preputial membrane and penis. The prolapsed sheath becomes dry, traumatized, lacerated, edematous, swollen, and fibrotic, causing phimosis. He reported screworm infestation as resulting in phimosis.

5. Paraphimosis:-

Paraphimosis results in edema, swelling and balanoposthitis. It may occur following erection of the penis through a stenotic preputial orifice or ring caused by a congenital or acquired stricture or by tumors. Spinal diseases or trauma may cause penile paralysis and paraphimosis (Wheat, 1951; Roberts, 1971). Roberts (1971) has reported a case of paraphimosis due to resection of the retractor penis muscle in bulls with pendulant sheaths. Chronic paralysis and prolapse/ paraphimosis could lead to necrosis of the penis.

6. Balanoposthitis:-

Balanoposthitis is common in the bull due to infectious or non-infectious causes. It may produce stenosis of the preputial ring, adhesions of the penis and prepuce to each other. Infectious agents commonly observed in the male's preputial cavity include: E.coli, C. pyogenes, Pseudomonas aeruginosa, Proteus sp., Aspergillus, Mucor, Absidia, Mycoplasma, Trichomonas fetus. IBR-IPV virus and other pathogenic and saprophytic organisms (Bakos et al., 1962; Bane, 1964; Beveridge and Johnstone, 1953; Bruner and Gillespie 1966). Roberts (1971) reported that no obvious balanoposthitis generally accompanies the presence of the above organisms in the preputial cavity with the possible exception of M. tuberculosis and the acute form of IBR-IPV virus infections. Williams (1943) described tuberculosis of the sheath, penis, prepuce, and penile lymph glands of bulls. This

avenue of invasion in the bull is invited by tuberculosis of the female genital tract. He described the gross lesions as characterised by enlarged, granulomatous bleeding lesions of the glans penis, adhesions of the penis, prepuce and sigmoid flexure with secondary phimosis. He also described several cases of actinomycosis affecting the sheath and glans penis, with lesions resembling those of tuberculosis.

Trauma, abrasions, lacerations of the prepuce or glans penis usually result in the introduction of the above organisms resulting in swelling, inflammation, pain and discharge. This is common in young bulls (Bane, 1964).

B. Miscellaneous:

Miscellaneous causes of loss of libido or inability to copulate include: (1) Hernias - umbilical and ventral hernias as well as deep pendulous abdomen may interfere or prevent normal copulation by affecting entry of penis into vagina (Oehme, 1968; Marsh, 1961) (2) Premature erection - may occur in certain bulls and prevent or interfere with normal intromission (Williams, 1943; Vandersluis, 1953). (3) Loss of sensory innervation of the glans penis prevents natural intromission and the "Thrust" reflex necessary for ejaculation and leads to pronounced decline in sex drive (Vandersluis, 1953; Aronson and Cooper, 1966). (4) Urinary calculi - occassionally a cause of acute pain, obstruction and rupture of the urethra and cause reluctance to copulate and inability to ejaculate. Vandersluis (1953) reported

urinary calculi in a bull with inability to copulate and exhibiting impotentia coeundi. (5) Others - pain caused by infection of the genital organs or peritoneum may be a cause of impotency or refusal to copulate (Williams, 1943). Roberts (1971) described several bulls that were slow or refused to mount due to severe seminovesiculitis or acute brucella orchitis.

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SECTION II

A clinico-pathologic and bacteriologic study of the testes and epididymides of indigenous bulls in Northern Nigeria

Summary

Clinical and post-mortem lesions of the reproductive organs of 4730 indigenous bulls in Northern Nigeria were studied. The bulls, belonging to the nomadic Fulani herdsmen ranged from 3 weeks to 10 years old.

Gross lesions observed were; testicular atrophy/degeneration (0.59%), testicular hypoplasia (0.66%), orchitis (0.44%), cryptorchidism (1.29%), epididymitis (0.08%) and spermatic granuloma (0.15%). Scrotal dermatophilosis of varying degrees of severity was observed in 204 bulls (4.3%) with most cases occurring during the rainy season. This condition did not appear to significantly affect spermatogenesis.

Histopathologic lesions and their prevalence were: testicular degeneration, 18.60%; hypoplasia, 5.30%; orchitis, 8.10%; and epididymitis, 1.40%. Testicular degeneration was observed more commonly in the middle-aged and older bulls (6-10 years). Bacterial organisms associated with some of the lesions in the present study were; Corynebacterium pyogenes, Staphylococcus spp, Streptococcus spp. and Brucella abortus. In addition to hereditary factors, age, trauma and trypanosomiasis were also identified as significant factors in the etiology of testicular and epididymal pathology.

All the gross and histopathological lesions, except those of streptotrichosis, occurred randomly throughout the year without regard to season and with no breed disposition.

Introduction

In the study of the pathology of reproduction, the male has been accorded a minor role (Zemjanis, 1974). The advent of artificial insemination and subsequent studies of bovine semen have demonstrated conclusively that there are varying degrees of infertility in bulls. Observations in many parts of the world show that testicular (and epididymal) pathology is a contributing factor to economic losses in livestock industry and can be a major cause of bull wastage (Lagerlof, 1938; McEntee, 1970). The low reproductive efficiency of indigenous cattle in Nigeria is considered to be due to nutrition, diseases, management and their interaction (Kumi-Diaka, 1976). There is little information on the reproductive performance of bulls and their role in bovine infertility problems has not been fully investigated in Nigeria.

The objective of this investigation is to attempt to determine some of the causes and incidence of testicular and epididymal pathology of the bulls indigenous to Northern Nigeria.

Materials and Methods

The scrotum and its contents from 4730 bulls (Rahaji, White Fulani or Bunaji and Sokoto Gudali breeds) belonging to nomadic Fulanis were collected from abattoirs in some towns in Kaduna and Kano States of Nigeria. Specimens were collected soon after slaughter from 20 slaughter slabs and 3 abattoirs from January to December (1977-1979). Prior to slaughter, bulls were examined and grouped into age determined by the eruption or wearing pattern of the teeth. The age groups comprised of young (0-2 years), middle age (2.5-5 years) and old (5.5 years and over).

The testes were removed from the scrotum and dissected from the tunica vaginalis. The spermatic cord was severed 0.5cm above the dorsal pole of each testis. Each testis was carefully examined for gross lesions such as adhesions, hematoma degeneration, atrophy/hypoplasia, inflammation. The shape and consistency, described as normal, soft , or hard and fibrotic were recorded for each testis.

The epididymis was dissected from each testis and examined for gross lesions such as inflammation, segmental aplasia, spermatic granuloma , atrophy.

Histopathological study:

Small sections (about 0.5 - 1.0cm thick, and 1.0cm wide) were cut from each testis, and fixed in Bouin's solution for 72 hours. Sections were trimmed and processed in a Tissuematon*, embedded in paraffin, cut at 4 - 6 microns, mounted and stained with hematoxylin and eosin (H & E), using Gurr's mounting media with cover slip. Other stains employed included; Periodic Acid-Schiff (P.A.S.), Brown and Brenn (B. & B), Masson's Trichrome (MT) and Van Giesson (V.G.). Epididymal sections were similarly processed and stained for histopathological examination. The histopathological study was carried out in 2000 out of the 4730 bulls.

For histopathological examination, slides were initially screened under low-power (x100) magnification, and detailed examinations were done under high dry power (x450) and oil immersion (x1000). The presence and conditions of the germinal epithelial and sertoli cells were studied and any deviations from the normal noted. The interstitial tissues were examined and any lesions and their frequency recorded. Degrees of testicular degeneration were scored as shown in Table 1. The epididymal sections were similarly studied and any histopathological changes observed were recorded.

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* Fisher Scientific Co. Chicago, Illinois, USA.

Bacteriological study:

Sterile swabs were taken from all cases of gross testicular and epididymal lesions and subjected to culture and identification of microorganisms. Samples were streaked on blood agar and McConkey plates and incubated aerobically at 37°C for 48 hours. Gram stain was employed to study the type of microorganisms present. For isolation of brucella organisms, sterile swabs were inoculated into Brodie and Sinton liquid medium (BSL), the latter incubated at 37°C in 5 - 10% CO^2 for 4 days. After the 4 days, 0.2ml of the BSL was inoculated into Farrell solid medium (FSM), incubated at 37°C in 5-10% CO^2 for 4 days. Examination of the colonies on FSM, isolation, and further characterization of Brucella spp was done according to the methods of Brodie and Sinton (1975) and Morgan et al. (1978).

Serological study:

Blood samples were taken from the jugular veins of bulls showing clinical evidence of testicular abnormality (palpation of scrotum) into clean dry tubes, allowed to clot and the serum filtered off for agglutination test to detect brucella infections. Serum agglutination test (SAT) was used as recommended by the World Health Organization (WHO, 1953). Serum was diluted 1:12.5, 1:25, 1:50. etc.. The sensitivity of the serum was such as to indicate 25, 50, 100, etc. I.U. of antibodies per ml. respectively if positive. Readings were taken at 50 per cent agglutination.

Hematological study:

Blood samples were taken as previously described. Whole blood was used to determine the packed cell volume (PCV), hemoglobin concentration (Hg), total white blood cells and the differential counts by the method of Coles (1974).

Blood samples were also examined for blood parasites.

Results

The incidence and type of scrotal abnormalities detected are shown in Tables 2-5.

Gross findings:

Lesions observed in the 4730 bulls examined were: testicular atrophy/degeneration, 0.59% (28 bulls): testicular hypoplasia, 0.66% (31 bulls): orchitis, 0.44% (21 bulls); cryptorchidism, 1.29% (61 bulls); epididymitis, 0.08% (4 bulls) and spermatic granuloma, 0.15% (7 bulls), all involving the head of the epididymis. Figure 1 shows gross testicular hypoplasia.

The hypoplastic testes were without regard to age but the spermatic granuloma were observed in bulls between the ages of 3 to 7 years. The orchitis was without regard to age, and trauma

Table 1*: Grading of testicular degeneration

Lesion Type	Grade	Distribution of Lesion
Testicular epithelial degeneration	Mild	20 affected tubules or less/LPF*
	Moderate	21-50 affected tubules/LPF*
	Severe	Most tubules affected (50+)

+ LPF = Low power field (x100).

* Leslie, B.; Stuart, Y and Carroll, E.J. (1968): Am. J. Vet. Res. 29: 1173-1181.

was observed to be the initial/primary cause in most cases (16 bulls) as indicated by the presence of old wounds and scars. Three cases were superimposed with severe scrotal dermatophilosis.

Scrotal dermatophilosis of varying degrees of severity, and three of which probably resulted in testicular atrophy, was observed in 204 bulls. There was no age incidence and most of the cases (62.2%) occurred during the rainy season. About 37.8% occurred during the dry season. The difference in the incidence between the two seasons was highly significant ($P < .01$).

Thickening of the tunica vaginalis and adhesions between it and the adjacent tissues was observed in 14 bulls; one of which was in association with chronic epididymitis.

Conditions such as anorchia, neoplasms, varicocele, hydrocele and scrotal hernia were not observed.

Histopathologic lesions:

The results of the findings of the scrotal contents of 2000 bulls are summarised in Table 4. The type of lesions and their prevalence were: testicular degeneration, 18.60% (372 bulls); hypoplasia, 5.30% (105 bulls); orchitis, 8.10% (163 bulls); epididymitis, 1.4% (27 bulls).

Testicular hypoplasia of varying degrees of severity are shown in Figs 2 and 3.

Figs 4 and 5 show varying degrees of testicular degeneration (pyknosis Most of the degeneration was mild and moderate with the highest incidence in the older bulls (6-10 years). Testicular degeneration occurred throughout the year with little or no difference in the season or breed of bulls involved. Degenerative changes were frequent but often differed in degree in different sections of the testis.

Bacteriologic findings:

Brucella abortus organisms were isolated in 27 bulls showing various signs of gross testicular and epididymal pathology, predominantly orchitis and degeneration. Corynebacterium pyogenes, Staphylococcus spp and Streptococcus spp were isolated from 11 cases of orchitis and periorchitis, 8 cases of testicular degeneration, one case of spermatic granuloma and 2 cases of epididymitis. Amongst the latter organisms isolated, C. pyogenes was most predominant.

Hematologic findings:

The P.C.V., hemoglobin and blood cell counts were all within normal range in all the bulls examined. Parasitological examination of the blood revealed severe infections of Babesia bovis in 18 bulls, severe infection of Trypanosome vivax in four bulls and a mixed infection of T. vivax and B. bovis in two other bulls. All the bulls

infected were concurrently showing clinical signs of various testicular and epididymal lesions of varying degrees of severity.

Serologic findings:

Thirty-one bulls were serologically positive for brucella infection. B. abortus was isolated from the testes and epididymis of 27 out of the 31 bulls serologically positive for brucellosis. All the bulls infected with brucellosis were showing clinical signs of testicular and epididymal lesions of varying degrees of severity.

Table 2: Gross pathologic conditions of the scrotal contents of indigenous bulls in Northern Nigeria

Location of Abattoir	No. and breed of bulls*	Cryptorchidism	Testicular atrophy	Testicular hypoplasia	Orchitis	Spermatic granuloma	Epididymitis	Total No. of cases
Kano	2400R	33	14	17	14	1	3	82
	1200WF	15	3	2	3	1	0	24
Danbatta	78WF	1	0	2	0	0	0	3
Bunkure	36R	2	3	0	1	0	0	6
Kadawa	6SG	0	0	0	2	1	0	3
Tiga	10SG	0	0	0	1	0	0	1
Zaria	350WF	4	5	1	0	3	0	13
Samaru	87WF	1	2	1	0	0	1	5
Funtua	143WF	0	0	0	0	0	0	0
Bakore	3SG	0	1	0	0	1	0	2
Kaduna	417WF	5	0	8	0	0	0	13
Total	4730	61	28	31	21	7	4	152
% age		1.30	0.59	0.66	0.44	0.15	0.08	3.21

* Breeds: R.= Rahaji; WF = White Fulani; S.G. = Sokoto Gudali

Table 3: Gross pathologic changes in the testes of 4730 indigenous bulls in Northern Nigeria.

Lesion	Distribution of lesions					
	Affected bulls		Unilateral		Bilateral	
	(No.)	(%)	(No.)	(%) of affected bulls	(No.)	(%) of affected bulls
Testicular degeneration	28	0.59	20	71.43	8	28.57
Testicular hypoplasia	31	0.66	23	74.19	8	25.81
Orchitis	21	0.44	15	71.43	6	28.57
Cryptorchidism	61	1.29	61	100.00	0	0.00
Epididymitis	4	0.08	4	100.00	0	0.00
Spermatic granuloma	7	0.15	7	100.00	0	0.00
Total	152	3.21	130	- -	22	- -

Table 4: Histopathologic findings in the testes of 667 out of 2000 indigenous bulls in Northern Nigeria.

	Affected bulls		Distribution of lesions			
			Unilateral		Bilateral	
	(No.)	(%) of total bulls	(No.)	(%) of affected bulls	(No.)	(%) of affected bulls
Testicular degeneration	372	18.60	102	24.40	270	72.60
Orchitis	163	8.10	100	61.30	63	38.70
Testicular hypoplasia	105	5.30	95	90.50	10	9.50
Epididymitis	27	1.40	19	70.40	8	29.60
Total	667	33.40	316	- -	351	- -



Fig 1: Unilateral testicular hypoplasia; Left testis approximately $\frac{1}{3}$ right testis.

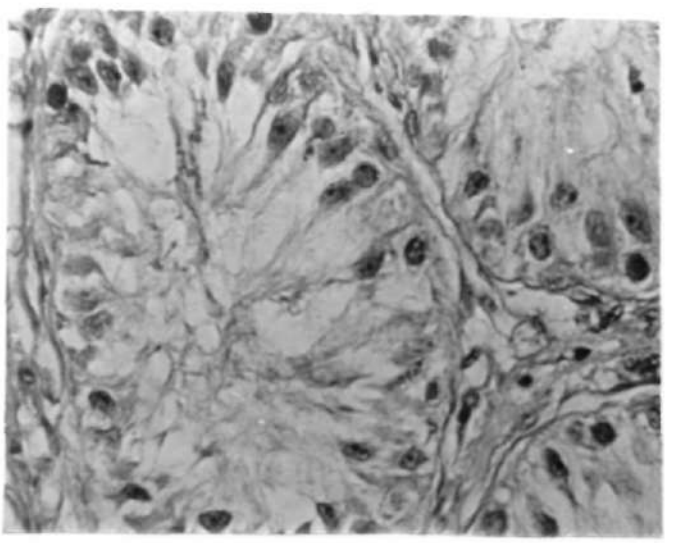


Fig 2. Testicular hypoplasia involving almost 98% of the seminiferous tubules. Note arrest of spermatogenesis at primary spermatocyte stage. x 450

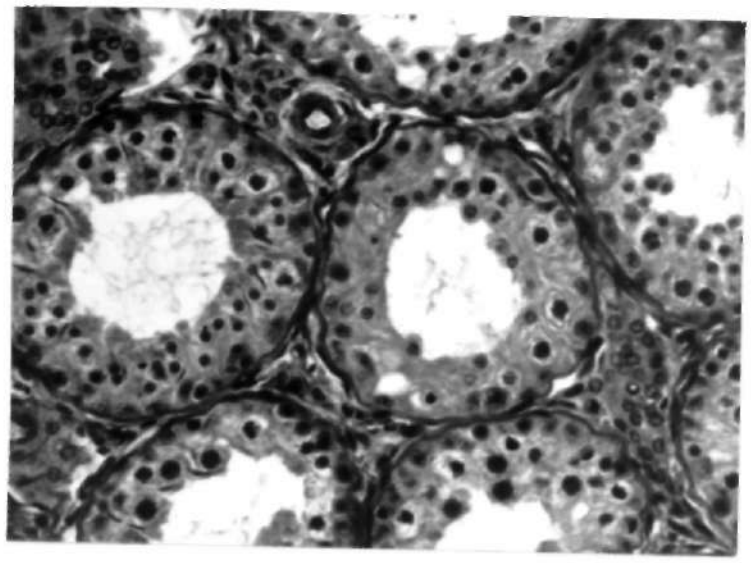


Fig 3: Severe testicular hypoplasia. Note scantiness of germinal cells and partial obliteration of lumen. x 450

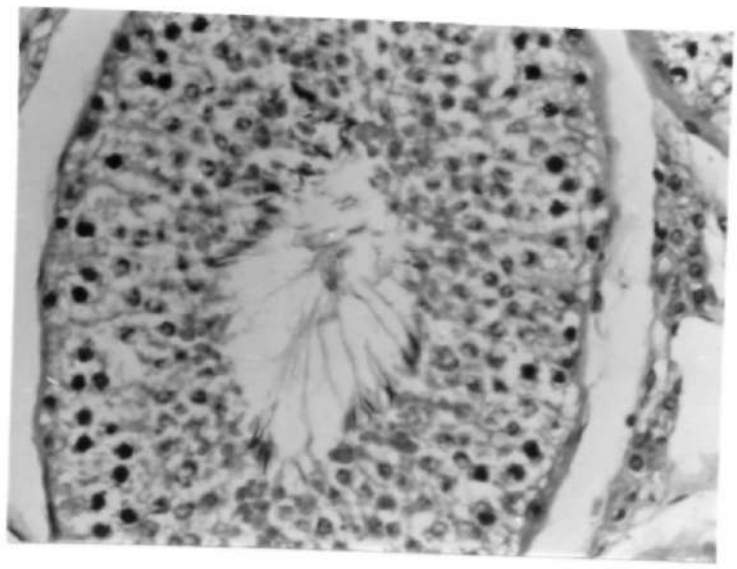


Fig 4: Moderate testicular degeneration. Note pyknosis and karyorrhexis. x 450

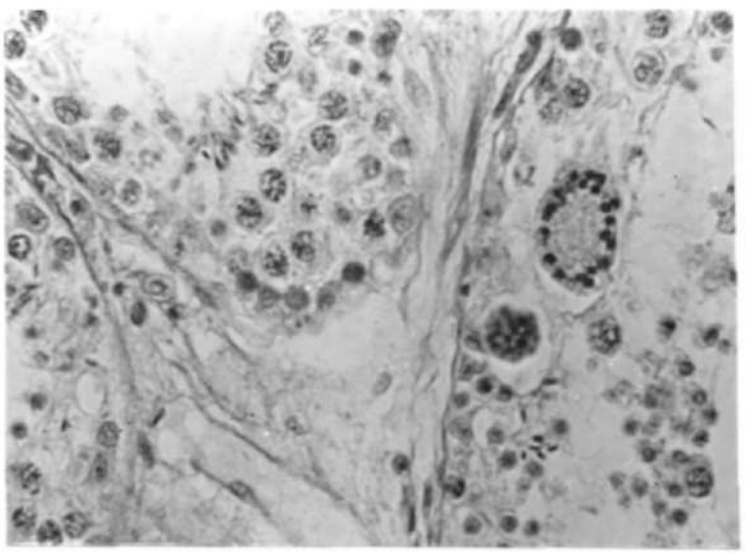


Fig 5: Severe testicular degeneration. Note giant cell, desquamation and sloughing of germinal cells. x 450

Discussion

The incidence of scrotal content abnormalities was lower than reported elsewhere by other investigators (Lagerlof, 1938; Haq, 1949; Vander-Sluis, 1953; Barker, 1956; Carroll et al., 1963). This might be due to the larger number and the composition of bulls studied, their age and physical conditions, criteria used to classify the various pathologic lesions, breed differences, and environmental factors and their interactions. The cited investigators used smaller number of bulls.

The incidence of clinical orchitis (0.44%) was far lower than the 24% reported in Sweden by Largerlof (1938) and the 25% reported in Canada by Barker (1956). Gibbons (1957) reported that orchitis is usually unilateral and uncommon if trauma and degenerative changes are excluded. The findings in this survey are in conformity with Gibbons observation. Trauma was observed to be the primary cause of most of the orchitis seen in this study. This may partly be due to the type of husbandary practice by the local herdsmen which subjects their cattle to injuries. The isolation of C.pyogenes organisms in some cases is in accord with the results of other investigators (Gibbons, 1957; Konig, 1964; Jubb and Kennedy, 1970). Although the mere presence of the organism is no evidence of cause and effect, C. pyogenes has been implicated as one of the etiological agents of orchitis, testicular

degeneration and seminovesiculitis (Jubb and Kennedy, 1970; Roberts, 1971).

Testicular degeneration was the most common histopathologic lesion observed. The gross and microscopic lesions of the various degrees of severity of degeneration were in agreement with other reports (Lagerlof, 1938; Knudsen, 1954; Jubb and Kennedy, 1970; Watt, 1974). The incidence of 18.6% was significant. In a similar study, a monthly incidence of 20% testicular degeneration was observed, with no breed difference or seasonal pattern (Kumi-Diaka, 1976). Bulls in the tropics are considered to be under thermal stress especially during the hot season, and as such are expected to produce poor quality semen as a result of abnormal spermatogenesis (Casady et al., 1953; Donaldson, 1963; Skinner and Louw, 1966). The present study indicates that testicular pathologic lesions such as degeneration, have no seasonal pattern. In a previous report, spermatogenesis was reported to be active throughout the year, with no significant retrogression during the hot season (Kumi-Diaka, 1976). The present result which indicates that testicular degeneration is most common in middle-aged and older bulls (2.5 - 10 years) is in agreement with the findings of Hooker (1944); Knudsen (1954); Galloway (1961); Collins et al. (1969) and McEntee (1970). It is generally accepted that advancing age is associated with permanent degenerative changes in the testes of man and domestic animals (Lancester, 1956; Rao, 1971;). The Fulani herdsmen do not practice

culling, and excessive use of bulls, especially when they are aged, may predispose to testicular degeneration and contribute to infertility problems.

Infectious, nutritional, hormonal and other factors as possible causes of testicular degeneration have been reported (Lagerlof, 1938; Moore, 1948; Gibbons, 1957; Roberts, 1971;). The isolation of brucella organisms in some cases of testicular degeneration and orchitis is significant. Orchitis due to brucellosis in bulls has been reported (Jubb and Kennedy, 1970; Roberts, 1971). This leads to infertility and abortion as the organisms are venereally transmitted to the cows (Roberts, 1971). Besides, orchitis due to brucella suppresses spermatogenesis.

In this study, Trypanosome vivax was seen in some bulls with severe testicular degeneration and/or orchitis. It is probable that the changes in body temperature associated with T. vivax infection may contribute to injury of germinal epithelial cells as has been reported by Payne (1956) in mice. Babesiosis as a cause of testicular degeneration and/or orchitis has not been reported. However the pyrexia associated with this disease could have adverse effect on the germinal epithelial cells as has been reported in tick-borne fever in rams (Watson, 1964).

The incidence of 0.66% gross testicular hypoplasia was lower

than those reported in the literature (Lagerlof, 1934; Carroll et al., 1963). Some difficulties were experienced in distinguishing hypoplasia from atrophy, and this made the assessment of the incidence of the former difficult. Bishop (1970) and Jubb and Kennedy (1970) considered that because of the variable expressions of hypoplasia, differentiation between it and atrophy/degeneration is either difficult or not valid.

Among the cases of testicular hypoplasia seen, there was no evidence of trauma and no bacterial organisms isolated upon culture. The hereditary nature of the condition has been established (Lagerlof, 1934, 1938; Errikson, 1943; Laing and Young, 1956; Gibbons, 1957;).

All the cases of cryptorchidism observed were unilateral and inguinal, with an incidence of 1.29%. This is in conformity with the observations of Blom and Christensen (1947), Bishop (1972) and Galloway (1974). The cause of bovine cryptorchidism was not established in the present study but its hereditary nature is well established (Couvreux, 1943; Bishop, 1972). No age incidence was observed and this finding is in agreement with Gibbon's report (1957).

The prevalence and manifestations of epididymitis were in agreement with the observations of Watt (1974) and Doolette (1953). The bacteria isolated in some of the cases in the study reported here of epididymitis and periorchitis have also been reported as among the

infectious agents causing the conditions (Gunn et al., 1942; Roberts 1971 and Edgar, 1959). The failure to isolate bacterial organisms from some of the orchitis and epididymitis leads to the suspicion that trauma might be the cause. However this could also indicate old and sterile lesions.

The immediate cause of the clinical cases of spermatic granuloma could not be ascertained. In one case, the appearance of the affected testis/epididymis indicated traumatic etiology as reported by Turnbull and McKay (1973).

The scrotal streptotrichosis observed did not appear to affect testicular function. However, in the most extreme, chronic cases, there was thickening of the scrotum and moderate microscopic testicular degeneration and inflammation. But streptotrichosis, as the primary cause of the degeneration, could not be substantiated in the present study. However it is probable that the thickening could interfere with the thermoregulatory activity of the scrotum.

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Section III

Quantitative estimation of spermatogenesis in normal and grossly abnormal testes of indigenous bulls sent for slaughter

Summary

Spermatogenesis in normal and grossly abnormal testes, and the significance of sertoli cell index (SCI) as a measure of testicular degeneration, was studied in 264 indigenous bulls.

The means of the testicular weight and dimensions and the seminiferous tubular diameter were lower than reported for the Bos taurus breeds. The mean seminiferous tubular diameter was smaller than reported for the Bos taurus, though within the range. Inter-month and intra-month variations were highly significant ($P < .01$).

A mean percentage of 77.6% spermatogenesis was reported for the year. Active spermatogenesis was observed throughout the year, with a peak during the rainy season and extending towards the pre-dry season. This was consistent with previous reports. Severe testicular degeneration was found to influence spermatogenesis. This was reflected in the decrease in SCI.

Mild degeneration had no significant effect on spermatogenesis, and did not significantly change the basic/normal ratio of SCI.

SCI as a measure of spermatogenesis is complicated, since the latter is influenced by such factors as age of the male and level of nutrition, among other things.

Introduction

The world human population depends on animals as a major source of protein. Daily protein consumption per capita in most developing countries including Nigeria is low. With the advent of artificial insemination, bull fertility has become more and more important, and extensive studies of the histological elements of the bovine testes have been made by various investigators (Lagerlof, 1939; Moore, 1948; Laing and Young, 1956; Humphrey and Ladds, 1975). However, a thorough study of the histological picture of the testes of the indigenous bulls in Nigeria has not been undertaken.

The objective of the investigation was to study spermatogenesis in grossly normal and abnormal testes of indigenous bulls in Northern Nigeria.

Materials and Methods

Sources of the testes:

The scrotum and its contents from 264 Bunaji (White Fulani) bulls were collected for a period of 12 months, at a rate of 22 sampled bulls

a month, from various abattoirs in Kaduna and Kano states of Nigeria. The bulls, varying in age from 2 to 7 years were in good physical conditions at the time of slaughter. Pre-slaughter clinical examination included palpation of the scrotal contents to ascertain the presence or absence of gross pathologic lesions, such as orchitis, testicular degeneration and epididymitis.

The testes were removed soon after the animals were slaughtered, examined and immediately dissected and weighed on a torsion balance. The dimensions- length and width, were then taken. Histological sections were prepared as already described in section II of this investigation, for both the normal and grossly abnormal testes.

Microscopic studies:

Slides were first screened under low power (x100) magnification and later studied in detail under high dry (x450) and oil immersion (x1000). Five different points on each slide were selected (as described in section II) and 10 transversely cut seminiferous tubules (50 tubules per slide), were examined. Tubular diameter was taken with an eyepiece micrometer scale which was calibrated with a stage micrometer at x450. The diameter, measured from one basement membrane to another basement membrane of the seminiferous tubules represented in every case, the average measurements of 50 tubules, taken at the

various points. Only tubules with perfectly clear transverse sections were measured. The germinal cells and sertoli cells within the selected tubules were counted and the process of spermatogenesis studied. Spermatogenesis was assessed by examining sections for: (1) preservation of architecture of tubules and (2) number of tubules containing spermatids per low power field (x100) expressed as a percentage. Sertoli cells index (SCI) was determined for both normal and abnormal spermatogenesis and the results compared. The SCI was obtained as follows: a differential count of the germ cells and sertoli cells was made in the 50 selected seminiferous tubules per slide. The total number of counted germ cells in the cross sections of the tubules was divided by the total number of sertoli cells found in the cross sections of tubules. The resultant values were referred to as SCI (Kumi-Diaka and Dennis, 1978). The degree of testicular degeneration was related to SCI.

The results of all the findings were subjected to statistical analysis of variance.

Results

One hundred and fourteen of the selected bulls had grossly normal testes, 10 bulls had unilateral hypoplasia/atrophy and 140 bulls had

gross testicular degeneration and/or orchitis as revealed by size, consistency, color, sensitivity to touch and post-mortem findings such as adhesions between the tunics and the testes.

Gross dimensions:

Table 1 shows the testicular biometric data. The average monthly lengths of the right and left testes were 11.86 ± 1.30cm and 11.53 ± 1.40cm respectively and the average monthly diameters of the right and left testes were 4.90 ± 0.60 cm and 5.03 ± 0.50 cm respectively. The inter-month and intra-month differences in both the lengths and diameters were not significant ($P > .05$).

The average weights of the right and left testes were 165.45 ± 9.43 gm and 167.55 ± 8.55 gm respectively. The mean total weight of the pair was 289.67 ± 11.3 gm. The inter-month and intra-month variations for the bulls were found to be highly significant ($P < .01$) as shown in Table 3. The monthly average scrotal-wall thickness was 0.45 ± 0.05 cm. Both inter-month and intra-month variations were not significant ($P > .05$).

Microscopic evaluation of the seminiferous tubules:

Microscopic evaluation revealed: normal testes in 116 bulls; unilateral hypoplasia in 8 bulls and testicular degeneration and orchitis in 140 bulls (slight degeneration in 60 bulls, moderate

degeneration in 50 bulls and severe degeneration in 30 bulls).

The peripheral layer of the seminiferous tubular epithelium was composed of germinal cells, a greater number of which was spermatogonia, at rest or in mitosis and the sertoli cells. Two types of spermatogonia cells (S_{pg}) designated S_{pgA} and S_{pgB} - were identified; with the latter predominating in most cases (Table 2 and Fig.1).

The average monthly diameter of the seminiferous tubules was 222.7 $\pm$ 12 microns. Both the inter-month and intra-month variations ($P < .01$) were highly significant (Table 3). The average number of sertoli cells per tubule was 13.0 $\pm$ 3.0, and both the inter-month and intra-month variations ($P < .01$) were highly significant. These sertoli cells were polymorphic, with their bases on the basement membrane. They had scanty cytoplasm with inconspicuous nuclei. The S_{pgA} and S_{pgB} cells averaged 3.3 $\pm$ 2.0 and 5.4 $\pm$ 3.9 in number respectively per tubule. The inter-month and intra-month variations were highly significant ($P < .01$). The S_{pgA} were round to oval in shape, had one to two nucleoli and a single nucleus with finely dispersed chromatin. This gave a somewhat "dust-like" appearance to the cells as described by Cole and Cupps (1959). Likewise the S_{pgB} were round to oval in shape, and have nuclei with chromatin material which clumped together. This gave the cells a crust-like appearance (Figs.1 and 2). The more

centrally placed primary spermatocytes (fig.1) had a monthly average of 41.0 ± 20.2 per tubule. The transitory secondary spermatocytes were excluded from this study as they were difficult to identify and were rarely seen. The round and elongated spermatids averaged 41.0 ± 9.8 and 35.0 ± 8.3 per tubule respectively. Both the inter-month and intra-month variations were highly significant ($P < .01$). These cells were more centrally located within the tubules than the spermatocytes.

Fig.2 shows the gradation from Spg^A and Spg^B to mature spermatozoa in the process of normal spermatogenesis. The monthly average percentage spermatogenesis in normal and slightly degenerated testes was 77.6 ± 3.1% (Table 2). Moderately and severely degenerated testes averaged 59.7 ± 8.4% and 31.1 ± 11.0% spermatogenesis respectively. Both inter-month and intra-month variations were highly significant ($P < .01$). The difference in percentage spermatogenesis in slightly degenerated testes and in normal testes was not significant ($P > .01$). Fig.3 shows typical severe testicular degeneration.

The difference in the number of germinal epithelial cells in the normal and abnormal testes was reflected in the SCI. The average SCI of normal testes was 11.5 with a range of 9 to 16. The average SCI of slightly degenerated testes was 11.0, ranging from 5 to 14. The moderately degenerated testes had an average SCI of 8.8 and a range of 6 to 11. The average SCI of the severely degenerated testes was 5.4

ranging from 3 to 9. Both the inter-month and intra-month variations between the normal testes (and slight degeneration) and severe degeneration were highly significant ($P < ,01$).

Table 1: Mean biometric data of the testicles from 264 indigenous bulls in Northern Nigeria

Month	Right testis Length (cm) M	SD	Left testis Length (cm) M	SD	Right testis Diameter (cm) M	SD	Left testis Diameter (cm) M	SD	Right testis Weight (gm) M	SD	Left testis Weight (gm) M	SD	Scrotal thickness (cm) M	SD
1977: JAN	12.0	± 1.10	12.2	± 1.0	4.9	± .83	5.2	± .48	158.7	± 8.9	166.7	± 11.6	0.40	+ .04
FEB	10.3	± .67	9.9	± .81	4.2	± .47	4.6	± .63	130.4	± 9.10	133.5	± 8.5	0.40	± .05
MAR	10.1	± .36	10.4	± .93	4.5	± .33	4.8	± .81	108.5	± 3.40	114.2	± 3.4	0.38	± .01
APR	9.6	± .18	9.5	± 1.0	3.9	± .75	3.9	± .30	101.9	± 1.8	103.0	± .9	0.42	± .06
MAY	8.0	± .21	7.5	± .24	3.6	± .60	3.8	± .08	58.1	± 2.2	55.1	± 54	0.50	± .09
JUN	11.9	± 1.13	11.8	± 1.1	5.5	± .88	5.6	± .94	172.00	± 17.2	184.1	± 12.1	0.51	± 0.10
JUL	14.0	± 2.10	13.5	± 2.13	4.6	± .47	4.5	± .54	163.4	± 4.9	162.5	± 9.8	0.48	± 0.07
AUG	16.0	± 2.70	17.0	± 4.76	5.8	± .93	5.5	± .50	205.0	± 21.9	231.9	± 16.4	0.46	± 0.05
SEP	15.1	± 3.83	15.1	± 2.80	7.0	± .94	7.7	± .89	324.0	± 26.7	324.0	± 22.1	0.40	± 0.03
OCT	13.4	± 1.60	12.7	± 1.20	5.8	± .80	6.0	± .38	158.5	± 10.4	152.7	± 11.5	0.50	± 0.01
NOV	11.0	± 1.10	9.2	± .47	4.5	± .36	4.3	± .4	121.1	± 4.3	100.7	± 2.3	0.40	± 0.07
DEC	10.5	± .55	9.5	± .31	5.0	± .10	4.4	± .03	284.0	± 2.4	282.2	± 3.4	0.48	± 0.03
Total	142.3	± 15.53	138.3	± 16.75	59	±7.49	60.3	± 5.98	1985.6	±113.20	2010.6	± 102.54	5.33	± 0.61
Aver.	11.86	± 1.30	11.53	± 1.40	4.9	± 0.60	5.03	± 0.50	165.45	± 9.43	167.5	± 8.55	0.44	± .05

M = Mean
SD = Standard deviation

Table 2: Histological quantitative evaluation of spermatogenesis in Bunaji bulls in Northern Nigeria

Month	Tubular diameter (u) M	SD	No. of Sertoli Cell M	SD	No. of Sp"gonia A M	SD	B	No. of stcytes I	II	No. of stids R M	SD	L M	SD	% Spermatogenesis M	SD
JAN	244 ± 19		16 ± 4		2 ± 1.2		1 ± .9	22±9.9	—	89±18.1		3±1.4		80.0 ± 2.5	
FEB	207 ± 8		13 ± 2		6 ± 1.8		5 ± 2.3	33±15.1	—	15±3.0		150±61		80.1 ± 1.7	
MAR	171 ± 4		19 ± 7		5 ± 2.4		9 ± 2.9	30±20.2	—	13±3.1		10±1.1		69.8 ± 0.6	
APR	218 ± 9		10 ± 1		3 ± 1.8		4 ± 1.8	28±17.4	—	18±1.8		6±1.0		65.3 ± 0.6	
MAY	180 ± 8		15 ± 3		2 ± 1.6		2 ± 1.1	27±11.4	—	8±1.0		63±3.7		57.0 ± 1.4	
JUN	224 ± 17		9 ± 3		2 ± 1.7		5 ± 1.9	42±19.3	—	88±11.1		20±11.0		70.2 ± 2.2	
JUL	258 ± 16		7 ± 2		3 ± 2.4		6 ± 7.1	64±27.7	—	29±3.1		20±1.9		71.8 ± 1.6	
AUG	261 ± 14		13 ± 4		3 ± 2.6		11 ± 3.6	35±25.3	—	119±2.1		15±1.7		85.5 ± 2.1	
SEP	264 ± 14		17 ± 5		4 ± 2.4		4 ± 2.1	60±16.2	—	108±226.0		57±11.1		82.9 ± 3.7	
OCT	255 ± 21		9 ± 1		2 ± 1.6		3 ± 2.5	45±21.3	—	127±17.9		12±.9		89.3 ± 5.1	
NOV	183 ± 13		14 ± 5		3 ± 2.4		4 ± 3.1	72±31.8	—	117±21.4		39±5.1		92.6 ± 8.7	
DEC	207 ± 11		14 ± 4		4 ± 1.7		11 ± 8.7	33±26.3	—	112±9.1		25±2.0		86.3 ± 5.9	
TOTAL	2672 ± 154		156 ± 41		39 ± 24.1		65 ± 4.7	491±241.9	—	495±117.6		420±99.9		930.8± 37.1	
AVE.	222.7± 12		13 ± 3		3.3±2.0		5.4± 3.9	41±20.2	—	41±9.8		35±8.3		77.6± 3.1	

M = Mean
SD = Standard deviation
Sp"gonia = Spermatogonia A and B
Stcyte 1 = Primary spermatocyte
Stcyte II = Secondary spermatocyte
Stid R = Round spermatid
Stid L = Elongated spermatid

Table 3: Analysis of variance of the variations in testicular and spermatogenic parameters in indigenous bulls in Northern Nigeria

Parameters	Sources of variation	S.S	D.F.	***Mean-square
Seminiferous tubular diameter	Between months	429116.8472	11	39010.6225
	Within months	498315.4461	588	847.4752
Sertoli cells	Between months	785.6933	11	71.4267
	Within months	9916.1400	588	16.8642
Spermatogonia-A	Between months	342.8844	11	31.1713
	Within months	2541.2756	588	4.3219
Spermatogonia-B	Between months	534.3711	11	48.5792
	Within months	7464.3222	588	12.6944
Primary spermatocyte	Between months	55204.7806	11	5018.6164
	Within months	239225.1377	588	406.8455
Elongated spermatid	Between months	383929.2531	11	34902.6594
	Within months	2947008.3402	588	5011.9189
Round spermatid	Between months	48241.6309	11	4385.6028
	Within months	586354.1674	588	997.2010
% Spermatogenesis	Between months	2764.2372	11	251.2934
	Within months	35739.5961	588	60.7816
Testicular weight	Between months	276058.2576	11	25096.2052
	Within months	2189772.5274	588	3724.1029

*** Between and within months variations were highly significant for all parameters.

SS = Sum of square

DF = Degree of freedom

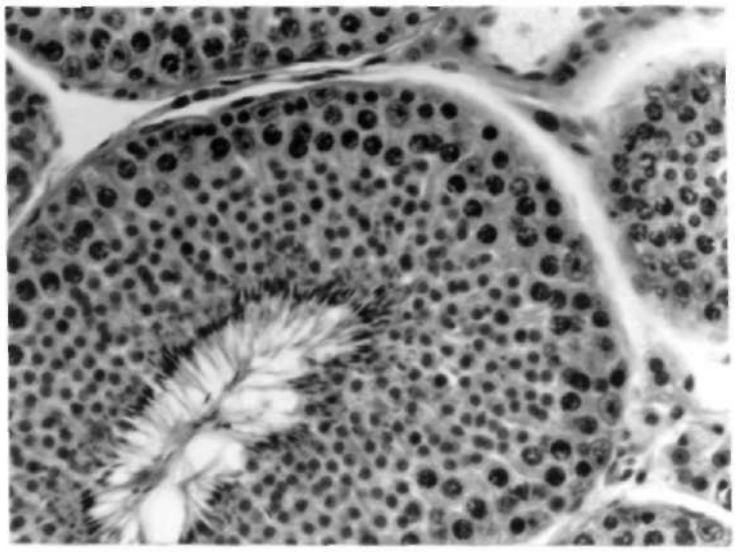


Fig.1 Process of spermatogenesis in a normal testis showing the germinal and sertoli cells. Note the prominent spermatogonia -A and -B x450

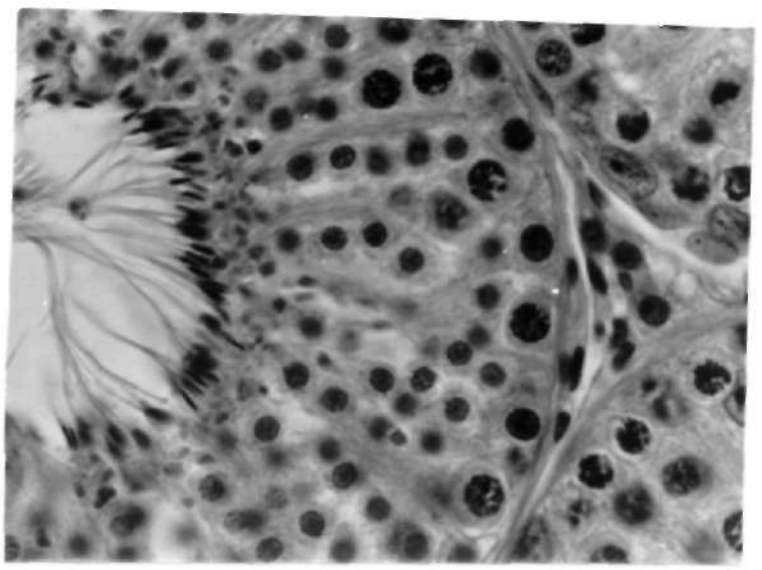


Fig.2: Process of spermatogenesis in a normal testis showing the gradation of spermatogonia A to mature spermatozoa. x 450

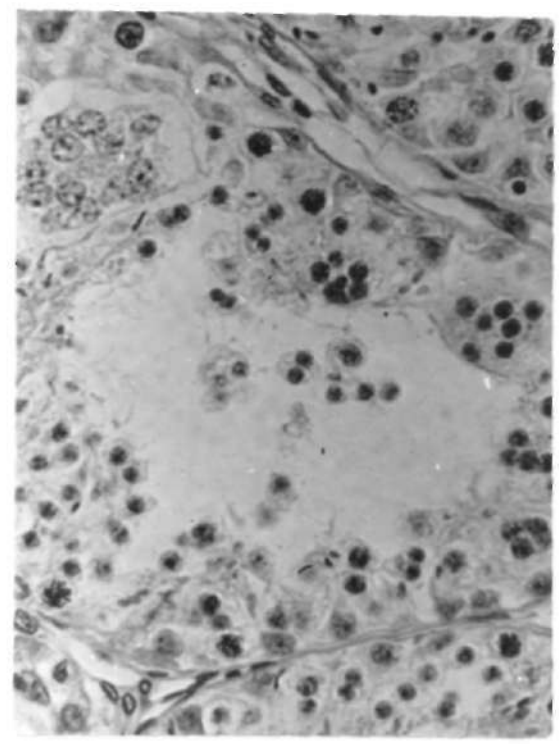


Fig. 3: Cross section of the testis showing severe testicular degeneration. Note reduction in the number of germinal epithelial cells. x 450

Discussion

The mean values obtained for the length, diameter, and weight of the testes were lower than those reported for Bos taurus breeds (Ellenberger-Baum, 1939). According to Igboeli and Rakha (1971), the testes of local bulls are, in general, much smaller than those of exotic breeds. This is reflected in the mean ejaculate volume and total sperm output of ejaculate. A high positive correlation has been reported between testicular size and sperm production (Willet and Ohms, 1957; Hupp, et al. 1962; Hahn, et al. 1969).

The significant inter-month and intra-month variations observed might reflect individual variations, and might also possibly be due to the breed of Bos indicus used in the study, their age, size, pathologic, physiologic and environmental factors such as nutrition.

The mean diameter of the seminiferous tubules provides a useful and simple guide for assessing testicular development and function (Hay, et al. 1961). The values obtained for the tubular diameter were inconsistent with those reported by Lagerlof (1939). The range of 180 u to 264 u was within the range of 170 um to 300 um, but less than the maximum that Lagerlof (1939) reported for the Bos taurus. Tubular diameter is dependent on the stage of the seminiferous epithelial cycle. Testicular size, in general,

can be influenced by nutritional, environmental, physiological and pathological conditions (Lagerlof, 1939). One or the other of these factors might have affected the measurements obtained in this study.

Spermatogenic activity was manifested by the presence of germinal cells and the various stages in the development and maturation of spermatozoa in the seminiferous tubules. The mean percentage spermatogenesis, expressed by the number of tubules containing spermatids per low power fields (x100), was 77.6 ± 3.1%. The significant inter-month and intra-month variations might be due to: different ages of the bulls, pathological and physiological conditions, seasonal, nutritional and other factors yet to be identified.

Bulls in the tropics are considered to be under temperature stress and, as such, would be expected to produce poor quality semen - a reflection of disturbed spermatogenesis (Casady, et al 1953; Dutt, 1960; Skinner and Louw, 1966). The present results do not support this view, and indicate that there is active spermatogenesis throughout the year. The mean monthly percentages of spermatogenesis and correlation of the gross testicular dimensions indicated increased activity early in the rainy season and extending towards

the end of the pre-dry season; a factor in keeping with the better nutrition during these periods. This finding is consistent with previous reports (Kumi-Diaka, 1976).

Sertoli cell index (SCI) as a measure of testicular degeneration was studied. Moderate and severe degenerations affected the normal or basic ratio. The SCI decreased with the severity of degeneration, indicating disturbed spermatogenesis. This, in part, was due to the fact that more germinal cells were affected, and sometimes sloughed off, in severe degeneration. In this study, sertoli cells appeared fairly constant in all cases as the total number of the germinal cells decreased with increased severity of degeneration. It could, therefore, be concluded that the sertoli cells were highly resistant to the factors which were harmful to the germinal cells as reported by Faulkner (1969). It was also obvious from this investigation that slight degeneration showed no significant effect on spermatogenesis. The significance of SCI as an index of testicular degeneration, and therefore, the degree of spermatogenesis, is not clear, in view of the many factors involved in its determination. This view is in conformity with the reports of an earlier study (Kumi-Diaka and Dennis 1978).

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SECTION IV

A clinico-pathologic study of the accessory genitalia, prepuce and penis of indigenous bulls in Northern Nigeria

Summary

Clinical, post-mortem and histological studies of the penis, prepuce and accessory genitalia of 4730 indigenous bulls in Nigeria were carried out. The bulls varied in age from 3 weeks to 10.5 years.

The most common clinical condition with regard to the external genitalia was preputial prolapse (0.4%). Four of the lesions had resulted in phimosis. Balanoposthitis occurred in 6 bulls (0.13%); phimosis in 7 bulls (0.15%); paraphimosis in 2 bulls (0.04%); penile fracture in 8 bulls (0.17%); penile deviations in 4 bulls (0.08%); hematoma in 9 bulls (0.19%) and adhesions in 11 bulls (0.23%). Adhesion between the prepuce and penis was found to cause phimosis. Posthitis and balanoposthitis resulted in cases of phimosis and paraphimosis. The bacterial flora commonly observed in the preputial cavity included Corynebacterium pyogenes, Staphylococcus aureus, and C. renale. Their significance in cases of balanoposthitis could not be ascertained in the present study. Other clinical conditions of the prepuce and penis such as penile tumors and urolithiasis were not seen.

Persistence of the frenulum as a cause of phimosis was seen in two bulls.

Seminal vesiculitis was the most common condition clinically and histologically diagnosed with regard to the accessory genitalia. Grossly 3 bulls had unilateral left seminal vesicular hypoplasia, 7 had bilateral atrophy and 26 bulls had seminovesiculitis of varying degrees of severity. The bacterial flora isolated from suppurative seminovesiculitis in 2 bulls included C. pyogenes, beta-hemolytic Escherichia coli and Staphylococci. The significance of these organisms could not be ascertained.

The most common histopathologic changes observed in the seminal vesicles were varying degrees of severity of inflammatory reactions - mild, in 119 bulls (5.95%), moderate in 69 bulls (3.45%) and severe in 28 bulls (1.40%). The overall incidence of 10.8% was higher than reported in the literature, and was rather difficult to account for in this study.

No gross or histopathologic lesions were observed in the prostate glands, pelvic urethrae and ampullae in all the bulls investigated.

Introduction

The testes perform a dual function of spermatogenesis and steroidogenesis. But the viability of spermatozoa depends on the physical and physiological status of both the testes, epididymes and the accessory genitalia. The seminal vesicles produce about 85% of the seminal fluid (Roberts, 1971). Fructose, the major energy source for spermatozoa is a component of this fluid; and spermatozoa acquire active motility when they enter the seminal fluid. Any intrinsic or exogenous factors which disturb the normal function of the accessory genitalia, especially the seminal glands, may alter the viability of spermatozoa and hence affect fertility.

Certain pathological conditions of the entire penile gland and prepuce may affect libido and mounting ability of the bull. These conditions, may therefore lead to infertility and/or sterility, though spermatogenesis may be normal (Zemjanis, 1971; Roberts, 1971; McEntee, 1962). Other genital conditions, such as seminovesiculitis, may manifest themselves in preputial discharges (Ball et al, 1968).

The causes of infertility in bulls indigenous to Nigeria have not been well documented. It has been however, observed that under-nutrition and malnutrition rather than organic diseases play a more significant role in infertility (Kumi-Diaka, 1976).

The objectives of this study were:

(a) to study the gross and histopathological conditions of the accessory genitalia with particular emphasis on the seminal vesicles, and (b) to study the gross pathological conditions of the penis and prepuce which may interfere with fertility in indigenous bulls of Northern Nigeria.

Materials and Methods

The entire penis and preputial sheath of 4730 White Fulani (Bunaji), Rahaji and Sokoto Gudali bulls (aged 3 weeks to 10.5 years) were examined clinically and at post-mortem in 10 abattoirs in Kano and Kaduna States of Nigeria for a period of 12 months. These bulls came from different parts of Northern Nigeria. Pre-slaughter examinations following adequate restraint included palpation of entire genitalia and retraction of the penis from the preputial sheath, whenever possible, using electroejaculator. The accessory genitalia were clinically examined by rectal palpation. The bulls examined in the previous sections (II-III) were included in this study.

Grossly the penis and prepuce were carefully examined for: discharges, lacerations, posthitis, balanitis, paraphimosis, phimosis, ruptures, hematoma, abscesses, adhesions, deviations and

and other congenital or acquired abnormalities of the retractor penis muscles and sigmoid flexure. The accessory genitalia were examined *in situ* as soon as the bulls were slaughtered, before being dissected for further gross examination, within 30 minutes after slaughter. Grossly, these glands, especially the seminal vesicles, were examined for: symmetry, size (hyperplasia, hypoplasia, aplasia), lobulations (exaggerated, reduced, smooth, normal) and other gross abnormalities.

Histopathological studies were carried out on the accessory glands of 2000 bulls. Sections, about 5mm thick and 10mm long, from the glands - ampullae, seminal vesicles, prostates and pelvic urethrae - were cut and fixed in Bouin's solution for 48 hours. Paraffin sections were cut at 5 to 6 microns and stained with hematoxylin and eosin (H & E), Masson's trichrome (M T), Van Gieson (V G) and Brown and Brenn (B & B). Aerobic bacteriological study of the penis, prepuce and accessory genitalia was carried out on the same 2000 bulls, using blood and McConkey agar plates, incubated at 37°C for 48 hours. Staining was carried out, using Gram stain. Stained or isolated bacteria were identified according to the method of Merchant and Packer (1967).

Results

Gross findings

An overall 1.87 per cent incidence of gross pathologic

conditions was encountered, and the detailed results are summarised in Tables 1,2 and 3. Preputial prolapse was encountered in 22 bulls (0.47%). Four of the cases were severe and chronic and had resulted in phimosis. Other conditions observed were; balanoposthitis (0.13%), phimosis (0.15%), paraphimosis (0.04%), hypospadias (0.13%), penile fracture (0.17%) and penile deviation (0.08%). Fig 1 shows a case of hypospadias and typical penile fracture is illustrated in Fig 2. Adhesions were seen in 11 bulls, aged 2.5 to 5 years, hematoma in 9 bulls and abscess anterior to the sigmoid flexure in one bull. The adhesions between the prepuce and penile urethrae were mostly slight, except in one case which had resulted in phimosis. At post-mortem, further adhesions were observed in the region of the sigmoid flexure (Fig.3), and between the latter and the adjacent tissues in 7 bulls whose ages ranged between 6 and 10 years.

The hypospadias were observed at birth in 2 bull calves, and 4 other cases in 3 to 4 weeks old bull calves. Conditions such as tumors of the penis and urethral calculi were not seen.

A total of 36 bulls showed gross lesions of the seminal vesicles; 3 bulls had left unilateral hypoplasia, (left 5 gm vs right 78.9 gm; left 26.7 gm vs right 101.30 gm; left 29.91 gm vs right 95.8 gm). Seven bulls aged 3 - 7.5 years had bilateral

atrophy of the seminal vesicles (average weight of 27.5 gm) and 26 bulls had seminovesiculitis (Fig.4). Seminovesiculitis was characterised by reduction in or complete loss of lobulations, enlargement, firmness and induration in some cases and peritoneal adhesions. No gross lesions were rectally palpated or seen at post mortem in the ampullae, prostate glands and pelvic urethrae of any of the bulls. Grossly normal pelvic genitalia is shown in Fig.5.

Bacteriological findings

Mixed bacterial flora from the prepuce of 950 bulls included; Escherichia coli both hemolytic and non-hemolytic; Corynebacterium pyogenes; Staphylococcus aureus; Streptococci spp and Proteus vulgaris. The seminal vesicles of 2 bulls contained purulent exudates from which C. pyogenes and hemolytic E. coli were isolated.

Histopathologic findings

Table 3 shows the results of the histopathologic findings of the pelvic genitalia. Seminovesiculitis was observed in 216 bulls (10.8%). Of this, 193 cases (9.65%) were unilateral and 23 cases (1.15%) bilateral. The severity of the condition was variable. Mild seminovesiculitis was seen in 69 (3.45%) bulls and severe case in 28 (1.40%) bulls. The mild forms consisted mainly of multifocal, perivascular infiltration of mononuclear cells; there was no fibrosis

and only a few stromal spaces were affected. The moderate seminovesiculitis was exhibited by multifocal and in some cases diffuse, stromal infiltration of neutrophils and mononuclear cells, and moderate fibrosis (MT-stain) of the interstitium. In chronic severe seminovesiculitis, there was diffuse perivascular and subepithelial infiltration of mononuclear cells, and varying degrees of fibrosis (Fig.6).

There was no histopathologic lesion observed in the prostate glands, pelvic urethrae and ampullae of any of the bulls examined.

Table 1: Gross defects of the penis, prepuce and accessory organs of indigenous bulls in Northern Nigeria

Location of Abattoir	No bulls examined	Breed*	Preputial prolapse	Balanoposthitis	Phimosis	Paraphimosis	Penile rupture	Penile deviation	Hypospadias	Seminovesiculitis	Seminovesicular atrophy	Total cases
Kano	3600	2400R	15	5	3	0	0	2	3	9	3	40
		1200WF	6	0	0	2	2	2	0	2	0	14
Danbata	78	WF	0	0	0	0	3	0	0	1	0	4
Bunkure	36	R	0	0	0	0	0	0	0	2	0	2
Kadawa	6	SG	0	0	0	0	0	0	0	0	0	0
Tiga	10	SG	0	0	0	0	0	0	0	0	0	0
Zaria	350	WF	0	0	0	0	0	0	1	0	3	4
Samaru	87	WF	1	1	1	0	1	0	2	5	0	11
Funtua	143	WF	0	0	3	0	2	0	0	0	0	5
Bakori	3	SG	0	0	0	0	0	0	0	0	0	0
Kaduna	417	WF	0	0	0	0	0	0	0	7	1	8
Total	4730		22	6	7	2	8	4	6	26	7	88
% age			0.47	0.13	0.15	0.04	0.17	0.08	0.13	0.55	0.15	1.87

* Breed: R = Rahaji; WF = White Fulani; S.G. = Sokoto Gudali

Table 2: Gross pathologic observations of the genitalia of 4730 indigenous bulls in Northern Nigeria

Gross lesions	No of bull affected	Percent affected
Preputial prolapse	22	0.47
Balanoposthitis	6	0.13
Phimosis	7	0.15
Paraphimosis	2	0.04
Penile rupture	18	0.17
Penile deviation	4	0.08
Hypospadias	6	0.13
Seminovesiculitis	26	0.55
Seminovesicular atrophy	7	0.15
Total	88	1.87

Table 3: Histopathologic observations of the pelvic genitalia of 4730 indigenous bulls in Northern Nigeria

Histopathologic lesions	No of bulls affected	Per cent affected
Seminovesiculitis:		
unilateral	193	9.65
bilateral	23	1.15
Ampullitis	0	0.00
Prostatic lesions	0	0.00
Urethral lesions	0	0.00
Total	216	10.80%



Fig.1: Hypospadias in a two-day old bull calf



Fig.2: Hematoma following penile rupture in a bull

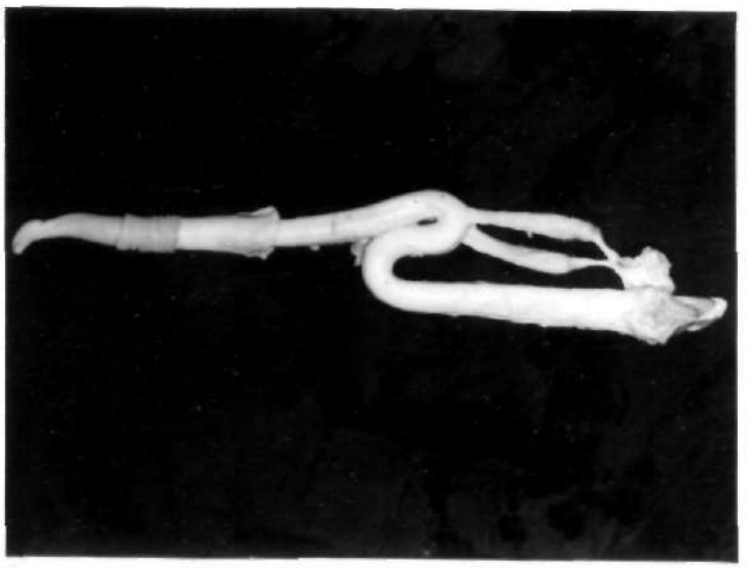


Fig.3: Severe adhesions in the region of sigmoid flexure, in a bull, with resultant phimosis



Fig.4: Seminal vesiculitis in a bull with loss of lobulation and fusion of ampullae

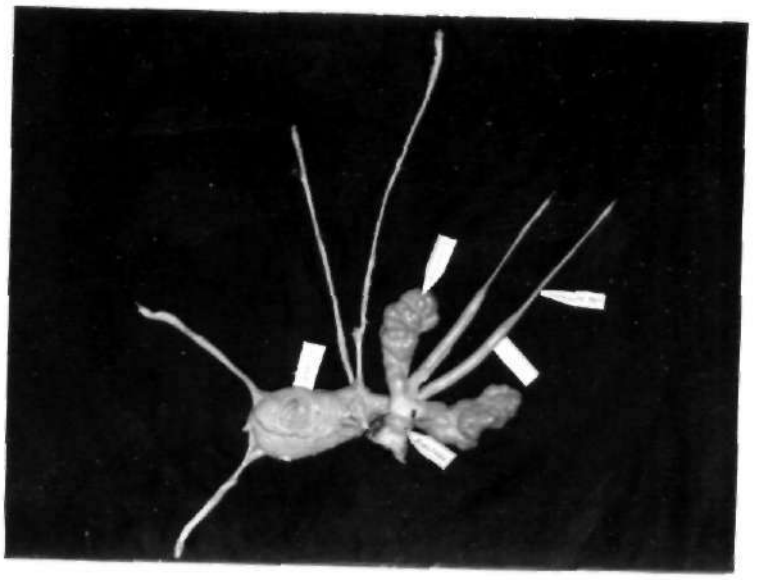


Fig.5: Normal pelvic genitalia of bull showing normal pelvic urethra, prostate gland, seminal vesicles and ampullae

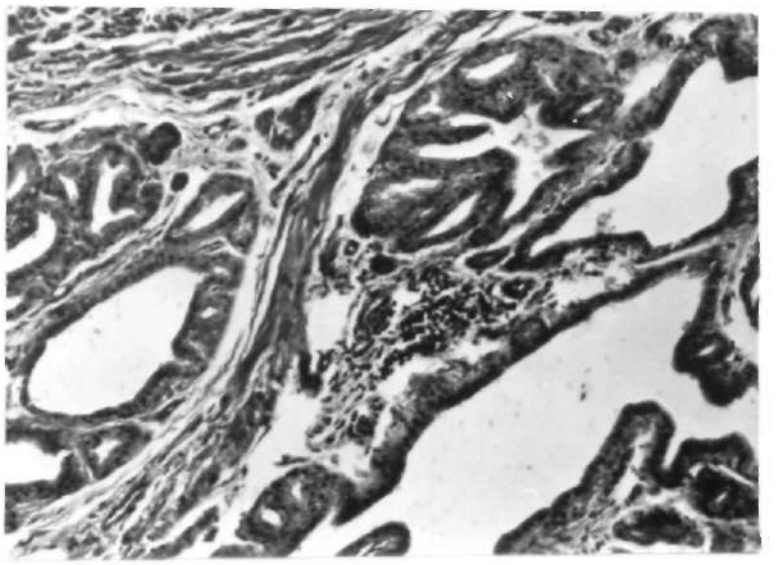


Fig.6: Severe seminovesiculitis. Note mononuclear cells and fibrosis in the interstitium x450

Discussion

The most common gross pathologic finding was prolapse of the prepuce which resulted in stricture and phimosis. This has been reported by Wheat (1951) who stated that such strictures would require surgical amputation to effect protrusion of the penis. It was observed in this study that preputial prolapse of chronic duration was a common cause of posthitis. This observation was in accordance with the findings of Donaldson and Aubrey (1960). They observed that the condition was common in Bos indicus and less so in the Bos taurus breed. Roberts (1971) postulated that chronic preputial prolapse is probably an inherited trait associated with pendulous sheath and large preputial orifice. The Bos indicus in Northern Nigeria have prepuce with large pendulous sheath and relaxed preputial sheath/membrane and penis. Certain factors such as chronicity of the prolapse and trauma may lead to edema and fibrosis, thus resulting in phimosis. The significance of the bacterial organisms isolated from the lesions could not be ascertained as most of them were also isolated from bulls with apparently normal prepuce and penis. Bane (1964) reported that trauma of the prepuce and penis usually resulted in the introduction of bacterial organisms. From this study, it appears the bacterial flora encountered may be a normal flora of the prepuce.

According to Morgan et al. (1946), Rempt and Zwanenburg (1949), Bakos et al. (1962) and Bane (1964), cultural studies of the genital mucous membranes of bulls indicate that Pseudomonas aeruginosa, C. pyogenes, C. renale and E. coli are rather frequently present. C. pyogenes and C. renale have been incriminated in abortions (Smith and Jones, 1966). Although these appear as normal flora they could become pathogenically important in case of traumatic prolapse.

Adhesions seen in the region of the sigmoid flexure were likely to interfere with free movement of the penis and prepuce at erection. Some of these adhesions might be due to trauma and inflammation. The penile fracture observed had resulted in hematoma, adhesions and phimosis. Farquharson (1952) and Roberts (1971) reported that adhesions in the region of the sigmoid flexure often prevented obliteration of the S-curve and protrusion of the penis during erection. Gibbons (1956) reported that common causes of preputial and penile adhesions in bulls are horn injuries. This study reported here is in accordance with the previous findings.

The cause of the penile fractures could not be ascertained. Wheat (1951) and Gibbons (1956) stated that the cause of penile fractures and hematomas are most frequently due to accidents during

coitus, when a sudden bending and trauma of the penis is produced. It is assumed that the same could be true of the Fulani bulls, where young, active, inexperienced bulls are left with the heifers/cows at all times.

Spiral penile deviation of unknown etiology was seen in 4 bulls. Ashdown and Combs (1967) reported the same, but not the frequency, in the Bos taurus breed. They attribute the cause to anatomical defects of the muscle, erectile tissues or fasciae of the penis or defective retractor penis muscle.

Tumors of the penis are considered to be common, especially in young bulls (Wheat, 1951; Smith & Jones, 1966). McEntee (1969) reported that transmissible fibropapilloma is significant in young bulls, especially when housed together. The findings in this study contrasted with that finding. No tumor of any kind was seen in any of the 4730 bulls examined. This might be due to many factors such as: management practices, breed of bulls involved, environmental factors, age and other factors yet unknown. Fulani cattle are never restricted (housed) and hence the chances of young bulls mounting each other and injuring the prepuce are minimized.

Persistence of the penile frenulum as a cause of phimosis was observed in 2 bulls (19 and 25 months old). Ashdown (1962) reported persistence of the penile frenulum in young Bos taurus bulls.

Other clinical conditions of the prepuce and penis, such as urethral calculi and tuberculosis of the penis, reported by other investigators (Wheat, 1951; Smith & Jones 1966; Roberts, 1971), were not seen in the present study.

The incidence of vesiculitis observed was higher (10.8%) than reported elsewhere in the literature (0.85% by Blom and Christensen, 1947; 4.7% by McEntee, 1962; 2.5% by Carroll et al., 1963; 2.46% by Ball et al 1964). Seminal vesiculitis has been reported to be more common in young bulls because of their homosexuality when confined than in middle-aged bulls (Ball et al., 1968; Roberts, 1971). The bulls included in this study were between 3 months and 10.5 years old and most of the conditions observed were in the middle-aged bulls (3 - 7 years). In Nigeria clinical examination of the reproductive organs is seldom performed prior to sexual maturity and thereafter. Confinement of cattle is not a normal husbandry practice of the nomadic Fulani herdsmen. The higher incidence of the condition observed in this study cannot therefore be adequately explained. Many factors and their interactions may explain, in part, the 10.8% incidence of the disease encountered. These include: age of bulls, breed differences, etiological agents involved, season, husbandry practice, nutrition and management. Smith and Jones (1966) noted metaplasia of the

epithelial lining of the seminal vesicles in bulls due to avitaminosis-A. In general, the type of histopathologic lesions observed in this study was in agreement with the lesions described by Galloway (1964); Carroll et al. (1968); Roberts (1971) and Bouters (1964).

The bacterial organisms isolated in some of the seminovesiculitis were in confdrmity with the findings of Bouters, (1964); Galloway, (1964); Roberts (1971). At best these organisms could only be incriminated as a possibe cause of the purulent seminovesiculitis observed. There was no positive evidence that they were the primary cause of the lesion. However, the presence of C. pyogenes could be significant, as although found in the normal genitalia of both cows and bulls, could become important as secondary infections following such factors as injuries or changes in the physiology (Hancock and Kelly, 1948).

The prostate glands and ampullae appeared to be resistant to many of these microbial and other noxious agents as no gross or histopathologic lesions were observed in them. Smith and Jones (1966) reported that the prostate gland in bulls is a little more than rudimentary in size and its diseases rare. They observed that the seminal vesicles and the bulbo-urethral glands tend to harbour

infections because of their complicated structure and poor drainage.

In an earlier study, 2 bulls with seminovesiculitis also had epididymitis. It is possible that a focal infection could spread to other parts of the genitalia, either by the ascending or descending route. This observation has been made by Carroll et al., (1968).

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SECTION V

The pathology of bovine male genitalia experimentally infected with Corynebacterium pyogenes

Summary

Twelve 20-to-22 month-old Bunaji bull calves belonging to the nomadic Fulani were used in the study. They were divided into two groups, namely, experimental and control, comprising of 8 and 4 bull calves respectively. The experimental animals were further divided into stressed and non-stressed groups. All the experimental animals were injected intravenously via the jugular vein with viable Corynebacterium pyogenes suspension, ranging from 4×10^{12} to 4×10^{21} organisms per dose. The control animals were similarly injected with formalin-killed organisms. The C. pyogenes strain was isolated and identified from bovine suppurative mastitis.

Rectal temperatures of the experimental animals began to rise from 4 to 5 days post infection (P.I.) and reached a peak on day 12 P.I. Thereafter, moderate pyrexia was maintained in most of the stressed-infected bull calves. Physical condition of animals ranged from fair to good in the non-stressed, infected and the control groups, to cachexia in the stressed infected group.

Clinically, varying degrees of orchitis, epididymitis and seminovesiculitis were detected in the infected groups. Hematological findings revealed leukocytosis (predominantly neutrophils) at

the height of infection. This dropped to near-normal level around 6 weeks P.I. but slight neutrophilia persisted in the stressed, infected group throughout the course of infection. The hematological values for the control group remained relatively stable and within normal range throughout the experimental period.

At post-mortem, varying degrees of inflamation-enlargement, adhesions, and hemorrhages - were observed in the testes, epididymes and seminal vesicles in the experimental groups. There was no significant lesion in other glands and/or organs. No gross lesions were found in the control animals. Bacteriologically, pure culture of C. pyogenes was isolated from different parts of the genitalia of the infected animals. Few colonies of Escherichia coli, Staphyococci and C. pyogenes were isolated from the genitalia of the control animals. Histopathological lesions observed in the different parts of the genitalia in the infected animals included varying grades of inflammation and degeneration. The testes and epididymes were most affected and the prostate and ampullae the least affected. There were no significant lesions in the genitalia of the control animals. There were greater sperm morphologic abnormalities in the infected bulls than in the non-infected control bulls. The highest percentage of spermatogenesis (96%) was recorded in the control bulls and the lowest (66%) in the stressed-induced, infected bulls.

The study indicated that C. pyogenes infections could play a role in genital pathology and any factor that may significantly reduce the resistance of the host could be a major factor in the severity of C. pyogenes infections.

Introduction

The genus Corynebacterium is found in animals and man. A number of species are pathogenic for animals but these species rarely infect man (Ballard et al., 1947; Lopez et al., 1966) Corynebacteria are Gram-positive, small pleomorphic rods. In Gram-stained smears from tissues they may be confused with streptococci, staphylococci and listeria.

Corynebacterium pyogenes is a common pyogenic bacterium in cattle, sheep, goats, swine and occassionally horses and birds (Merchant, 1935; Cowle, 1962; Smith, 1966; Addo and Dennis, 1977). It is believed that it occurs in a great variety of suppurative conditions as primary infections, secondary to trauma or in association with other pyogenic organisms (Merchant 1935; Addo, 1976; Addo and Dennis, 1977). It causes bronchopneumonia, mastitis and umbilical infections (Brooks and Hucker, 1944; Morse, 1949; Dennis and Bamford, 1966). In cattle, C. pyogenes causes suppurative endometritis, mastitis, and seminal vesiculitis (Dawson, 1951; Smith, 1966).

C. pyogenes has been isolated in abortions and is thought to be the primary cause (Maxwell, 1943; Sorum 1953; Wilson 1959). Experimental production of C. pyogenes abortion in sheep and cattle has been achieved (Hinton, 1972; Addo and Dennis, 1979). It has been incriminated as a cause of seminal vesiculitis in the bull, though experimental production of the disease by inoculation has not been achieved (McEntee, 1962; Blom and Christensen, 1965; Carroll et al., 1968). Other investigators have reported the isolation of Brucella abortus; C. pyogenes and other bacterial organisms in cases of bovine orchitis and epididymitis (Konig, 1964; Roberts, 1971). Hancock and Kelly (1948) observed that C. pyogenes might be excreted in the semen of clinically healthy bulls for a long period of time. The significance of this in the transmission of genital C. pyogenes is obvious.

The objective of this study was to investigate the significance of C. pyogenes as the primary etiologic agent in genital pathology of the indigenous bulls (Bos indicus) in Northern Nigeria.

Materials and Methods

Animals:

Twelve 20-to-22-month-old White Fulani (Bunaji) bull calves used in this study were housed and grazed in 2 separate fenced

paddocks. Pre-conditioning for 10 weeks included monthly weighing, fecal sample examination for parasites, recording daily rectal temperature, examination for blood parasites, collection and culturing of semen, weekly paipation of pelvic genitalia, and weekly hematological examination.

Inoculum:

C. pyogenes strain was isolated and identified from a case of bovine suppurative mastitis at the Kano Farm Centre, by the method employed by Addo and Dennis (1977). Characterization of the isolate was according to standard diagnostic keys (Carter, 1973; Cowan and Steel, 1970; Addo and Dennis, 1977). It was a beta-hemolytic Gram positive pleomorphic; catalase negative; non-motile; liquefied gelatin in 48 hr.; reduced litmus milk; coagulated casein in 24 hr. The strain was subcultured aerobically at 37°C for 36 hr. The organisms were grown in a 250 ml flask containing 200 ml brain heart infusion broth (Difco Laboratories, Detroit, Michigan, U.S.A.) for 24 hr. at 37°C and harvested by centrifuging at 3000 rpm/5 minutes. The supernatant was discarded and the sediment (cells) suspended in phosphate buffered physiological saline (PBS). The cells were washed 3 times in sterile PBS and resuspended in sterile PBS. Serial dilutions of the suspension were made and 0.1 ml aliquot of each dilution was aerobically cultured on blood and McConkey agar

plates at 37°C for 48 hr, after which bacteria colonies were counted. The number of viable bacteria per millilitre in a suspension was determined, using the different dilution factors.

Formalin-killed antigens were prepared by growing living C. pyogenes in brain heart infusion broth for 48 hr.; harvested in 0.2% formal PBS, and then checking for sterility by streaking on 5% defibrinated blood and McConkey (Oxoid, Ltd., London, England) agar plates and incubating at 37°C for 48 hr. Concentrations per ml were adjusted as described above.

Experimental design: The 12 bull calves were divided into 2 comparable groups based on age and body weight. Each group was housed in a separate pen for the duration of the experiment. The 2 groups were as follows:

a) Group A - Stressed bulls: This group comprised 5 experimental and 2 control bulls. In addition to grazing the available pasture, animals were given mineral licks daily and water ad lib. To create stress and thereby reduce the resistance of the animals to infection, the bulls were given a daily intravenous (I.V.) injection of 30 mg dexamethazone via the jugular vein for 12 days prior to inoculation with C. pyogenes suspension. They were given a further I.V. injections of 30 mg dexamethazone daily post-infection for 2 weeks. Table 1 shows the scheme of inoculation and dosages.

b) Group B - Non-stressed bulls: There were 4 experimental and one control bull calves in the group. An attempt was made to avoid any form of stress in the animals. They were fed 10 kg of cotton seed and mineral licks daily, in addition to grazing. No dexamethazone injections were given. Table 1 shows the scheme of inoculation and dosages of C. pyogenes suspensions given to the bulls.

The inoculum for each animal was suspended in 5 mls sterile PBS and injected I.V. via the jugular vein. All the control bulls were given formalin-killed C. pyogenes suspensions i.v.

In a preliminary study using serial dilutions of C. pyogenes suspensions, it required 5 mls of 4×10^{17} microorganisms (C. pyogenes) given intravenously to produce pyrexia, anorexia and lethargy in poorly fed, emaciated bulls. The same dosage had no significant effect on well-fed, healthy-looking bulls (Kumi-Diaka, 1977 unpublished data). In the present study under-nutrition in addition to dexamethazone, has been used as one of the stress factors.

Table 1: Bovine inoculation schedule with C. pyogenes, and stressed with or without dexamethazone.

Bull tag	No. of organisms	Dexamethazone (30 mg)
Stressed		
1643	4×10^{12}	+
1646	4×10^{12}	+
1650	4×10^{19}	+
1594	4×10^{19}	+
1676	4×10^{21}	+
277*	4×10^{19}	+
255*	4×10^{21}	+
Non-Stressed		
237	4×10^{12}	-
246	4×10^{19}	-
247	4×10^{21}	-
248**	4×10^{21}	-
233*	4×10^{19}	-

* = Control bull calves given inactivated C. pyogenes antigen

** = Bull calve - died 8 days post-inoculation

\+ = Dexamethazone injection

\- = No dexamethazone given

Clinical observations: Rectal temperatures were recorded daily in the morning before grazing, and body weight recorded fortnightly post-inoculation. Close observations were made for obvious changes in behavior. Scrotal contents were palpated and pelvic genitalia were rectally palpated weekly for gross pathologic lesions.

Hematological study: Blood samples were taken weekly for determination of packed cell volume (PCV), total red blood cells (RBC), total white cell count (WBC) and differential cell count, and hemoglobin concentration (Hb). The clinicopathological procedures were carried out according to standard techniques (Benjamin, 1961; Medway et al., 1969).

Semen analysis: Weekly semen samples were collected, where possible by electroejaculation or rectal massage and analysis done according to standard semen evaluation procedures for breeding soundness (Zemjanis, 1970). Semen was bacteriologically examined, using blood and McConkey agar plates.

Gross pathologic examination: Bull calves were slaughtered, first one at 6 weeks post-inoculation (P1) and the rest at approximately weekly intervals thereafter. Gross examination of the entire animal with particular attention to the genitalia was carried out for presence of lesions.

Bacteriological study: Sterile swabs were taken from the epididymis, testis, vas deferens, ampulla, seminal vesicle and prostate. In some cases swabs were also taken from inguinal lymph nodes, urinary bladder, kidney, liver and lungs. All the specimens were streaked on McConkey, blood and phenylethanol agar plates containing 7% defibrinated sheep or cattle blood. The plates were incubated aerobically at 37°C for 48 hr and C. pyogenes and other microorganisms were isolated and identified by standard keys (Carter, 1973; Cowan and Steel, 1970).

Histopathological study: Sections, about 1 cm long and 1 mm thick, were taken from different parts of the entire genitalia and fixed in Bouin's solution for 36 hrs. The tissues were trimmed and processed in a tissuematon*, embedded in paraffin, cut at 5 - 6 microns and stained with hematoxylin and eosin (H & E), periodic acid schiff (PAS), Van-Gieson (VG) and Masson's trichrome (MT). Sections were first screened under low power magnification (X100) and then detailed studies done under higher magnification (X450) and oil immersion (X1000) for histopathologic lesions and bacteriological examination.

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* Fisher Scientific Co. Chicago, UK, U.S.A.

Results

Clinical findings:

Rectal temperatures of infected animals began to rise from 102.8°C on days 4 and 5 post-infection (P.I.), reaching a peak of 105.6°C on days 12 - 14 P.I. (Appendix, Table 1). Thereafter there was a decline in temperature and a moderate pyrexia was maintained in most of the infected bull calves. Bulls in the control group exhibited no significant temperature changes to the killed antigens.

The physical conditions of all the stressed-infected animals remained fair at the early stages of infection. In the late stages of the disease there was gradual emaciation, fairly good appetite, haggard facial expressions, poor gait and anemia. There was weak heart sound and weak pulse on auscultation and percussion; and lachrymation was observed in most of the animals. All the bulls in this group became cachectic at the terminal end of the experimental period.

The physical conditions of the non-stressed infected group remained fair to good throughout the course of infection without significant loss of appetite and body conditions. On auscultation and percussion respiratory and circulatory organs were normal. There was neither coughing nor oculonasal discharge. Animals in the control group were in good bodily conditions throughout the experimental period (Appendix, Table 2).

Appendix, Tables 1 - 7 summarise the clinical, pathological, bacteriological and hematological findings in both the stressed and non-stressed infected bulls. These included varying degrees of orchitis, epididymitis and seminovesiculitis. No gross abnormality was clinically observed in the genitalia of the control, non-infected bulls.

Hematological findings:

The weekly values of the hemogram of the 2 groups are summarised in Appendix, Tables 3 and 4. The total white blood cell count began to rise on the 2nd week post infection (P.I.) in all the infected animals, and reached a peak in most of them on the 6th week (P.I.). Differential count of the white cells indicated neutrophilia, accounting for the leukocytosis. From the 6th week P.I. total white cell count dropped to within normal level, but slight neutrophilia persisted in the stressed infected group. In some cases the neutrophils showed marked toxic granulation.

A significant decrease in the packed cell volume (P.C.V.) was first observed in the 2 infected groups (stressed and non-stressed) at the 6th week P.I. This low level P.C.V. persisted in the stressed infected groups until the termination of the experiment. The mean hemoglobin (Hb) concentration dropped following infection and reached its lowest level in the stressed infected group. The hematological values for the control group remained within normal range throughout the experimental period.

Post-mortem findings: Appendix, Table 5 summarises the gross findings in the genitalia of the bull calves after slaughter. Varying degrees of inflammation - enlargement and adhesions - were observed in the testes, epididymes and seminal vesicles in the infected groups (Fig 1). The prostate glands and ampullae showed no gross lesions. The kidneys and liver of 3 bulls from the stressed-infected group had small multiple white foci, the size of pin-head. There was no indication of pneumonia, and other organs were grossly normal. There was no significant lesion in any organ in the control animals.

Bacteriological results:

Table 6 in Appendix summarises the isolation of bacteria from different organs and semen from the control and infected animals. C. pyogenes were the most predominant bacteria isolated in all cases, alongside with few colonies of Escherichia coli, and Staphylococci. Very few colonies of these organisms were isolated in some of the control animal

Histopathological findings: The changes observed in the genitalia and accessory genital glands are summarised in Appendix Table 7, and Figs. 2 - 5. In general, the lesions in the testes and epididymes were characterised by mild to severe tubular epithelial degeneration and proliferation of fibroblasts and inflammatory cells (neutrophils, lymphocytes and/or plasma cells) in the interstitium.

All the lesions observed in the different organs in the affected animals were similar but varied in extent and degree of severity. In general the stressed-infected group was more affected than the non-stressed infected group. Among the infected groups, the testes and epididymes were most and the prostate and ampullae the least affected. There was no significant lesion in the pelvic urethrae and the penis. There was no significant lesion in the genitalia of the control animals. This latter group scored the highest percentage of spermatogenesis.

Semen examination findings: The effect of C. pyogenes infection of bulls on their spermiogram is summarised in Tables 2 and 3 (Text). There were greater abnormalities in sperm morphology (3 - 12 weeks P.I.) in infected bulls (stressed and non-stressed) than in non-infected bulls. The difference in other parameters in the 2 groups was not significant. The classification of sperm abnormality was based on that of Zemjanis, (1970). Morphological sperm cell defects observed included, degenerated acrosomes, enlarged head, detached head, abaxial mid-piece, coiled and bent tails, distal and proximal cytoplasmic droplets.

Table 2: Spermiogram of some bulls infected with C. pyogenes, 3 - 4 weeks post infection (PI)

Parameters	Bull Tag Nos.					
	Stressed-infected bulls		Non-stressed infected		Control bulls	
	1646	1676	247	237	277	233
Semen volume (mls)	4.8	5.1	4.7	7.5	6.0	5.4
Semen color	cream	cream	milky	cream	cream	cream
Motility*	+	++	++	+++	++	++++
Concentration x 10^9	0.56	0.67	0.49	0.61	0.54	0.59
Abnormality %						
Primary	19.0	21.0	16.0	25.0	9.0	15.0
Secondary	9.0	11.0	11.0	13.0	6.0	7.0
Total %	28.0	33.0	27.0	38.0	15.0	22.0

*+ = poor; ++ = fair; +++ = good; ++++ = very good

Table 3: Spermiogram of some bulls infected with C. pyogenes, 5 - 7 Weeks PI

	Bull Tag Nos					
	Stressed-infected bulls		Non-stressed infected bulls		Control bulls	
	1646	1676	247	237	277	233
Semen vol. (mls)	5.1	5.9	4.8	6.3	5.4	5.0
Semen color	cream	cream	cream	cream	milky	cream
Motility	+	++	++	+++	++	++++
Concentration x 10^9	0.54	0.61	0.53	0.60	0.87	0.68
Abnormality %						
Primary	23.0	27.0	21.0	28.0	8.0	17.0
Secondary	11.0	10.0	15.0	17.0	4.0	11.0
Total %	34.0	37.0	36.0	45.0	12.0	28.0



Fig 1: Scrotal contents showing acute orchitis and epididymitis following experimental C. pyogenes infection. Note adhesions and thickening of tunica albuginea in the stressed infected animals.

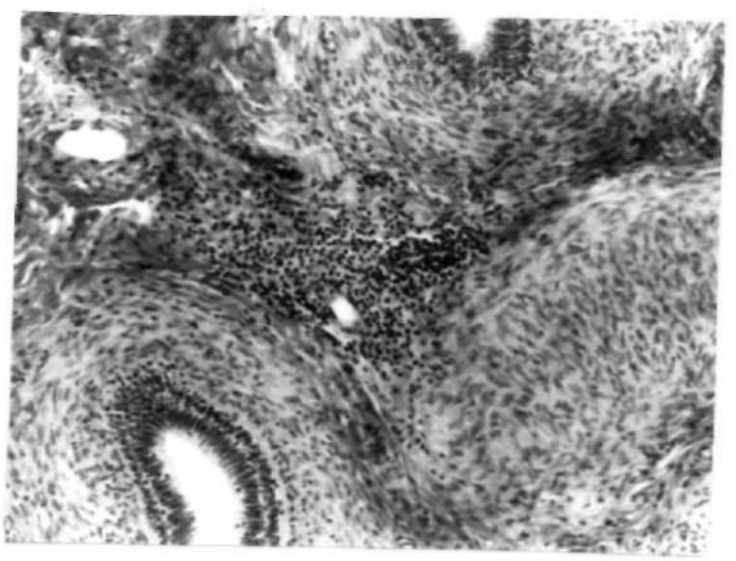


Fig 2: Vas deferens
Note inflammatory process - infiltration of mononuclear cells in the interstitium as a result of experimental C. pyogenes infection x 450.

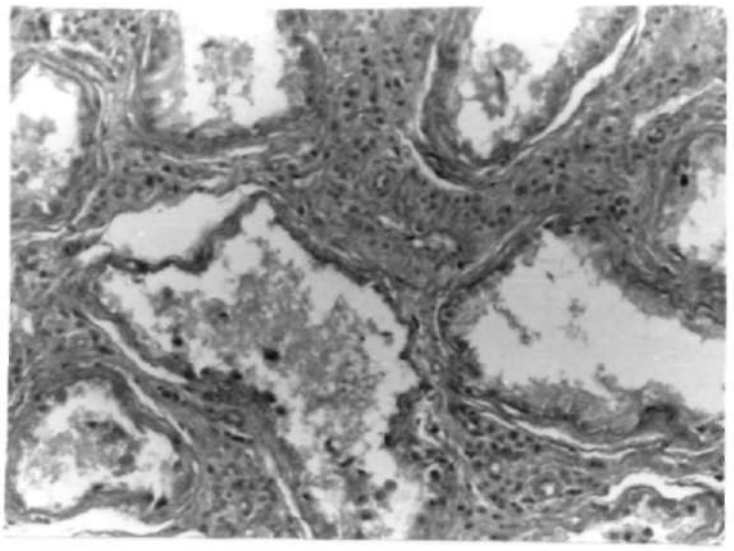


Fig 3: Epididymis:
Note degeneration and sloughing of tubular epithelial cells due to experimental C. pyogenes infection. x 450

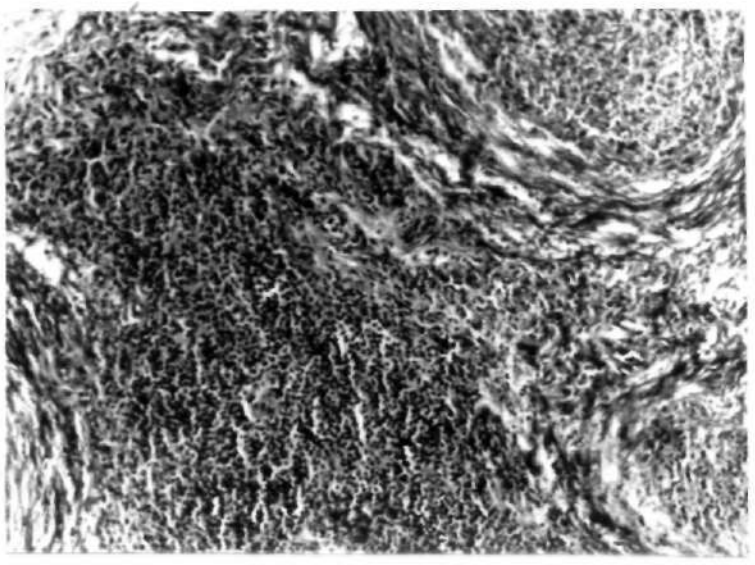


Fig 4: Testis showing acute orchitis. Note loss of architecture and massive infiltration of mononuclear cells following experimental C. pyogenes infection. x 450

Table 1: Daily temperature recordings (^{o}F) of bulls inoculated with *Corynebacterium pyogenes*

	Bull tag nos.										
DAYS	Stress-infected bulls					Non-stressed infected bulls			Control bulls		
PI*	1594	1643	1646	1650	1676	237	246	247	233	255	277
0.	101.8	102.0	102.3	102.1	102.0	102.3	102.8	102.4	101.8	102.0	101.0
1.	102.0	101.8	101.7	101.8	102.1	100.8	102.1	102.0	100.6	102.0	102.8
2.	102.2	101.8	102.0	102.0	102.1	101.2	103.8	100.1	102.0	101.8	100.8
3.	101.8	102.0	102.4	102.1	103.8	101.2	102.1	102.1	102.1	101.6	102.0
4.	102.8	103.1	103.4	102.8	103.0	102.8	103.3	103.0	102.3	102.0	101.8
5.	103.1	103.1	103.8	103.3	103.1	103.4	103.8	103.2	102.1	102.0	102.2
6.	103.9	103.9	104.0	103.8	103.4	103.7	103.8	103.4	101.6	103.1	102.1
7.	104.3	104.3	104.0	103.8	103.9	103.8	104.2	103.8	101.4	102.1	101.8
8.	104.9	104.2	104.8	104.3	104.1	104.1	104.8	103.8	102.3	102.4	102.4
9.	104.9	104.1	105.1	104.7	104.3	104.8	104.9	104.1	102.2	102.3	102.1
10.	104.4	105.1	105.1	104.8	104.2	104.8	104.9	104.8	101.7	102.2	101.4
11.	104.8	104.6	104.8	105.1	104.1	104.6	104.5	104.1	102.2	100.8	101.8
12.	105.6	105.3	104.8	105.0	104.8	104.3	104.1	103.5	101.8	102.1	102.2
13.	105.0	104.7	104.1	104.7	104.0	104.0	104.0	103.1	102.2	100.6	102.2

Table 1: (Cont.)

	Bull tag nos.										
DAYS	Stress-infected bulls					Non-stress infected bulls			Control bulls		
PI*	1594	1643	1646	1650	1676	237	246	247	233	255	277
14.	104.6	104.1	104.5	105.1	104.5	103.1	103.4	103.0	101.6	101.4	101.2
15.	104.6	104.8	103.6	104.7	104.3	103.6	103.0	103.1	102.1	101.6	100.2
16.	104.1	103.9	103.1	104.8	104.1	103.0	103.6	103.1	102.2	102.8	102.4
17.	104.5	103.5	103.0	104.0	104.0	103.8	103.1	103.0	102.1	101.8	102.8
18.	103.9	103.0	103.8	104.6	103.3	103.8	103.0	103.0	102.1	101.6	102.0
19.	103.9	103.0	103.6	103.1	103.3	103.1	103.0	103.8	102.4	102.7	102.0
20.	103.0	103.8	103.6	103.0	103.1	103.2	103.2	103.7	102.6	102.0	103.1
21.	103.1	103.2	103.4	103.0	103.7	103.0	103.1	103.0	102.0	102.1	102.
22.	103.4	103.1	103.1	102.7	103.5	103.1	103.0	103.1	102.8	101.7	102.8
23.	103.9	103.0	103.2	103.4	103.0	103.8	102.8	103.3	101.9	102.1	102.1
24.	103.1	103.0	103.8	103.0	103.0	103.8	103.2	103.0	101.7	102.0	101.9
25.	103.0	103.1	102.9	103.8	103.1	103.1	103.5	103.8	102.2	102.3	101.6
26.	103.1	103.6	103.0	103.9	103.6	103.5	103.0	103.1	102.0	102.0	102.0
27.	103.7	103.4	103.9	103.1	103.3	103.2	103.0	103.6	102.3	101.8	102.4
28.	103.1	103.1	102.8	103.5	103.1	103.0	102.7	103.3	101.9	101.8	102.1
29.	103.4	103.0	102.4	103.5	103.2	103.2	103.3	103.1	101.8	100.9	102.1

Table 1: (Cont.)

DAYS	Bull Tag Nos.										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
PI*	1594	1643	1646	1650	1676	237	246	247	233	255	277
30.	103.4	102.9	102.8	103.5	103.2	103.0	103.1	103.1	101.8	100.9	102.1
31.	103.3	102.4	102.8	103.2	103.0	103.0	103.0	103.0	102.0	101.7	102.0
32.	103.2	103.3	103.1	103.4	103.0	103.1	103.4	102.9	102.1	102.1	102.0
33.	103.3	103.1	102.9	103.3	103.3	103.1	103.1	102.8	101.1	102.1	102.1
34.	103.4	103.0	102.9	103.1	103.4	102.9	103.2	102.8	100.8	102.0	102.0
35.	103.2	103.0	102.6	103.4	103.1	102.9	103.1	103.0	102.1	101.9	102.2
36.	103.2	103.4	102.7	103.6	103.0	102.7	103.4	102.1	102.3	102.0	101.9
37.	103.3	103.1	103.0	102.8	103.1	103.0	103.1	102.9	101.3	102.0	101.5
38.	103.1	103.0	102.8	103.0	103.0	103.4	103.1	103.0	102.4	102.1	100.5
39.	103.5	103.0	103.5	103.0	103.1	102.6	102.4	102.0	102.3	102.3	101.0
40.	102.8	103.0	103.0	103.4	102.8	102.8	102.6	102.0	101.7	101.9	102.6
41.	103.0	103.4	103.1	103.4	102.7	103.0	102.1	102.4	101.6	102.0	102.4
42.	103.1	103.3	103.1	103.1	103.0	103.1	102.9	102.8	102.1	102.0	102.1
43.	-	102.8	103.0	103.4	103.0	103.4	102.8	102.8	102.1	102.1	102.2
44.	-	102.8	103.2	103.1	103.0	102.8	103.1	102.7	102.0	101.9	102.0
45	-	102.4	103.2	103.2	103.1	102.9	102.8	103.0	102.2	101.7	102.0

Table 1: (Cont.)

	Bull tag nos.										
DAYS	Stress-infected bulls					Non-stress infected bulls			Control bulls		
PI*	1594	1643	1646	1650	1676	237	246	247	233	255	277
46.	-	103.1	103.4	102.4	103.0	102.0	102.0	102.6	102.0	100.9	100.8
47.	-	103.1	103.0	103.1	103.0	102.0	102.1	102.8	102.1	101.8	101.8
48.	-	103.2	103.1	103.0	103.3	103.0	102.1	102.7	102.4	102.0	101.1
49.	-	103.4	102.9	103.0	103.0	102.4	102.2	103.0	102.0	102.1	101.9
50.	-	103.4	103.0	-	102.6	102.7	102.2	101.8	102.0	102.0	100.9
51.	-	103.0	103.4	-	102.5	102.8	101.8	102.0	102.0	102.3	102.1
52.	-	102.5	103.0	-	102.7	102.1	102.2	102.9	102.1	102.5	102.0
53.	-	102.9	103.0	-	102.1	102.6	102.1	102.0	102.5	102.0	102.0
54.	-	103.0	102.6	-	102.2	102.7	102.2	102.0	101.9	101.8	102.0
55.	-	103.0	103.0	-	103.0	102.6	102.0	102.1	102.0	102.0	102.1
56.	-	103.0	103.0	-	103.1	102.6	102.0	101.7	102.1	102.0	102.2
57.	-	103.1	103.3	-	103.0	-	102.0	102.2	102.2	101.4	102.2
58.	-	102.9	103.1	-	102.9	-	102.0	101.0	102.0	101.7	102.4
59.	-	100.4	102.8	-	103.0	-	102.1	102.1	102.3	102.0	100.5
60.	-	102.1	102.8	-	103.0	-	103.1	102.2	101.5	102.0	102.2
61.	-	100.3	102.8	-	103.1	-	102.9	103.1	102.0	101.7	1102.0

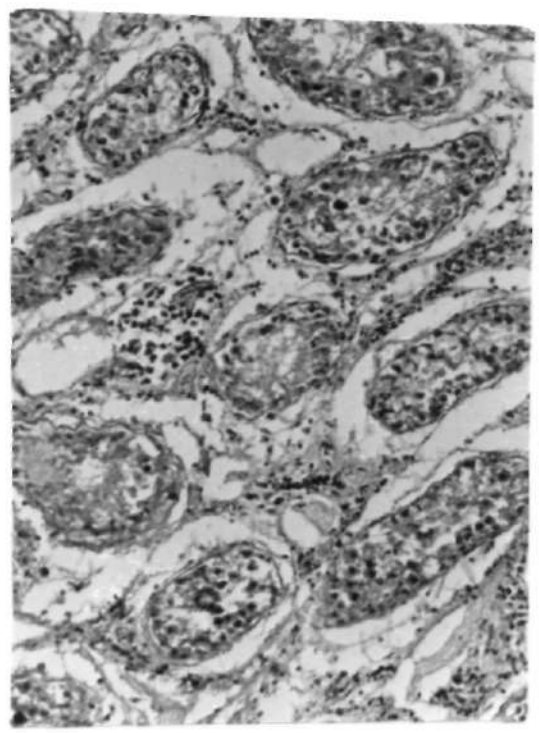


Fig 5: Testis showing severe testicular degeneration of both tubular and interstitial cells due to experimental C. pyogenes infection. x 450.

Discussion

Rectal temperatures rose by day 5 P.I. reaching a peak by day 12. Virtually all microorganisms have the propensity to induce pyrexia (Youmans et al., 1975). Intravenous injection of microorganisms stimulates phagocytosis and the resulting phagocytic target host cells (leukocytes) produce endogenous pyrogen into the blood stream. Youmans et al. (1975) reported that this endotoxin pyrogen acts on the central nervous system (CNS), alters the chemoregulatory activity of the hypothalamus-diencephalon and causes a rise in body temperature. Most infectious diseases follow a febrile course. Clinical experience has shown that recording the temperature is the easiest and most objective means of recognising an infectious process and evaluating its severity and duration as shown in the present study.

The hemogram indicates varying degrees of leukocytosis in all infected groups of animals, as exhibited by neutrophilia mainly. In general the clinicopathological results were in close agreement with the results of other investigators (Holman, 1945; Reda and Hathout, 1951; Smith et al., 1972). Leukocytosis is in response to invasions by pyogenic bacteria (Ham, 1965; Wintrobe, 1965). Practical use is made of this fact by determining the total and differential white cell counts of the blood in the diagnosis of suspected infections as done in the present study. It was observed

that some polymorphs had marked azurophilic granules in their cytoplasm. This observation was in conformity with the report of Ham (1965). It is reported that such "toxic granulations" appear in the blood during systemic infections (Ham, 1965). The activities of the observed leukocytes were basically phagocytic and this represented a non-specific method of coping with the bacterial invasion (Ham, 1965; Wintrobe, 1965; Smith et al., 1972).

The infection was more severe and of more prolonged duration in the stressed bulls than in those not stressed. This was mainly due to the fact that the stress-induced agent (Dexamethazone) employed was anti-inflammatory and reduced resistance to infectious agents (Youmans et al., 1975). This accentuated and prolonged the effect of the C. pyogenes infection in the affected animals. Ham, (1965) and Wintrobe, (1965) reported that corticosteroids reduced chemotaxis, and are antiphagocytic. They stated that, in large quantities, these agents actually cause a breakdown of lymphocytic tissues, leading to lymphopenia, stress and decreased resistance to infection. This is reflected in the present study.

Opinion differs as to the role of C. pyogenes infections in bull infertility. But the organisms have been incriminated as a cause of seminovesiculitis, testicular degeneration, epididymitis and orchitis (McEntee, 1962; Blom and Christensen, 1965; Carroll et al., 1968; Roberts, 1971).

Various degrees of severity of inflammation and degeneration of the genitalia in the experimentally infected bulls were observed. The general clinical manifestations observed agreed only in part with the observations of Cowie (1962). He observed oculonasal discharges, cachexia, coughing, anorexia and staggered gait in the infected animals; and petechiation of tissues, pneumonia and necrosis of liver, among other things, at post-mortem. This partial discrepancy might be due partly to the infective dose and pathogenicity of C. pyogenes, the resistance of the breed of bulls involved, the degree and duration of infection and some unidentified environmental and/or endogenous factors. However the histopathological manifestations of the varying degrees of inflammation and degeneration were in conformity with the descriptions of Lagerlof (1939), Blom and Christenser. (1972) and Humphrey and Ladds, (1975).

Testicular tissues are metabolically active, undergoing continuous spermatogenic and steroidogenic activities. These properties render them susceptible to injury (Cole and Cupps, 1977). In the C. pyogenes infected bulls there was elevation of body temperature ranging from 103.0 to 105.6^{o}F. It is conceivable that this persistent pyrexia might contribute to epididymal and testicular cell damage as reported by Payne (1956) in mice and Watson (1964) in rams. It is also inevitable that the damage to the genitalia could be due to direct effect of the organisms and/or their toxins.

Genital inflammation in the bull has been reported to be caused by such potentially pathogenic organisms as C. pyogenes, E. coli and Mycoplasma, in the absence of Brucella abortus (Blom and Dam, 1964; Blom and Erno, 1967; 1970). The stress-induced, infected bulls continually lost weight despite fairly good appetite. This observation tends to suggest infection-induced malnutrition. Changes in reproductive organs in malnutrition have been reported (Dutt, 1959; Roberts 1971).

The highest percentage of spermatogenesis (96%) was recorded in the control bulls and the lowest (66%) in the stress-induced, infected bulls. Sperm abnormalities were highest in the stressed infected and lowest in the control bulls. This trend continued with time in the infection. Lagerlof, (1934) and Knudsen (1954) reported that any injury to the spermatogenic epithelium nearly always results in lowered fertility which could be recognised by the presence of excessive number of morphologically abnormal sperms in the semen. In an earlier study testicular degeneration was found to decrease the rate of sperm production (Kumi-Diaka, 1976). The epididymal lesion observed in the C. pyogenes infected bulls could also contribute to the sperm abnormality. Lagerlof, (1934) and Gustafson (1964, 1965) reported that epididymal dysfunction gave rise to a lowering of sperm vitality, certain sperm tail defects and detachment of sperm heads.

It is known that C. pyogenes and other bacterial organisms may be isolated from the genitalia of apparently normal animals (Bruner and Gillespie, 1973; Hancock and Kelly, 1948). The present study also tallies with the previous findings. Low grade infections had no clinical significance on healthy well-fed animals. However the results of the present investigation strongly indicate that stress (induced or spontaneous) and other factors that significantly reduce the resistance of the animal can be major factors in C. pyogenes infections.

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A P P E N D I X

TABLES 1 - 7

Table 1: (Cont.)

	Bull Tag Nos.										
DAYS	Stress-infected bulls					Non-stress infected bulls			Control bulls		
PI*	1594	1643	1646	1650	1676	237	246	247	233	255	277
62.	-	101.1	102.6	-	103.3	-	102.7	102.2	102.1	101.7	102.2
63.	-	104.8	103.1	-	103.2	-	102.8	102.2	102.2	101.9	101.9
64.	-	-	102.7	-	103.4	-	102.8	103.0	102.4	102.3	102.0
65-70	-	-	102.2	-	102.2	-	102.5	102.8	101.8	100.9	102.4
71-78	-	-	-	-	102.8	-	102.1	102.3	102.0	102.4	-
79-84	-	-	-	-	102.1	-	102.5	102.0	102.3	-	-

* Post inoculation.

Table 2: Clinical findings in bulls experimentally infected with C.pyogenes

Bulls	Weeks PI*	Physical condition of bulls	Testes		Epididymis		Seminal vesicle		Ampulla		Semen
			Left	Right	Left	Right	Left	Right	Left	Right	
Stressed											
1643	3-6	Fair	S	S	N	N	N	N	N	N	NA
1646	3-4	Fair	S	N	N	N	N	N	N	N	NC
1650	3-4	Poor	S	S	S	N	S	S	N	N	NA
1594	3-4	Fair	Se	N	Se	N	N	N	N	N	NA
1676	3-4	Fair	N	N	N	N	N	N	N	N	NC
Non-Stressed:											
247	3-4	Fair	N	N	N	N	N	N	N	N	NC
237	3-4	Fair	N	Se	Se	Se	S	S	N	N	NC
246	3-4	Fair	N	N	N	N	N	N	N	N	NA
Controls:											
277	3-4	Good	N	N	N	N	N	N	N	N	NC
233	3-4	Good	N	N	N	N	N	N	N	N	NC
255	3-4	Good	N	N	N	N	N	N	N	N	NA

*PI = Post infection:
N = Normal
S = Swollen
NC = Normal color-creamy to milky
NA = Not available
Se = Sensitive.

Table 2 (Cont.)

Bulls	Weeks PI*	Physical condition of bull	Testes		Epididymis		Seminal vesicle		Ampulla		Semen
			Left	Right	Left	Right	Left	Right	Left	Right	
Stressed:											
1643	7-8	Poor	S	S	N	N	N	N	N	N	NA
1646	5-9	Poor	S	S	Se	N	N	N	N	N	NC
1650	5-6	Poor	S	S	S	S	N	N	N	N	NA
1594	5	Poor	S	N	Se	N	N	N	N	N	NA
1676	5-11	Fair	N	N	Fi	N	S	S	N	N	NC
Non-Stressed:											
247	5-9	Fair	Se	Se	N	N	Se	N	N	N	NC
237	5-7	Good	Se	Se	N	N	S	S	N	N	NC
246	5-11	Good	N	N	Soft	N	RL	N	N	N	NC
Controls:											
277	5-9	Good	N	N	N	N	N	N	N	N	NC
233	5-10	Good	N	N	N	N	N	N	N	N	NC
255	5-11	Good	N	N	N	N	N	N	N	N	NA

*PI = Post infection: N = Normal Fi = Fibrotic S = Swollen RL = Reduced lobulation.

Table 2: (Cont.)

Bulls	Weeks PI*	Physical condition of bull	Testes		Epididymis		Seminal vesicle		Ampulla		Semen
			Left	Right	Left	Right	Left	Right	Left	Right	
Stressed:											
1643	9	Poor	F	F	F	N	N	N	N	N	NA
1646	10	Poor	F	F	F	N	N	N	N	N	NC
1650	7	Poor	Soft	S	F	F	N	RL	N	N	NA
1594	6	Poor	F	F	S	N	N	N	N	N	NA
1676	12	Pair	N	N	F	N	N	S	N	N	NC
Non-Stressed:											
247	10	Good	N	Soft	N	N	S	S	N	N	NC
237	8	Fair/Good	N	N	N	N	RL	RL	N	N	NC
246	12	Good	N	N	N	F	RL	N	N	N	NA
Controls:											
277	10	Good	N	N	N	N	N	N	N	N	NC
233	11	Good	N	N	N	N	N	N	N	N	NC
255	12	Good	N	N	N	N	N	N	N	N	NA

*PI = Post infection: N = Normal RL = Reduced lobulation

F = Firm S = Swollen NC = Normal colour NA = Not available.

Table 3: Hemogram of bulls 3rd-4th week pre-inoculation with C.pyogenes.

Parameters	Bull tag nos.										
	Stress - infected bulls					Non-stress infected			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	8700	10500	8600	10000	10300	13500	6100	7500	8570	8500	9710
Hemoglobin (Hb) Gm/dl	10.9	12.1	12.1	9.2	10.8	11.0	10.6	12	9.6	11	11.8
Hematocrit (PCV) %	34	32	34	29	32	31	32	36	31	31	35
W.B.C. Differential %											
Basophils	0	0	0	0	0	0	0	0	0	0	0
Juveniles	0	0	0	0	0	0	0	0	0	0	0
Bands	0	0	0	0	0	0	0	0	0	0	0
Segments	47	48	45	53	42	20	25	26	29	38	35
Lymphocytes	27	38	37	38	43	70	70	62	60	49	48
Monocytes	2	11	2	0	2	1	1	0	1	1	2
Eosinophils	24	13	14	9	13	9	4	11	10	12	15

Table 4: Hemogram of 1st week bulls post-inoculation with C.pyogenes.

Parameters	Bull tag nos.										
	Stress - infected bulls					Non-stress infected				Control bulls	
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	14700	13400	12150	14500	13300	9850	13500	11710	9500	8800	10500
Hemoglobin (Hb) Gm/dl	8.7	7.5	12.3	9.7	7.1	10.1	10.6	9.7	11	10.1	10.1
Hematrocrit (PVC) %	27	24	30	28	30	29	30	31	34	31	30
W.B.C. Differential %											
Basophils	0	0	0	0	0	0	0	0	0	0	0
Juveniles	0	0	0	0	0	0	0	0	0	0	0
Bands	0	0	0	0	0	0	0	0	0	0	0
Segments	51	59	47	53	48	31	40	33	28	30	27
Lymphocytes	36	33	43	39	44	62	50	61	56	52	61
Monocytes	2	1	1	1	1	1	1	1	1	1	1
Eosinophils	11	7	9	7	7	6	9	5	15	17	11

Table 4 (Cont.)

2nd Week post inoculation

Parameters	Bull tag nos.										
	Stress - infected bulls					Non-stress infected			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	15300	15500	14700	14900	19400	13800	18500	18700	8300	10800	10810
Hemoglobin (Hb) Gm/dl	7.5	6.6	8.8	8.9	7.1	10	7.5	8.1	10.5	10.9	11.3
Hematocrit (PCV) %	24	28	24	31	28	30	28	25	31	32	31
W.B.C. Differentials %											
Basophils	0	0	0	0	0	0	0	0	0	0	0
Juveniles	0	0	0	0	0	0	0	0	0	0	0
Bands	0	0	0	0	0	0	0	0	0	0	0
Segments	56	59	54	49	60	54	64	46	28	30	30
Lymphocytes	40	35	42	46	36	45	34	47	56	50	55
Monocytes	2	1	0	1	2	1	1	3	1	2	1
Eosinophils	2	7	4	4	2	0	1	4	15	18	14

Table 4 (cont.)

Parameters	3rd Week post-inoculation										
	Bull tag nos										
	Stress-infected bulls					Non-stress infected			Control bulls		
	1594	1643	1646	1650	1676	237	247	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	16900	16300	15900	13100	19100	17300	16500	17000	8700	10000	9700
Hemoglobin (Hb) Gm/dl	7.5	6.7	7.4	8.1	7.1	8.1	7.5	9.0	9.4	10.4	10.2
Hematocrit (PCV) %	24	27	24	27	28	31	30	25	30	30	32
W.B.C. Differentials %											
Basophils	0	0	0	0	0	0	0	0	0	0	0
Juveniles	0	0	0	0	0	0	0	0	0	0	0
Bands	0	0	0	0	0	0	0	0	0	0	0
Segments	57	64	53	49	57	56	58	51	35	34	35
Lymphocytes	42	33	44	45	42	43	40	40	52	51	46
Monocytes	1	0	2	1	1	1	0	1	1	2	2
Eosinophils	0	3	1	5	0	0	2	8	12	13	17

Table 4 (cont.)

4th Week post-inoculation

Parameters	Bull tag nos.										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	16400	16100	15000	16300	18500	17500	16800	15000	9000	9800	9515
Hemoglobin (Hb) Gm/dl	7.0	6.5	9.5	8.0	7.5	8	8.4	9.5	9.7	10.2	10.1
Hematocrit (PCV) %	18	20	23	25	22	27	30	28	30	35	29
W.B.C. Differentials %											
Basophils	0	0	0	0	0	0	0	0	0	0	0
Juveniles	0	0	0	0	0	0	0	0	0	0	0
Bands	0	0	0	0	0	0	0	0	0	0	0
Segments	54	58	50	57	54	50	60	45	30	30	33
Lymphocytes	45	41	45	41	44	48	38	49	60	50	51
Monocytes	1	1	1	2	2	2	1	0	1	2	1
Eosinophils	0	0	4	0	0	0	1	6	9	18	15

Table 4 (cont.)

5th Week post-inoculation

Parameters	Bull tag nos.										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	14800	14510	15300	15400	17000	13500	15510	16000	7200	9000	9510
Hemoglobin (Hb) Gm/dl	7.0	5.5	10.1	8.0	7.0	9.0	9.1	8.4	10.0	10.0	10.9
Hematocrit (PCV) %	19	20	22	25	23	27	31	29	30	31	34
W.B.C. Differentials %											
Basophils	0	0	0	0	0	0	0	0	0	0	0
Juveniles	0	0	0	0	0	0	0	0	0	0	0
Bands	0	0	0	0	0	0	0	0	0	0	0
Segments	48	57	51	55	48	38	51	40	31	35	36
Lymphocytes	50	42	48	43	49	58	48	58	54	45	45
Monocytes	20	1	1	1	2	1	1	0	1	0	1
Eosinophils	0	0	0	1	1	3	0	2	14	20	18

Table 4 (cont.)

Parameters	6th Week post-inoculation Bull Tag Nos.										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	15800	11600	13700	11750	17500	11900	13300	11030	10500	9600	9300
Hemoglobin (Hb) Gm/dl	6.8	4.8	7.8	8.5	7.8	12.6	10.6	10.4	10.3	9.4	10.0
Hematocrit (PCV) %	19	16	22	19	21	30	22	31	35	30	30
W.B.C. Differentials %											
Basophils	0	0	0	0	0	0	0	0	0	0	0
Juveniles	0	0	0	0	0	0	0	0	0	0	0
Bands	0	0	0	0	0	0	0	0	0	0	0
Segments	41	37	42	36	50	28	33	34	38	37	32
Lymphocytes	58	59	56	60	49	60	63	54	51	44	52
Monocytes	1	1	2	0	0	1	1	2	2	1	1
Eosinophils	0	3	0	4	1	11	3	10	9	18	15

Table 4 (cont.)

7th Week post-inoculation

Parameters	Bull tag nos.										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	-	8600	10200	11000	16800	11600	9400	10500	8900	9500	11000
Hemoglobin (Hb) Gm/dl	-	5.8	8.3	8.5	8.1	12.0	10.7	8.1	11.4	11.9	10.1
Hematocrit (PCV) %	-	18	20	20	21	28	30	29	29	31	30
W.B.C. Differentials %											
Basophils	-	0	0	0	0	0	0	0	0	0	0
Juveniles	-	0	0	0	0	0	0	0	0	0	0
Bands	-	0	0	0	0	0	0	0	0	0	0
Segments	-	39	33	36	50	21	25	24	38	32	31
Lymphocytes	-	56	66	63	46	67	62	55	51	56	51
Monocytes	-	1	1	1	1	2	0	2	2	2	2
Eosinophils	-	4	0	0	3	10	13	19	9	10	16

Table 4 (cont.)

8th Week post-inoculation

Parameters	Bull Tag Nos										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	-	10300	11500	-	13500	9900	10210	11500	7800	6500	9800
Hemoglobin (Hb) Gm/dl	-	6.1	10.4	-	8.1	10.1	10.0	9.3	7.5	11.0	9.9
Hematocrit (PCV) %	-	21	19	-	25	32	29	33	25	33	30
W.B.C. Differentials %											
Basophils	-	0	0	-	0	0	0	0	0	0	0
Juveniles	-	0	0	-	0	0	0	0	0	0	0
Bands	-	0	0	-	0	0	0	0	0	0	0
Segments	-	38	35	-	39	21	27	31	29	31	34
Lymphocytes	-	60	61	-	55	68	61	53	55	49	48
Monocytes	-	1	1	-	1	2	0	2	1	1	2
Eosinophils	-	1	3	-	5	9	12	14	15	19	16

Table 4 (cont.)

9th Week post-inoculation

Parameters	Bull tag nos										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	-	8900	9870	-	11600	-	10800	11000	8150	8700	12500
Hemoglobin (Hb) Gm/dl	-	8.6	12.0	-	8.0	-	10.0	8.5	7.5	10.4	10.4
Hematocrit (PCV) %	-	21	21	-	28	-	31	30	29	31	31
W.B.C. Differentials %											
Basophils	-	0	0	-	0	-	0	0	0	0	0
Juveniles	-	0	0	-	0	-	0	0	0	0	0
Bands	-	0	0	-	0	-	0	0	0	0	0
Segments	-	30	32	-	35	-	36	28	34	31	25
Lymphocytes	-	60	55	-	55	-	60	61	54	53	60
Monocytes	-	2	2	-	1	-	1	2	1	2	1
Eosinophils	-	8	10	-	9	-	3	9	11	14	14

Table 4 (cont.)

10th Week post-inoculation

Parameters	Bull tag nos.										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	-	-	10400	-	11500	-	9580	10800	7510	8500	8300
Hemoglobin (Hb) Gm/dl	-	-	9.3	-	9.8	-	9.4	10.0	10.8	11.0	12.6
Hematocrit (PCV) %	-	-	24	-	26	-	28	29	32	34	27
W.B.C. Differentials %											
Basophils	-	-	0	-	0	-	0	0	0	0	0
Juveniles	-	-	0	-	0	-	0	0	0	0	0
Bands	-	-	0	-	0	-	0	0	0	0	0
Segments	-	-	29	-	23	-	20	23	31	26	23
Lymphocytes	-	-	61	-	65	-	73	66	54	56	58
Monocytes	-	-	2	-	2	-	2	1	2	1	2
Eosinophils	-	-	8	-	10	-	5	10	13	17	17

Table 4 (cont.)

11th Week post-inoculation

Parameters	Bull tag nos.										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	-	-	-	-	10500	-	10300	-	8500	7900	-
Hemoglobin (Hb) Gm/dl	-	-	-	-	9.4	-	9.6	-	9.1	8.6	-
Hematocrit (PCV) %	-	-	-	-	24	-	30	-	31	29	-
W.B.C. Differentials %											
Basophils	-	-	-	-	0	-	0	-	0	0	-
Juveniles	-	-	-	-	0	-	0	-	0	0	-
Bands	-	-	-	-	0	-	0	-	0	0	-
Segments	-	-	-	-	30	-	27	-	30	32	-
Lymphocytes	-	-	-	-	62	-	66	-	59	51	-
Monocytes	-	-	-	-	1	-	3	-	1	0	-
Eosinophils	-	-	-	-	7	-	4	-	10	17	-

Table 4 (cont.)

12th Week post-inoculation

Parameters	Bull Tag Nos										
	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
R.B.C. x 10^6/ul	-	-	-	-	-	-	-	-	-	-	-
W.B.C. Total	-	-	-	-	10900	-	10000	-	-	-	-
Hemoglobin (Hb) Gm/dl	-	-	-	-	11.9	-	10.6	-	-	-	-
Hematocrit (PCV) %	-	-	-	-	29	-	30	-	-	-	-
W.B.C. Differentials %											
Basophils	-	-	-	-	0	-	0	-	-	-	-
Juveniles	-	-	-	-	0	-	0	-	-	-	-
Bands	-	-	-	-	0	-	0	-	-	-	-
Segments	-	-	-	-	21	-	23	-	-	-	-
Lymphocytes	-	-	-	-	71	-	71	-	-	-	-
Monocytes	-	-	-	-	0	-	1	-	-	-	-
Eosinophils	-	-	-	-	8	-	5	-	-	-	-

Table 5: Gross post-mortem findings in the genitalia of bulls experimentally infected with C. pyogenes

Findings	Bull tag nos.										
	Stress-infected bulls					Non-stress infected			Control bulls		
	1643	1646	1650	1594	1676	247	237	246	277	255	233
Physical condition											
Good	-	-	-	-	-	+	-	+	+	+	+
Fair	-	-	-	-	+	-	+	-	-	-	-
Poor	+	+	+	+	-	-	-	-	-	-	-
Serous atrophy of fat	+	+	-	-	+	-	-	-	-	-	-
Icterus	-	-	-	-	-	-	-	-	-	-	-
Abscess	-	-	-	-	-	-	-	-	-	-	-
Testes											
Enlarged	-	-	+B	+B	-	-	-	-	-	-	-
Adhesions	+B	+B	+B	+B	+B	-	+B	+B	-	-	-
Atrophy	-	-	-	-	-	-	-	-	-	-	-
Fibrosis	+B	+B	-	+L	+B	-	-	-	-	-	-
Hemorrhagic	-	-	-	+L	-	-	-	-	-	-	-
Abscess	-	-	-	-	-	-	-	-	-	-	-
Epididymis											
Enlarged	-	-	+B	+L	+L	-	-	-	-	-	-
Adhesions	+B	+B	+B	+B	+B	-	+R	-	-	-	+R
Hemorrhagic	-	-	-	+L	-	-	-	-	-	-	-

B = Both; L = Left; R = Right.

Table 5 (Cont.)

Findings	Bull tag n'os.										
	Stress-infected bulls					Non-stress infected			Control bulls		
	1643	1646	1650	1594	1676	247	237	246	277	255	233
Epididymis											
Abscess	-	-	-	-	+L	-	-	-	-	-	-
Fibrosis	+L	-	-	+L	-	-	-	-	-	-	-
Seminal vesicles											
Enlarged	-	-	+B	-	-	-	+R	-	-	-	-
Lobulations :- reduced	-	-	+B	-	-	-	+R	-	-	-	-
loss (smooth)	-	-	-	-	-	-	-	-	-	-	-
Abscessation	-	-	-	-	+R	+B	-	-	-	-	-
Fibrosis	-	-	-	-	-	-	-	-	-	-	-
Ampullae											
Enlarged	-	-	-	-	-	-	-	-	-	-	-
Fibrosis	-	-	-	-	-	-	-	-	-	-	-
Abscess	-	-	-	-	-	-	-	-	-	-	-
Inguinal lymph node											
Enlarged/Edematous	-	-	+	+	-	-	-	-	-	-	-
Abscess	-	-	-	-	-	-	-	-	-	-	-

B = Both; L = Left; R = Right.

Table 6: Isolation of C.pyogenes from various organs from bull calves experimentally infected with the organism.

Organs	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1643	1646	1650	1594	1676	247	237	246	277	255	233
Blood	-	-	-	-	-	-	-	-	-	-	-
Testis - R	+	+	+	+	-	-	+	-	-	-	-
- L	+	+	+	+	+	-	-	-	-	-	-
Epididymis - R	+	+	+	+	+	-	-	-	-	-	-
- L	+	+	+	+	+	-	+	-	-	-	-
Ductus deferens - R	-	+	+	+	-	-	-	-	-	-	-
- L	-	-	+	+	-	-	-	-	-	-	-
Prostate	-	-	-	-	-	-	-	-	-	-	-
S/vesicle - R	-	+	+	-	+	+	-	+	-	-	-
- L	+	+	+	-	+	-	-	+	+	-	-
Ampulla - R	-	+	-	-	-	-	-	-	-	-	-
- L	-	-	-	-	-	-	-	-	-	-	-
Urinary bladder	+	+	-	-	+	-	-	-	-	-	-
Kidney	-	-	-	-	+	-	-	-	-	-	-
Inguinal lymph node	-	-	+	+	-	-	-	-		-	-
Semen	NA	+	NA	NA	+	-	-	NA	-	-	-
Preputial cavity	+	+	-	-	-	+	-	-	-	-	+

NA = Not available; Semen could not be collected.

R = Right; L = Left

Table 7: Histopathological findings in organs from bulls experimentally infected with C.pyogenes organisms.

Findings	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
TESTIS											
Neutrophils	+	+	-	+	-	+	-	-	-	-	-
Hemorrhage	+	-	-	-	-	-	-	-	-	-	-
Lymphocytes	+	++	+++	+	+	+	+	+	-	-	-
Monocytes	-	-	+	+	-	-	-	+	-	-	-
Plasma cells	++	+++	++	+	++	+	+	+	-	-	-
Intertubular Fibrosis	+	++	-	+	+	+	-	-	-	-	-
Leydig cells hyperplasia	-	-	-	-	-	-	-	-	-	-	-
Seminiferous tubule:-											
-pyknosis	++	+	+	++	+	+	-	+	-	-	-
-Karyolysis	++	+	+	+	+	+	-	+	-	-	-
-Desquamation	+	+	-	+++	++	+	+	-	-	+	-
-Detachment of basement membrane	++	+	+	++	++	+	-	-	+	-	-
EPIDIDYMIS											
Neutrophils	+	-	-	+	-	-	-	-	-	-	-
Hemorrhage	-	-	-	-	-	-	-	-	-	-	-
Lymphocytes	++	+	++	++	++	+	+	-	-	-	-

Table 7 (cont.)

Findings	Stress-infected bulls				Non-stress infected bulls			Control bulls			
	1594	1643	1646	1650	1676	237	246	247	233	255	277
Monocytes	+	-	-	++	+	-	-	-	-	-	-
Plasma cells	++	+	++	+	+	+	+	-	-	-	-
Fibrosis	+	+	+	+	++	+	-	+	-	-	-
Desquamation of cells	-	-	-	-	+	++	-	-	-	-	-
Atrophy of tubular epithelium	-	-	-	-	-	-	-	-	-	-	-
Degeneration	+	++	+	-	++	+	-	-	-	-	-
PROSTATE											
Neutrophils	-	-	-	-	-	-	-	-	-	-	-
Lymphocytes	+	+	-	-	-	-	-	-	-	-	-
Macrophages	-	-	-	-	-	-	-	-	-	-	-
Fibrosis	-	-	+	-	-	-	-	-	-	-	-
Degeneration	-	-	-	-	-	-	-	-	-	-	-
SEMINAL VESICLES:											
Neutrophils	-	-	-	+	-	+	-	-	-	-	-
Lymphocytes	-	-	+	++	+	+	-	+	-	-	-
Plasma cells	-	-	+	+	+	+	+	+	-	-	-
Abscessation	-	-	-	-	-	-	-	+	-	-	-

Table 7 (cont.)

Findings	Stress-infected bulls					Non-stress infected bulls			Control bulls		
	1594	1643	1646	1650	1676	237	246	247	233	255	277
Fibrosis	-	-	-	+	+	-	+	+	-	-	-
Metaplasia	-	-	-	-	-	-	-	-	-	-	-
AMPULLAE											
Neutrophils	-	-	±	-	-	-	-	-	-	-	-
Macrophages	-	-	-	-	-	-	-	-	-	-	-
Lymphocytes	-	-	-	-	-	-	-	-	-	-	-
Fibrosis	-	-	-	-	-	-	+	-	-	-	-
Degeneration	-	-	-	-	-	-	-	-	-	-	-
PELVIC URETHRAE											
Inflammatory cells	-	-	-	-	-	-	-	-	-	-	-
Fibrosis	-	-	-	-	-	-	-	-	-	-	-
PENIS (C/S)											
Inflammation	-	-	-	-	-	-	-	-	-	-	-
Fibrosis	-	-	-	-	-	-	-	-	-	-	-

Key: + = Mild/few
++ = Moderate
+++ Severe

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INFERTILITY AND GENITAL PATHOLOGY OF BULLS INDIGENOUS TO NIGERIA

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Animals play a major role as a source of protein in the world. It is reported that the most important and widespread nutritional deficiency in the world is protein. Food shortage will inevitably become more critical and the protein shortage more acute as the world population increases.

The number of livestock in Nigeria is adequate and matches that of most developed countries and yet animal protein is at its lowest. Among the causes of inadequate supply are low reproductive performance and poor nomadic husbandry practices. Several factors such as inadequate nutrition, seasons and diseases such as Corynebacterium pyogenes infections, influence the reproductive performance of the animals. Although the literature is full of information on the female animal there is very little information on the reproductive performance of bulls indigenous to Nigeris. The present study was initiated to study the spermatogenesis and clinico-pathology and bacteriology of the indigenous bovine male genitalia; and the significance of the latter on the reproductive performance of the bull.

In a clinico-pathologic and bacteriologic study of the scrotal contents of 4730 indigenous bulls in Northern Nigeria 152 (3.21%) gross pathologic conditions were observed. These included: testicular degeneration (0.59%), testicular hypoplasia (0.66%), orchitis (0.44%), cryptorchidism (1.29%), epididymitis (0.08) and

spermatic granuloma (0.15%). Scrotal dermatophilosis of varying degrees of severity was observed in 204 bulls, with most cases occurring during the rainy season. This condition did not appear to significantly affect spermatogenesis.

The result of histopathologic study was significant. Severe testicular degeneration affected spermatogenesis significantly. Histopathologic lesions and their prevalence were: testicular degeneration, 18.60%; hypoplasia, 5.30%; orchitis, 8.10%; epididymitis, 1.40%. Testicualr degeneration was observed more commonly in the middle-aged and older bulls (6-10 years). Bacterial organisms isolated from some of the lesions were; Corynebacterium pyogenes, Staphylococcus spp. and Brucella abortus. In addition to hereditary factor, age, trauma and trypanosomiasis were also identified as significant factors in the etiology of testicular and epididymal pathology.

Spermatogenesis in apparently normal and grossly abnormal testes, and the significance of sertoli cell index (SCI), as a measure of testicular degeneration, was studied in 264 indigenous bulls of Nigeria. The means of the testicular weight and dimensions and the seminiferous tubular diameter were lower than reported for the Bos indicus breed. It was observed that the peripheral layer of the seminiferous tubules was composed of

germinal cells, a greater number of which were spermatogonia, at rest or in mitosis, and the sertoli cells. Other germinal cells - spermatocytes, spermatids and spermatozoa - were identified.

The average monthly diameter of the seminiferous tubules was 222.7 ± 12 microns. The sertoli cells averaged 13.0 ± 3.0 per tubule and spermatogonia - A and B cells averaged 3.3 ± 2.0 and 5.4 ± 3.9 per tubule respectively. The more centrally placed primary spermatocytes had a monthly average of 41.0 ± 20.2 per tubule. The round and elongated spermatids averaged 41.0 ± 9.8 and 35.0 ± 8.3 per tubule respectively. The monthly average percentage spermatogenesis in normal and slightly degenerated testes was 77.6 ± 3.1%. Moderate and severe degenerations averaged 59.7 ± 8.4% and 31.1 ± 11.0% spermatogenesis respectively.

The difference in the number of germinal epithelial cells in the normal and abnormal testes was reflected in the SCI. The average SCI of normal testes was 11.5. The average was 11.0 for slightly degenerated testes. The moderately degenerated testes had an average SCI of 8.8 and the severely degenerated testes scored an average SCI of 5.4. In general it was observed that severe degeneration influenced spermatogenesis. This was reflected in the decrease in SCI with severity of degeneration.

The clinico-pathological findings in a study of the accessory genitalia, prepuce and penis of indigenous bulls are reported.

With regard to the external genitalia, preputial prolapse (0.4%) was the most common clinical condition observed. Balanoposthitis occurred in 6 bulls (0.13%); phimosis in 7 bulls (0.15%); paraphimosis in 2 bulls (0.04%); penile fracture in 8 bulls (0.17%); penile deviations in 4 bulls; hematoma in 9 bulls (0.19%) and adhesions in 11 bulls (0.23%). An overall 1.87% incidence of gross pathologic conditions was encountered.

The preputial cavity was the most consistent site for mixed bacterial flora which included; Escherichia coli, Corynebacterium pyogenes; Staphylococcus aureus; Streptococci spp and Proteus vulgaris. The significance of these in cases of balanoposthitis could not be ascertained in the present study, as most of them were isolated from bulls with apparently normal genitalia.

The most common histopathologic changes observed in the seminal vesicles were varying degrees of severity of inflammatory reactions. No gross or histopathologic lesions were observed in the prostate glands, pelvic urethrae and ampullae in all the bulls. It has been reported that seminal vesicles and bulbo-urethral glands tend to harbor infections because of their complicated structure and poor drainage.

The pathology of the bovine male genitalia experimentally infected with Corynebacterium pyogenes was studied.. Eight experimental and 4 control bull calves were used. Stress was

induced in 4 of the experimental bulls by giving dexamethazone injections intravenously, prior to and after inoculation with C. pygenes suspension. All the 8 experimental bulls were inoculated intravenously with different dosage levels of living C. pyogenes organisms suspended in sterile PBS (physiological buffered saline). The control bull calves were inoculated with formalin-killed antigen.

Rectal temperatures of the experimental animals rose from 102°F to 105°F, 4 to 5 days post-infection (P.I). Physical conditions of the animals ranged from fair to good in the non-stressed, infected group and control group, to cachexia in the stressed infected group. At post-mortem, varying degrees of inflammation were observed in the testes, epididymes, and seminal vesicles in the stressed infected group. No gross lesions were found in the control group. Bacteriologically, pure culture of C. pyogenes was isolated from different parts of the genitalia of the infected animals. Few colonies of mixed flora were isolated from the genitalia of the control animals.

Histological changes observed in the genitalia included inflammation and degeneration. The testes and epididymes were most affected and the prostate and ampullae the least affected. The spermiogram indicated greater sperm morphologic abnormalities in the stressed infected animals than in the non-infected control

bulls. The highest percentage spermatogenesis was recorded in the control bulls and the lowest in the stress-induced infected bulls.

This study indicated that C. pyogenes infections could play a major role in genital pathology and any factor that may significantly reduce the resistance of the host could be a major factor in the severity of C. pyogenes infections/