Pathological findings in the bulbourethral glands of bulls

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SUMMARY: The bulbourethral glands of 323 *Bos indicus* or *B. indicus* crossbred bulls more than 1½ years old were examined in an abattoir study. Bulbourethral adenitis was diagnosed grossly and confirmed by histological examination in 4 (1.2%). Unilateral chronic interstitial inflammation was seen in 2 cases; one of these was associated with a degenerative-type seminal vesiculitis. In the others adenitis was bilateral; in one case it was associated with a concretion and foreign (plant) material in the principal duct of the left bulbourethral gland; in the other bilateral case, numerous calculi were present and microscopically, a chronic active and diffuse inflammation was observed. Chemical analysis of the calculi showed calcium oxalate and tricalcium phosphate to be the most important components. *Corynebacterium* spp was isolated from the lesion with multiple calculi but attempts to isolate *Chlamydia* spp, *Mycoplasma* spp and *Brucella abortus* from the 4 adenitis cases were unsuccessful. Congenital abnormalities such as glandular fusion (2.2%) or unilateral aplasia (0.6%) were also observed. Cysts were the most common finding (19.2%), and duct dilation was frequent (7.1%). The significance of these findings in relation to fertility is considered.

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Introduction

The gross anatomy and histology of the accessory sex glands (ASG) of bulls (ampullae, seminal vesicles, prostate, and bulbourethral glands) has been reviewed (Blom and Christensen 1947; Mann *et al* 1949; Trautmann and Fiebiger 1957; Trotter 1959; Banks 1981; Dellmann and Wrobel 1981).

The bulbourethral glands (BG) or Cowpers glands are paired and ovoid and are located on either side of the pelvic urethra cranial to the ischiatic arch. They are covered by a thick fibrous capsule and the bulbocavernous muscle (Blom and Christensen 1947; Dellmann and Wrobel 1981). At the time of ejaculation, secretions from the ASG blend into the seminal plasma which constitutes 64 to 70% of the ejaculate; 31 to 32% comes from the prostate and BG (Seidel and Foote 1970). The mucous-proteinaceous secretion produced by the BG clears the urethra during the ejaculatory stage (Banks 1981; Dellmann and Wrobel 1981) and it is the most important component of the first fraction of the ejaculate. Abnormalities of the BG may therefore compromise bull fertility.

There are few publications concerning the pathology of the BG (Weis 1964) and most described lesions are histological only (McEntee and Olafson 1953; Kanagawa 1961; Nobel *et al* 1961; Ball *et al* 1964; 1968; Carroll *et al* 1968; Todorovic and Petrovic 1985; Deschamps *et al* 1987). Grossly, bulbourethral adenitis was an uncommon finding in one abattoir survey (Bagshaw and Ladds 1974a) or was not seen (Blom and Christensen 1947).

The purpose of this paper is to report some unusual findings in the BG of bulls.

Materials and Methods

The BG of 323 *Bos indicus* or *B. indicus* crossbred bulls slaughtered at 2 abattoirs in Townsville, were collected and examined. The bulls originated from different herds and the reproductive histories were unknown. The approximate ages

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of bulls were estimated from the teeth at slaughter (Bellenger 1971; Ladds *et al* 1973) and bulls were grouped in 3 categories: young (1½ to 3½ years), adult (4 to 7 years) and old (>7 years).

Collection of specimens and gross examination were done as previously described (Bagshaw and Ladds 1974a; Ladds 1980). Because many BG were damaged at the abattoirs as a result of routine slaughtering procedures, they were discarded and only the intact glands were examined. All visible abnormalities were tabulated and where appropriate, blocks were fixed in Bouin's fluid for microscopy. Histological sections were stained with haematoxylin and eosin, and special stains when necessary. Glandular cavities greater than 4 mm in diameter were recorded as cysts and dilations of the excretory ducts greater than 2 mm in diameter were recorded as duct dilations (Bagshaw and Ladds 1974a). Congenital abnormalities were also recorded. Samples with gross pathology, different from that mentioned above, were collected for microbiological studies. Attempts were made to isolate aerobic and microaerophilic bacteria by standard procedures, *Brucella abortus* by selective media (Farrel 1974), *Mycoplasma* spp using solid media (Freund *et al* 1979) or broth (Etheridge *et al* 1979), *Chlamydia* spp by inoculation of embryonated eggs and cell culture in buffalo green monkey cells (Hobson *et al* 1982).

Using the SPSS program (Nie *et al* 1975), statistical analysis of some findings such as glandular fusion, cysts and duct dilation, were performed to determine any age and/or side effects.

Results

The principal findings in the BG of 323 bulls are summarised in Table 1. Normal glands were seen to be compact on the cut surface, grey-brown in colour and somewhat dry. Bulbourethral adenitis (BA) was grossly diagnosed and histologically confirmed in 4 bulls (1.2%); it was felt that 2 of these cases would have been clinically palpable by rectal examination. The ages of bulls with BA in 2 cases were 4 to 5 years and in the others >9 years old.

Attempts to isolate *Chlamydia* spp, *Mycoplasma* spp, and *Brucella abortus* from the 4 BA Cases were unsuccessful. A Gram positive Coryneiform bacillus was isolated from one sample. The organism produced small (0.6 mm) non-haemolytic grey opaque colonies on blood agar after 48 hr under aerobic incubation and was largely unreactive in a range of biochemical tests. On the basis of its colonial and cellular morphology and biochemical properties the organism was confined to the genus *Corynebacterium* (Cottral 1978).

The gross pathology and histological findings are given:

Case 1: The left BG in this 4-year-od bull was increased in size and on cross section a white-grey concretion, 3 x 5 mm in size, was attached to a filamentous black foreign body with an irregular surface. It was located in the glandular duct and was directed towards the urethral lumen. This foreign body measured 1 mm in width and 40 mm in length (Figure 1). The right BG contained a cyst, 5 x 6 mm in size. Microscopically, the most striking change was a severe periductal reaction with predominantly plasma cell infiltration, histiocytes and increased fibrous tissue. Hyperplastic epithelial change with scattered neutrophilic infiltration and subepithelial plasma cells were commonly found in ducts. Acinar compressive atrophy was also observed. The foreign body was attached to the wall of the duct; it transmitted polarized light and its structure was typical of a plant fibre (Figure 2). Less severe periductal interstitial inflammation was seen in the right BG; the cyst was lined by cuboidal epithelium. Severe chronic bilateral interstitial periductal bulbourethral adenitis was diagnosed.

Case 2: The left BG from a 9-year-old bull contained a grey focal area of fibrosis, 8 mm in diameter, associated with cysts. Histologically the left BG showed focal fibrosis, with many atrophic acini and a moderate accumulation of plasma cells and histiocytes. Some glandular acini were dilated with retained secretion in the lumen, which was lined by flat cuboidal

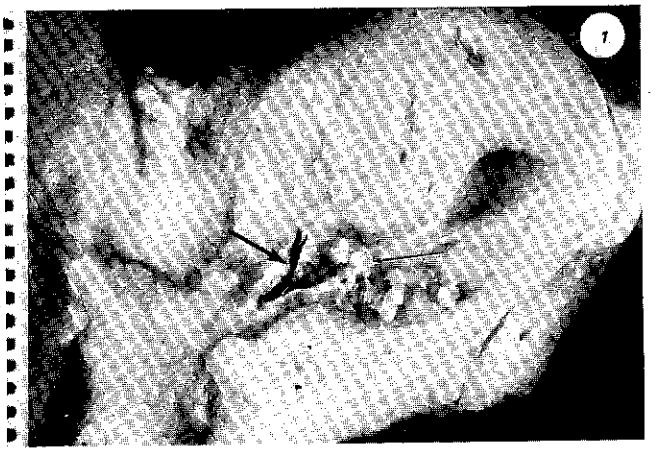

Figure 1. Plant material in duct of bulbourethral gland (large arrow). Note associated multiple soft concretions (small arrow) and duct dilation. Scale is in mm.

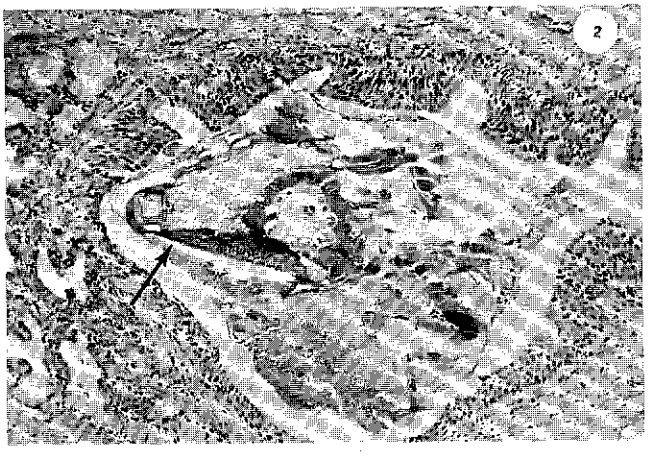

Figure 2. Photomicrograph of intraductal plant material (arrow) in bulbourethral gland shown in Figure 1. Some secretion is adherent to the foreign body and there is epithelial hyperplasia but little inflammation.

TABLE 1
Summary of lesions in the bulbourethral glands of 323 bulls

Item	Age Group (No.) Young (52)	Adult (196)	Old (64)	ND* (11)	Lesion Bilateral	Left	Right	(%) Totals 323
Adenitis	—	2	2	—	2	2	—	4 (1.2)
Lithiasis	—	—	2	—	1	—	1	2 (0.6)
Foreign body	—	—	1	—	—	1	—	1 (0.3)
Aplasia	—	1	1	—	—	—	2	2 (0.6)
Fusion	1[a]	5	1	—	—	—	—	7 (2.1)
Cysts	13[a]	32	14	2	39[b]	11	11	61 (19.2)
Duct dilation	2[a]	19	2	—	17[b]	3	3	23 (7.1)

*ND: Not determined
a: No significant differences ($P > 0.05$) were seen between age groups in the Chi-square test.
b: Significant differences ($P < 0.05$) in the Chi-square test were present between the bilateral, as compared with unilateral condition.

epithelium. The right BG showed some acinar degeneration with epithelial metaplasia, retained secretion and dilation; however, no inflammatory response was seen. In this case also, the seminal vesicles were affected by a degenerative inflammatory condition (Galloway 1964). The diagnosis was unilateral focal chronic bulbourethral adenitis associated with bilateral degnerative seminal vesiculitis.

Case 3: Both BG in this old (> 9 year) bull were increased in size. On the cut surface multiple cysts and bilateral calculi were obvious. Two conspicuous calculi in the right BG were white-grey in colour and had a rough-porous surface (Figure 3). Their weights and measurements were 4.2 g and 20 x 17 x 15 mm; and 1.7 g and 22 x 6 x 9 mm, respectively. Two other small calculi (2 x 3 mm) were also noted and a further 3 calculi (2 x 2 mm) were present in the left BG. Chemical analysis of calculi revealed their content as calcium oxalate (35%), and tricalcium phosphate (61%).

Histologically, severe periductal inflammation was noted with plasma cells especially, but also scattered histiocytes and neutrophils being present. Associated acinar atrophy and loss of normal glandular structure were features in many areas (Figure 4). Duct epithelium was sometimes hyperplastic and obvious papillary projections contained clumps of plasma cells. The cavity in which the calculi were located was lined by hyperplastic folded epithelium with intraluminal neutrophils. Subepithelial plasma cell accumulation was marked and the wall was surrounded by fibrous tissue. Intraluminal microconcretions, pink in colour, were also observed. PAS-positive retained secretion was found in some dilated acini which were lined by low cuboidal epithelium. Small Gram-positive bacilli which tended to clump or form chains were seen among inflammatory exudate in the lumens of some ducts. Chronic active diffuse bilateral bulbourethral adenitis associated with lithiasis was diagnosed.

Case 4: The left BG of this 5-year-old bull was pale in colour and soft in consistency. Its size was smaller than the opposite BG which contained a cyst 6 x 6 mm in size. Microscopically, the left BG had atrophic acini with low cuboidal epithelium, and periductal and interstitial fibrosis with scattered plasma cell and eosinophil infiltration. Focal mild left interstitial bulbourethral adenitis was diagnosed.

Other Cases: Developmental abnormalities were found in some cases. These included glandular fusion in 7 bulls (2.2%), and unilateral aplasia (right side) in 2 animals (0.6%); no statistical difference in the occurrence of these defects attributable to age could be demonstrated. Histologically, the glandular structures were normal except in one case in which multiple bilateral cysts were associated with glandular fusion. These cysts were lined by low cuboidal epithelium. Mucoproteinaceous material which distended the acini was noted and there was increased fibrous tissue in the interseptal wall.

Glandular cyst formation was the most common finding, being present in 61 bulls (19.2%) and again no statistical difference could be attributed to age. The condition was most frequently bilateral (Figure 5) ($P < 0.05$) and in unilateral cases there was about equal involvement of either side. Histologically, the cysts were lined by low cuboidal epithelium with PAS-positive secretion in the lumen. In some cysts, the wall was surrounded by increased fibrous tissue.

Duct dilation was observed in 23 bulls (7.1%) and again no age effect could be demonstrated. Such dilation was more often bilateral than unilateral ($P < 0.05$) but the frequency of dilation in either case was the same. Histologically, normal high columnar or transitional epithelium lined the ducts.

Figure 3. Cross section of enlarged bulbourethral gland in Case 3. Note large and small concretions (arrows), which microscopically were associated with chronic inflammation.

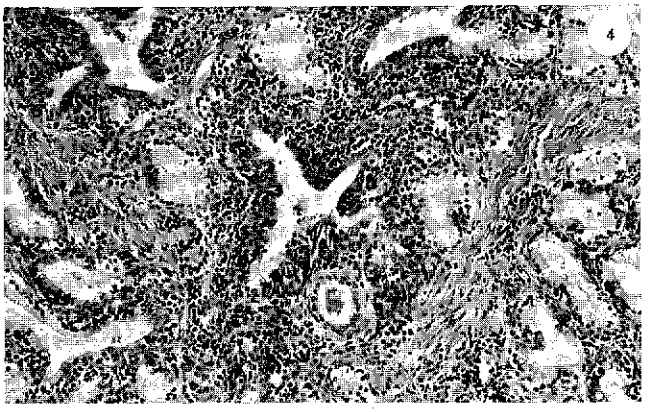

Figure 4. Photomicrograph of bulbourethral gland shown in Figure 3. Note marked periductal mononuclear cell infiltration, increased fibrosis and acinar atrophy.

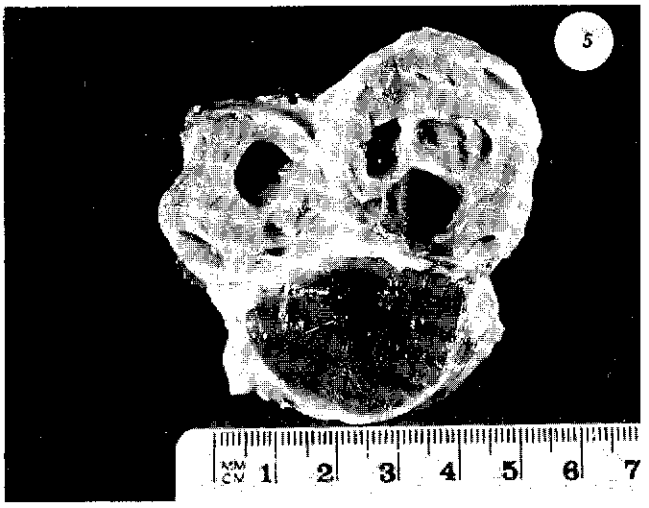

Figure 5. Cross section of both bulbourethral glands, urethra (arrow) and surrounding cavernous muscle. Note bilateral cyst formation in gland parenchyma.

Discussion

Because the BG are normally covered by the bulbocavernous muscle, they are not palpable on rectal examination (Blom and Christensen 1947). Clinical detection of enlarged BG has, however, been reported (Chenoweth and Osborne 1978). Abnormalities of the BG were not found by clinical or post-mortem studies in an important survey in Denmark (Blom and Christensen 1947). Our finding of BA in 4 bulls (1.2%) was somewhat higher than that reported by Bagshaw and Ladds (1974a) who noted this condition in 0.2% of 521 slaughtered bulls. Chenoweth and Osborne (1978) were able to demonstrate enlarged BG associated with a pain reaction in 2 of 702 (0.3%) bulls examined clinically; their finding is in close agreement with our estimation that 2 of 4 BA cases described here could have been demonstrated by rectal examination.

When only histological studies were considered as the basis for detecting inflammation, the occurrence of lesions was higher in other investigations than found here (Kanagawa 1961; Ball *et al* 1964, 1968; Carroll *et al* 1968; Bagshaw and Ladds 1974a; Todorovic and Petrovic 1985).

The association of BA with inflammation in other ASG as seen in Case 2 (bilateral degenerative seminal vesiculitis) in the present study has been reported in other studies on seminal

vesiculitis. Thus Ball *et al* (1964) mentioned BA in 2 of 14 affected bulls, and Ball *et al* (1968) noted BA in 11 of 72 young bulls in which the seminal vesiculitis syndrome was studied. Similarly BA was noted by Carroll *et al* (1968) in 3 of 10 bulls, and by Bachmann (1976) in 1 of 10 bulls with seminal vesiculitis. Bagshaw and Ladds (1974a) reported BA in 4 of the 17 bulls with chronic interstitial seminal vesiculitis and in 4 of 30 other bulls which were affected with degenerative seminal vesiculitis such as in one case reported here. In other studies, however, simultaneous BA and seminal vesiculitis was not observed either grossly or microscopically (Blom and Christensen 1947; Galloway 1964).

Various micro-organisms have been recovered from the BG. These include alpha-haemolytic streptococci from one bull with seminal vesiculitis (Galloway 1964), *Mycobacterium paratuberculosis* from 2 of 6 bulls with clinical paratuberculosis (Larsen and Kopecky 1970), and *Pseudomonas aeruginosa* from 2 of 12 old bulls from which the organism was also isolated from other genital tissues (Zudilin *et al* 1981). Other organisms such as *Haemophilus somnus* have also been isolated from the BG of 1 of 31 normal bull genital tracts (Humphrey *et al* 1982). Additionally, *Mycoplasma* spp and *Ureaplasma* spp have been recovered from 3 and 2 BG, respectively, of 6 bulls with seminal excretion of those organisms (Jurmanova *et al* 1983). In our cases, no mycoplasmas were isolated.

It is difficult to interpret the aetiological significance of our isolation of *Corynebacterium* spp from the BG of bull No 3 in the present study and its association with adenitis and lithiasis. Whether the organism was a primary or secondary pathogen is not clear. Although *Corynebacterium pyogenes* was the most common organism isolated from bull with seminal vesiculitis (Blom and Christensen 1947, 1965; McEntee 1962; Galloway 1964; Bagshaw and Ladds 1974a) or from semen (Hancock and Kelly 1948) no reference concerning this organism in the BG of bulls, was found.

Concretions due to chronic inflammation or precipitation of retained secretions are found in the seminal vesicle or prostate and are composed principally of organic component, phosphates and carbonates (Ladds 1985). However, no reference to such concretions in the bovine BG was found. The calculi observed in the present study were probably due to prior inflammation in both cases; acute inflammation with physiochemical change and crystal precipitation in the glandular duct seems probable. Calcium oxalate and calcium phosphate — the main components of the concretions observed here — are frequently seen in urinary calculi in ruminants (Manning and Blaney 1986a, 1986b). Calcium oxalate is extremely insoluble and can precipitate when the urinary flow is reduced (Manning and Blaney 1986b).

Although foreign vegetable bodies such as thorns are often found in the skin of animals and produce dermatitis (Jones and Hunt 1983) it is difficult to explain the origin of the plant material found in a BG of one bull in the present study. It possibly ascended from the prepuce or distal urethra and thus reached the pelvic urethra where the BG duct opens at the margin of the mucosal fold which forms the ventral border of the process named the urogenital recess (Blom and Christensen 1947). This would seem a logical site for an ascending foreign body to lodge. An alternative possibility, that the foreign body resulted from transrectal migration or penetration direct from the perineal region, was discounted because no lesion was seen surrounding the affected BG.

BA was bilateral in 2 cases and unilateral in the others which is in agreement with one other report (Ball *et al* 1968). Recognition of BA only in adult and old bulls agreed with the findings of Bagshaw and Ladds (1974a).

Our findings of congenital abnormalities in the BG, such as unilateral aplasia and/or glandular fusion are in agreement with those reported by others (Bagshaw and Ladds 1974a; Saunders and Ladds 1978).

Cysts in BG were frequently seen in our work. Whether this condition could affect the fertility is unknown, however, Blom and Christensen (1958) have noted that the fertility of a bull with a BG cyst was reduced even though the semen quality was normal.

We can conclude that despite its deep location, disease of the BG occurs and must be borne in mind when rectal examination is conducted in breeding bulls. It is always the responsibility of the veterinarian to interpret the results of the finding in terms of a diagnosis.

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References

Bachmann P (1976) — *Schweiz Archiv Tierheilk* **118:** 37

Bagshaw PA and Ladds PW (1974a) — *Aust Vet J* **50:** 489

Bagshaw PA and Ladds PW (1974b) — *Vet Bull Weybridge* **44:** 343

Ball L, Griner LA, and Carroll EJ (1964) — *Am J Vet Res* **25:** 291

Ball L, Yuong S and Carroll EJ (1968) — *Am J Vet Res* **29:** 1173

Banks WJ (1981) — *Applied Veterinary Histology,* Williams and Wilkins, Baltimore, p 477

Bellenger CR (1971) — *Aust Vet J* **47:** 83

Blom E and Christensen NO (1947) — *Skand Vet Tidskr* **37:** 1

Blom E and Christensen NO (1958) — *Royal Veterinary and Agriculture College Yearbook,* Copenhagen, p 101

Blom E and Christensen NO (1965) — *Nord Vet Med* **17:** 435

Carroll EJ, Ball L and Yuong S (1968) — *J Am Vet Med Ass* **152:** 1749

Chenoweth PJ and Osborne HG (1978) — *Aust Vet J* **54:** 463

Cottral GE (1978) — *Manual of Standardized Methods for Veterinary Microbiology,* edited by Cottral GE, Comstock Publishing Associated, Cornell University, Ithaca, New York, p 544

Dellmann HD and Wrobel KH (1981) — *Textbook of Veterinary Histology,* edited by Dellmann HD and Brown EM, 2nd Edn, Lea and Febiger, Philadelphia, p 282

Deschamps JC, Ott RS, McEntee K, Heath EH, Heinrichs RR, Shanks RD and Hixon JE (1987) — *Am J Vet Res* **48:** 137

Etheridge JR, Cotew GS Lloyd LC (1979) — *Aust Vet J* **55:** 356

Farrel ID (1974) — *Res Vet Sc* **16:** 280

Freund EA, Erno H and Lemcke RM (1979) — *Methods in Microbiology,* edited by Bergan T and Norris JR, Academic Press, London, vol 13, p 378

Galloway DB (1964) — *Acta Vet Scand* **5:** Suppl 2, 1

Hancock JL and Kelly WR (1948) — *Vet Rec* **60:** 669

Hobson D, Lee N, Quayle E and Beckett EE (1982) — *The Lancet* **2:** 872

Humphrey JD, Little PB, Stephens LR, Barnum DA, Doig PA and Thorsen J (1982) — *Am J Vet Res* **43:** 791

Jones TC and Hunt RD (1983) — *Veterinary Pathology,* 5th edition, Lea and Febiger, Philadelphia, p 1100

Jurmanova K, Weznik Z, Cerna J and Mazurova J (1983) — *Archiv Exper Vet Med,* Leipzig **37:** 421

Kanagawa H (1961) — *Jap J Vet Res* **9:** 14

Ladds PW (1980) — In *Current Therapy in Theriogenology,* edited by Morrow AD, WB Saunders, Philadelphia, p 1157

Ladds PW (1985) — In *Pathology of Domestic Animals,* edited by Jubb KVF, Kennedy PC and Palmer N, 3rd edition, Academic Press, USA, vol 3, p 410

Ladds PW, Dennett DP and Glazebrook JS (1973) — *Aust Vet J* **49:** 335

Larsen AB and Kopecky KE (1970) — *Am J Vet Res* **31:** 255

Mann T, Davies DV and Humphrey GF (1949) — *J Endocr* **6:** 75

Manning RA and Blaney BJ (1986a) — *Aust Vet J* **63:** 393

Manning RA and Blaney BJ (1986b) — *Aust Vet J* **63:** 423

McEntee K (1962 — *Proc 66th US Livest Sanit Assoc,* Washington, p 160

McEntee K and Olafson P (1953) — *Fert Ster* **4:** 128

Nie NH, Hull CH, Jenkins JG, Steinbremer K and Bent DH (1975) — *Statistical package for the Social Sciences,* 2nd edition, McGraw-Hill, New York, p 223

Nobel TA, Folman Y and Volcani R (1961) — *Refuah Vet* **18:** 54

Saunders PJ and Ladds PW (1978) — *Aust Vet J* **54:** 10

Seidel GE Jr and Foote RH (1970) — *Biol Reprod* **2:** 189

Todorovic D and Petrovic S (1985) — *Vet Glasnik* **39:** 441

Trautmann A and Fiebiger J (1957) — *Fundamentals of the Histology of Domestic Animals,* Comstock Publishing, Ithaca, New York, p 260

Trotter DM (1959) — *Am J Vet Res* **20:** 213

Weiss E (1964) — In *Joest's Handbuch der speziellen pathologischen Anatomie der Haustiere,* 3rd edition, edited by Pallaske G and Stunzi H, Parey, Berlin, vol 4, p 83

Zudilin VA, Shipitsyn AG and Korshunova LN (1981) — *Byull Vsesoyuznogo, Instituta Eksperimental'noi Veterinarii* **38:** 42

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