

## THE IDENTIFICATION AND MANAGEMENT OF REPRODUCTIVE DISEASES OF BULLS

### MANAGEMEN DAN IDENTIFIKASI PENYAKIT-PENYAKIT REPRODUKSI PADA SAPI PEJANTAN

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#### ABSTRAK

Resensi artikel ini menjelaskan kondisi penyakit yang dapat ditemui pada setiap tingkatan evaluasi fertilitas rutin dari sapi pejantan. Hal tersebut memberikan informasi pada kontrol dan manajemen penyakit yang menyerang kapasitas reproduksi pejantan untuk membantu para praktisi untuk memutuskan tindakan yang sesuai. Beberapa metode tambahan dari evaluasi fertilitas sapi pejantan dijelaskan, dengan beberapa komentar pada kesesuaiannya. Disimpulkan bahwa manajemen penyakit-penyakit reproduksi pada sapi jantan memerlukan pengertian/pengetahuan dari pejantan yang digunakan; perkiraan nilai ekonomis untuk memilih penanganan yang sesuai harus dievaluasi; observasi secara teratur; perhatian khusus terhadap nutrisi, pembuatan jadwal vaksinasi yang tepat; dan pembuatan program *culling* dan kriteria seleksi.

**Kata kunci:** Pemeriksaan fertilitas, sapi pejantan, tatalaksana pemeliharaan

#### ABSTRACT

This review article describes disease conditions that may be encountered at each stage of a routine breeding soundness evaluation of the bull. It provides information on the control and management of diseases affecting the reproductive capacity of bulls to help practitioners decide the most appropriate course of action. Some additional methods of evaluating the fertility of bulls are described, with comments on their suitability. It is concluded that the management of reproductive diseases in bulls requires an understanding of the bulls intended use; an objective estimate of the bulls economic value to allow treatment options to be evaluated; regular observation; careful attention to nutrition; development of, and strict adherence to, a vaccination schedule; and the development of culling and selection criteria.

**Key words:** Breeding Soundness, Bull, Management.

## INTRODUCTION

Anecdote suggests that a significant percentage of bulls are culled as a result of being reproductively unsound, in contrast to being removed from the breeding herd for routine managerial reasons such as age or superseded genetics. This premature loss of bulls from the breeding "team" can result from a range of conditions including developmental abnormalities, infectious disease, physical injury and degenerative conditions. These in turn can be influenced by management, nutrition and genetics.

The aim of this paper is to provide veterinarians involved in cattle practice with a practical and scientific framework for the diagnosis, pathology, control and management of major reproductive diseases encountered in bulls.

### **The General Physical Examination – Systemic Infectious Diseases That Affect the Reproductive Function of Bulls**

The negative effects of increased testicular temperature on spermatogenesis have been well documented (Kastelic *et al.*, 1997). Studies indicate that increases in scrotal temperature lasting for as little as 24 hours can reduce semen quality for more than a month (Austin *et al.*, 1961). Therefore, any systemic disease that raises the body and scrotal temperature of a bull for longer than 24 hours is capable of reducing fertility independently of any direct effects it may have on testicular tissue or the tubular reproductive tract.

The significance of specific diseases will depend on the locality of the bull. However, diseases which are commonly encountered in the Australian cattle industry include Bovine ephemeral fever (BEF) and Tick fever.

The pathogenesis and epidemiology of BEF is well described (St George, 1986; Uren, 1989). It has an incubation period ranging from two to ten days, with a mean of three days (Mackerras *et al.*, 1940). Importantly, the mean duration of clinical disease is considered to be longer in bulls compared to cows (5.3 and 3.2 days respectively), (Spradbrow and Francis, 1969). Although the virus is not considered to be directly cytopathic, there is a massive stimulation of interferon production from the host. Resultant inflammation of mesodermal tissue is responsible for the clinical signs, of which high body temperature is a consistent finding. Long term effects on spermatogenesis have been described

(Chenoweth and Burgess, 1972) and anecdote suggests that infertility in bulls resulting from BEF can occur regardless of whether the bull has been reared in areas where the disease is endemic or not (McCool and Ladds, 1992). A live attenuated vaccine (10 doses/20ml reconstituted vial) and an inactivated vaccine (500ml packs) are available in Australia to assist with control of the disease. There appears to be some resistance to the use of BEF vaccine in extensive herds due to anecdote of incomplete control (McCool and Ladds, 1992) and the inconvenience associated with the vaccination protocols in these situations. Although the effects of the disease on herd fertility may be reduced by the use of high joining percentages, its effects are likely to become increasingly evident as more economical use of bulls is pursued. Therefore, where possible, it is recommended that bulls in areas where BEF has been identified should be vaccinated against BEF prior to the high risk periods of summer and autumn (St George, 1986).

Tick fever epidemiology, clinical signs, treatment and prevention are well described (Lagos and Fitzhugh, 1970). Despite some breeds being more resistant to the effects of tick fever than others, if bulls are reared in tick-free areas and then moved to areas where ticks are endemic, vaccination against tick-fever is recommended. This recommendation is made not only from the point of view of the adverse effects of fever on semen quality, but also due to the threat of severe morbidity or mortality resulting from the disease.

Important points regarding the use of the vaccine are that it gives long-term immunity after a single dose; potential vaccine reactions are usually more severe as the animal gets older; and four to eight weeks are needed for effective immunity to develop. Thus, to avoid reduced semen quality from possible vaccinal reactions, vaccinating bulls three months or more prior to intended use is ideal, although not always practical in the pre-sale preparation. If the final destination of bulls is known well in advance, they can be vaccinated as weaners, resulting in minimal vaccinal reactions (Queensland Department of Primary Industries, 2002). It could be speculated that vaccinating bulls as they undergo puberty between eight to twelve months of age (Coulter, 1986) is best avoided until the long-term effects of any vaccinal reactions on semen quality are quantified.

## Local and Sexually Transmitted Infectious Diseases That Affect the Reproductive Function of Bulls

### Campylobacteriosis

Bovine venereal campylobacteriosis (BVC = Vibriosis), caused by *Campylobacter fetus* subsp *venerealis*, is a venereal disease that is widespread in most areas where cattle are bred naturally. The pathogenesis of the disease and effects on herd reproductive performance are well documented (Ball *et al.*, 1983, Roberts, 1986).

### Diagnosis

Direct culture of preputial smegma is considered the most reliable method of diagnosing *C. fetus* infection in the bull (Hum *et al.*, 1993).

Bulls are individually sampled by extracting smegma samples from the prepuce using negative pressure from a 20ml syringe attached to a plastic pipette. The pipette is stroked vigorously back and forth inside the prepuce, with a stroke length of approximately 15 cm. The collected sample is washed into 4 mls of phosphate buffered saline. Both *C. fetus* and *T. foetus* can survive up to 48 hours in this state (Ball *et al.*, 1983). However, the shorter the time between collection and culture, the better the result. Therefore, after waiting at least 10 minutes for the contents to settle, 1ml of supernatant is decanted and added to a transport enrichment medium (Landers TEM), (Lander, 1990). The transport media is kept at ambient temperature in the dark and sent to arrive at the laboratory within 24 hours. The campylobacter culture bottles are incubated for three days in a micro-aerophilic atmosphere before final culture.

It is important to recognise that both *C. fetus* and *T. foetus* can be spread by fomites. Therefore, clean, disposable plastic gloves should be used when handling the sampling equipment and sheath of each bull to prevent iatrogenic infection of subsequent bulls (Ball *et al.*, 1983).

More recently, the campylobacter IgA ELISA has been used to diagnose the presence of *C. fetus* infection in herds (Hum *et al.* 1994) by sampling cervical mucus from cows. It should be noted that specific antibody to *C. fetus* sub *fetus* may cause cross-reaction to the IgA ELISA (Hum *et al.*, 1994).

Fluorescent antibody techniques have also been used to identify *C. fetus* sub *venerealis* from culture media (McCool *et al.*, 1988), however the specificity of this technique has been questioned (Lander, 1990, Mellick *et al.*, 1965).

More recently, a rapid screening assay for *C. fetus* (in bovine semen) using the Polymerase Chain Reaction (PCR) restriction endonuclease analysis has been developed to complement isolation by culture (Eaglesome *et al.*, 1995). This assay, which permits differentiation between the two subspecies of *C. fetus* (*venerealis* and *fetus*) is highly sensitive, is completed within 10 hours, but is yet to be applied to routine diagnosis.

### Control and Management

Prior to the development of a vaccine in the mid 1960s, control of BVC required segregation of infected and non-infected herds, the use of artificial insemination and the use of antibiotics. These techniques were found to be disruptive to other herd management practices and were often ineffective (Hum *et al.*, 1993). Vaccination of infected bulls with commercially available vaccine<sup>1</sup> is currently considered very effective in preventing infection. It has also been shown that vaccination of infected bulls is usually therapeutic (Bouters *et al.*, 1973, Clark *et al.*, 1979). Due to these findings, control of Campylobacteriosis in the herd has primarily involved vaccination of the bulls (Clark *et al.*, 1968). However, due to documented failures of vaccination to eliminate infections in some bulls (Hum *et al.*, 1993, Vasquez *et al.*, 1983), this type of programme may allow carrier animals to remain in the herd. If eradication is required, the most likely programme to achieve this goal is to vaccinate all breeding animals in the herd (Hum *et al.*, 1993). The programme described by these authors involves initially vaccinating all heifers, older cows and bulls in accordance with the vaccine manufacturers instructions. At the time of the second vaccination, infusion of antibiotic or antiseptic into the prepuce of all bulls is recommended. In the following year, all bulls and replacement heifers are vaccinated. From the third year, all bulls should be vaccinated annually. It is recommended that the annual booster vaccination be given approximately a month prior to joining in seasonal herds to ensure protection is timed to peak during the period of high risk (BonDurant, 1997).

<sup>1</sup> Vibrovax - CSL

In the specific case of extensive northern herds, the effectiveness of bull only vaccination for the control of BVC is hindered by the presence of scrub bulls (McCool and Ladds, 1992). In addition, herds with limited paddock numbers and poor stock control may have a mustering efficiency as low as 50%, and a requirement to run weaner heifers with the main breeder herd. Thus, until stock control can be improved and populations of unmanaged bulls controlled, BVC will continue to have an influence on fertility in these herds.

There appear to be no scientific studies on the effectiveness of local treatment of the prepuce in clearing campylobacter infections in bulls, although success using a penicillin/streptomycin mix has been reported (Hum *et al.*, 1991). However, it is evident that the organism is relatively easy to kill *in vitro* as indicated by its fastidious requirements for successful culture (BonDurant 1997). With the removal of streptomycin from the available antibiotics for use in cattle, suggested topical antibiotic treatment includes a combination of 4g erythromycin and 10g neomycin (Irons 2002) mixed with approximately 30ml vegetable oil to ensure prolonged contact with the prepuce. It is suggested that the prepuce be rinsed out prior to treatment and that the antibiotic remain in contact with the prepuce for a minimum of 15 minutes. This is achieved by clamping the preputial orifice after antibiotic infusion and massaging the sheath. There is the possibility that antibiotics may not be necessary for local treatment of the prepuce for campylobacter. However, until the appropriate studies are done, the use of antiseptics such as manufacturer recommended dilutions of iodine, chlorhexidine or the acridine derivatives mixed with vegetable oil can only be suggested as possible alternatives where antibiotic use is not desirable. There is a suggestion that the use of wetting agents, instead of vegetable oil, may assist in allowing the penetration of therapeutic agents into the preputial crypts (BonDurant 2002). This may be of particular importance in the treatment of older bulls.

It is apparent that preventing BVC infection in bulls is preferable to treatment. Thus, the vaccination of all yearling bulls prior to leaving the stud, or their first breeding season, is recommended. It should be noted that the booster vaccination dose for bulls is 2½ times that recommended for cows. This is a reflection of the difficulty in stimulating an immune response within the bulls prepuce compared to the

reproductive tract of the female. In order to provide adequate immunity for the bull, oil based Freund's adjuvant is used in the vaccine (Ball *et al* 1983). Unfortunately, this can be irritant and some stud producers have been reluctant to vaccinate bulls prior to their sale due to the occurrence of large lumps at the injection site when using this vaccine. Until a vaccine is developed that doesn't cause such a severe local reaction, the lumps need to be marketed as a positive sign that the bull is protected against BVC to encourage producers to vaccinate bulls prior to exposure.

#### Trichomoniasis

Trichomoniasis is caused by the protozoan *Tritrichomonas foetus* (Rae, 1989). The pathogenesis of trichomoniasis in cattle has been well documented (BonDurant, 1997). Perhaps the most significant aspect of this organism is its ability to sequester within the deep preputial crypts of older bulls, resulting in chronic infections.

#### Diagnosis

Diagnosis of Trichomoniasis using direct microscopic examination of preputial fluids or culture media (perhaps utilising the field-adapted "In-pouch" system<sup>2</sup>), is preferred. *T. foetus* is well adapted to the micro-environment of the preputial cavity. It appears to utilise urine and other smegma components and produces proteolytic enzymes that may aid the organism in combating the hosts' response to it (BonDurant, 1997). However, caution is needed when interpreting visual results because at least one other *Tritrichomonas* "look alike" flagellate protozoan, perhaps of intestinal origin, is now recognised, and may lead to a false positive result (BonDurant, 1997). Therefore, in US studies, a two-step detection system is now proposed involving culture, followed by characterisation of any isolate by PCR (Felleisen *et al.*, 1998, Riley *et al.*, 1995).

Traditionally, it has been accepted that individual bulls should be tested a minimum of three times at weekly intervals to ensure they are negative (BonDurant, 1997). If individual bull diagnosis is required, this procedure is still recommended. However, this protocol may not be practical in extensive northern situations. If the aim is to simply diagnose the presence of Trichomoniasis in a herd where the expected prevalence is between 20% and

<sup>2</sup> Biomed Diagnostics, San Jose, Ca, USA.

40%, testing each bull only once should accurately detect the disease (Barling *et al.*, 1997). This can be calculated from the reported sensitivity and specificity of 82% and almost 100% respectively.

As with BVC the incidence of *T. foetus* in bulls in infected herds increases with age. This is presumably because of the increased prominence of crypts in the penile and preputial epithelium of older bulls. In view of persistent infection of *T. foetus* infection in the bull, as opposed to intermittent infections in cows, sampling of bulls is preferred for herd diagnosis.

Collection of smegma for culture of *T. foetus* in the bull is similar to the procedure used for the culture of *C. fetus*. Indeed, testing for *T. foetus* can be done in association with *C. fetus*. Thus, one ml of sediment from the bottom of the solution collected as described under the diagnosis of BVC is inoculated into the Tritrichomonas medium, obtained from the laboratory, and handled as previously described.

An important aspect of culturing *T. foetus* from the preputial smegma of bulls is that the chances of obtaining a positive result are reduced during periods of increased sexual activity (Clark *et al.*, 1983). This depletion of the preputial *T. foetus* population also accounts for the progressive reduction in cows that become infected as the breeding season progresses (Clark *et al.*, 1983). Thus, collection of preputial smegma for culture should be done prior to the breeding season, or at periods of reduced sexual activity in systems where bulls are left in all year.

#### **Control and Management**

With respect to managing Trichomoniasis, there is little research demonstrating the impact the organism may have on overall reproductive parameters of an extensively managed year-round calving pastoral herd. One Australian study compared the productivity of 73 Hereford cows bred to *T. foetus* infected bulls with 25 cows bred to non-infected bulls over a three year period (Clark *et al.*, 1983). At the end of the three year period, in this year-round mating environment, cows bred to infected bulls produced a mean of 17.6% less calves compared to cows bred to the non-infected bull. In the first and second years, the difference in calving percentages between the groups was 19.6% and 28.6% respectively, with this difference reducing to 7.6% in the third year. The mean days to first calving, the interval between the first and second calvings and the interval between second and third calvings was 40.2,

65.5 and 21.2 days longer respectively for cows bred to the infected bulls. From this study, it was apparent that the main impact of *T. foetus* occurred in the first two years following initial exposure to infected bulls.

Although an effective vaccine against *T. foetus* was developed by CSIRO (Clark *et al.*, 1983), it was never made commercially available in Australia. In the US, a similar vaccine lacks efficacy in bulls (BonDurant, 1997).

If trichomoniasis is determined to cause significant economic loss in a herd, the following strategy has been used to successfully eliminate it from a closed herd over a two-year period (Clark, 1974). The programme involves pregnancy testing all breeders and culling empty cows. All cows failing to calve are also culled and only bulls three years of age or younger are used for breeding. The success of this programme in controlling the disease may, in part, explain why trichomoniasis is not currently recognised as a significant cause of production loss in the southeastern cattle producing regions. Management practices incorporating some or all aspects of this programme have been in widespread use for many years in these areas. These practices were not specifically employed to reduce the incidence of trichomoniasis, but for the overall improvement they have on herd productivity (Mossman, 1984, Mossman and Hanly, 1977). As northern herds become more tightly managed and the use of pregnancy testing in all breeders increases, it could be expected that the prevalence of trichomoniasis will reduce.

#### **Infectious Bovine Rhinotracheitis**

Serological surveys have confirmed that IBR is widespread in the cattle population. For example one study found 95.8% of 400 bulls serologically positive from almost 60 herds, with the virus being isolated from the preputial cavities of 76% of 368 bulls, some of which had typical herpetic ulcers (see under balanoposthitis) (Bagshaw and Ladds, 1974).

It is possible that the bulls willingness to serve may be reduced when balanoposthitis is present during the active stages of the infection. Younger bulls are usually affected and the condition may last up to two weeks (see Balanoposthitis). In addition, IBR induced balanoposthitis can result in preputial prolapse and subsequent trauma (Wolfe *et al.*, 1983).

No specific treatment or control measures are currently used other than conservative management of bulls showing clinical signs of balanoposthitis, such as sexual rest, topical ointments or sling application as necessary.

#### **Mycoplasmosis**

*Mycoplasma bovis* is considered the most common *Mycoplasma* infection of the genital tract of bulls (Kirkbride, 1987). Although present in the prepuce and urethra of normal bulls (Sprecher *et al.*, 1999), the presence of *M. bovis* in the accessory sex glands testicles or epididymides is considered pathological (Kirkbride, 1987). In particular, infection of the epididymides may interfere with sperm maturation (Panangala *et al.*, 1981). It is unlikely that *M. bovis* gains access to the accessory sex glands, testicles or epididymides by retrograde infection. Most evidence suggests pathological infection enters via the respiratory or digestive tracts and becomes systemic before localising in these areas of the reproductive tract (Kirkbride, 1987).

#### **Control and Management**

No specific management techniques have been devised for the control of this organism in bulls. *M. bovis* appears to be ubiquitous in the bovine population, making efforts to apply quarantine techniques unproductive. Although macrolide antibiotics such as tylosin are effective against the organism *in vitro*, there is no data on the efficacy of systemic treatment in clearing the infection. There is also the possibility that many macrolides may soon not be available for use in cattle.

Semen from affected bulls may be used for artificial insemination if treated with appropriate antibiotics (Kirkbride, 1987).

#### **Ureaplasmosis**

*Ureaplasma diversum* has been identified as a cause of infertility and abortion in cows (Kirkbride, 1987). However, it is a common inhabitant of the prepuce and urethra of normal bulls, and is often present in normal bull semen (Kirkbride, 1987, Sprecher *et al.*, 1999). This indicates that specific requirements may be needed for *U. diversum* to influence herd fertility. A possibility is that there is variation in the virulence between the three serogroups, with herd fertility compromised only in the presence of more virulent serogroups. There is also the possibility *U. diversum*

may have a greater impact on herd fertility where artificial insemination is used, due to the need to penetrate the cervix and deposit semen directly into the uterus. One study reported improved fertility in a herd that had regularly used A.I. when natural breeding was again used (Doig *et al.*, 1979). There is no evidence *U. diversum* causes male infertility or lesions to the bulls reproductive tract under natural conditions, however bulls may transmit infection to cows (Kirkbride, 1987).

Control of *U. diversum* is focused on management of the cow herd and antibiotic treatment of semen used for artificial insemination. No specific control methods are used for the bull.

#### **Other Infectious Diseases**

Although not regarded as venereal diseases, other serious infectious diseases that may compromise herd fertility and which may be spread in semen, include pestivirus (Bovine Virus Diarrhoea) infection, and leptospirosis. Studies on BVD have suggested that BVD virus replicated in the seminal vesicles and prostate of infected bulls (Kirkland *et al.*, 1991). Serological or direct testing of genital fluids for these agents may at times be indicated. A PCR test for the detection of *Leptospira* spp. serovars in bovine semen, has recently been described (Heinemann *et al.*, 2000).

#### **Physical Examination of the Reproductive Tract**

##### **Rectal Examination**

##### *Accessory Sex Glands (ASG)*

These include the seminal vesicles, prostate, bulbourethral glands and ampullae – the enlarged dorsal termination of the ductus deferens. In the bull, important disorders of the ASG significantly involve only the seminal vesicles, and to a lesser degree the ampullae. These disorders may be congenital, and possibly inherited as in the case of so-called segmental aplasia of mesonephric duct derivatives, or be inflammatory such as in seminal vesiculitis (Cavalieri and Van Camp, 1997). Importantly, animals with congenital defects of the ASG are predisposed to seminal vesiculitis (Blom, 1979, BonDurant *et al.*, 1999) probably because ascending infection is facilitated.

##### *Seminal Vesiculitis*

Seminal vesiculitis appears to present either as an acute condition with swelling of the glands commonly noted in young bulls, or as a chronic syndrome of sclerosing inflammation in older bulls.

In a north Australian study the incidence of seminal vesiculitis was 9% with the chronic and sclerosing infection of older bulls the main manifestation (Bagshaw and Ladds, 1974). Reports from North American herds suggest a range of 0.85% to 10%. However, an individual herd prevalence of 49% has been reported (Cavalieri and Van Camp, 1997).

Various infectious agents including viruses, chlamydia, mycoplasma (see above), bacteria, fungi, and even *T. foetus* have been isolated from inflamed seminal vesicles but the pathogenesis is unclear. Probably a virus (possibly bovine herpesvirus-1) is responsible for the acute syndrome in young bulls, with bacterial or other microbial infection representing a secondary event (McEntee, 1990). *Actinomyces pyogenes* is commonly isolated from chronic, or abscessed forms of the condition (Cavalieri and Van Camp, 1997).

Infection of the seminal vesicles may occur by ascending or descending infection, or haematogenously, but irrespective of the initial site of infection, it is clear that intra-ductal spread to and from other ASG, the epididymides, and even the testes, occurs.

Seminal vesiculitis can cause a reduction in semen quality, and depending on the causative agent, treatment results may not be satisfactory. Affected bulls are often culled prematurely (Cavalieri and Van Camp, 1997).

#### Diagnosis

Diagnosis of seminal vesiculitis is by detection, on rectal palpation, of irregular gland size, lobulation, or consistency. Crepitus and possibly pain may also be present. Diagnosis may also be supported by presence in the ejaculate, of erythrocytes, leukocytes (possibly visible in the semen as floccules of purulent material), fibrin, and perhaps bacteria, although caution is needed to ensure that these products did not originate elsewhere in the genital tract. In this regard, the use of rectal massage of the ASG in obtaining semen from suspect bulls, may aid diagnosis. A technique for collecting vesicular fluid samples with minimal contamination has been described (Parsonson *et al.*, 1971). It involves sedating the bull, catheterising the penis, washing the external penis with 1% povidone-iodine solution, several flushes of the urethra with sterile saline, removal of the first catheter, re-washing the external penis with povidone-iodine and the insertion of a

sterile catheter. Collection of accessory sex gland fluid is then completed by rectal massage of the glands.

Transrectal ultrasonography may also be used to aid the diagnosis. The ultrasonographic appearance of the seminal vesicles has been described (Weber *et al.*, 1988).

#### Control and Management

Close confinement and the feeding of high-energy diets appear to be associated with an increased prevalence of the disease (Cavalieri and Van Camp, 1997). Age is also a factor, with young, peripubertal bulls and aged bulls (nine years or older) most at risk. Anecdote suggests breed may also be a factor, however one study found no significant differences between *Bos indicus* and *Bos taurus* bulls maintained in a similar environment (Weber *et al.*, 1988).

Treatment is difficult due to the lobulated structure of the gland and the high lipid content of the secretory cells. This difficulty has resulted in many treatment regimens being described (Cavalieri and Van Camp, 1997), however sexual rest and isolation from other bulls, with or without the inclusion of antibiotic therapy, are most commonly used.

#### Examination of the Scrotum, Testicles, Penis and Prepuce

##### *The Scrotum*

Severe scrotal dermatitis, resulting for example from Chorioptic mange, or (rarely) mycotic infection, may induce testicular degeneration, mostly as a consequence of impaired thermoregulation.

Marked swelling of the scrotum by fluid accumulating secondary to generalised ascites has been described affecting a number of bulls in separate herds. The importance of interpreting scrotal circumference measurements in such cases of scrotal hydrocele has been stressed (Arbitt *et al.*, 1995, Marcus *et al.*, 1997). In these herds neither the precise cause(s) nor pathogenesis of hydrocele were ascertained, but infections with *Ostertagia* sp. and the blood-borne protozoan *Eperythrozoon* sp. (Welles *et al.*, 1995) were identified in bulls in separate herds and may have been causally involved. In these outbreaks, decreased semen quality was again attributed to compromised testicular thermoregulation. Following resolution of the hydrocele, a period of up to 4 months was required for semen quality to be restored.

### *The Testicles and Epididymides*

#### Cryptorchidism

The occurrence of cryptorchidism in cattle is considered to be approximately 0.1%, which is low compared to other species (Marcus *et al.*, 1997). There are indications that the condition is heritable (Larson, 1980, Roberts, 1986), however the heritability and mechanism of inheritance are not well defined. It can not be ruled out that environmental factors such as hormones or toxins may affect foetal gonadal development and the subsequent phenotypic expression of cryptorchidism (Marcus *et al.*, 1997). Thus, not all cases of cryptorchidism can be assumed to be the result of heritable factors. The condition is considered more common in Polled Herefords and Shorthorns (Steffen, 1997).

The left testicle is more commonly retained compared to the right, with one study finding that the left testicle was retained in 69% of cases (St Jean *et al.*, 1992). There appears to be no semen production from cryptorchid testicles, with evidence that a unilateral cryptorchid testicle will progressively reduce the function of a normal, scrotal testicle (Marcus *et al.*, 1997). Serum testosterone levels in bulls with cryptorchid testicles progressively decline with age (Marcus *et al.*, 1997), meaning that their use as teaser animals or in commercial herds (in the case of a unilateral cryptorchid) is limited.

The diagnosis of this condition is self evident.

Due to the perceived tendency for the condition to be heritable via an autosomal recessive mechanism (Steffen, 1997), affected stud bulls should be culled. It is also recommended that the sire, dam and siblings of affected bulls be identified for monitoring should further cases of cryptorchidism be identified. In addition to cryptorchidism, other conditions that can result in reduced testicular function include hypoplasia, degeneration, inflammation and infection.

#### Testicular Hypoplasia

The existence of partial testicular hypoplasia is well documented and is thought to be heritable (Madrid *et al.*, 1988). Testicular hypoplasia refers to underdevelopment of the testis. The testis and testis/bodyweight ratio are smaller than normal, but importantly, in contrast to testicular degeneration, the hypoplastic testis never had developed to normal size. The condition is usually unilateral, most commonly affecting the left testicle (Steffen, 1997).

On histological examination, the prevalence of defective seminiferous tubules varies more or less in proportion to testis size; distinctly hypoplastic testicles have a high proportion of non-vacuolated, small diameter, and often "Sertoli-cell-only" seminiferous tubules.

The condition has been identified in at least 12 breeds and has been characterised as autosomal recessive in Swedish Highland cattle (Steffen, 1997). It is suggested that routine breeding soundness evaluation procedures such as scrotal circumference measurement, testicle palpation and semen evaluation will select strongly against testicular hypoplasia reducing the need for specific identification and control techniques.

#### Testicular Degeneration

Mild degeneration of some seminiferous tubules has been reported in up to 90% of beef bulls, indicating that almost all young bulls have some testicular degeneration (Ball, 1966). However, these mild changes have little effect on semen quality. Although the fate of seminiferous tubules with mild degenerative changes is unclear, the increasing prevalence of tubules with atrophic epithelium as bulls age indicate that the process is continuous and intensifies as the bull gets older (Humphrey and Ladds, 1975, McEntee, 1958).

There are many possible causes of testicular degeneration recorded in the literature (Van Camp, 1997). Some of these are listed in Table 1.

It is interesting that with regard to the effect of high environmental temperatures and humidity *per se* on bull fertility, that quantitative histological studies of testes of *B. taurus* bulls in Northern Australia were unable to demonstrate a greater occurrence of testicular degeneration in mid summer than in mid winter (Ross, 1977). This is perhaps a reflection of the effectiveness of the testicular cooling mechanisms in healthy bulls, compared to bulls suffering fever as a result of infectious disease.



**Table 1. Some causes of testicular degeneration in the bull<sup>3</sup>.**

Cause of degeneration	Classification
Hypothalamic/Pituitary dysfunction	E, H
Orchitis, periorchitis, epididymitis	Inf
Acute fever/Hyperthermia	Inf
Autoimmunity	H, Inf
Heavy metals, cadmium, griseofulvin, gossypol, fescue, oestrogens, testosterone	Tox
Testicular trauma	Tr
Testicular biopsy	Tr, Iat
Prolonged recumbency	Inf, Tr
Chromosomal aberrations	H, C
Freemartin co-twin	C, H
Pampiniform phlebitis/varicocele	Inf, H, Tr
Inguinal hernias	H
Cryptorchidism	H, C
Prolonged stress	M, I, Tr, Iat
Irradiation	Iat
Testicular neoplasia	Neo
Sever malnutrition	Nut
Vitamin A deficiency	Nut
Zinc deficiency	Nut
Scrotal fat	Nut, H
Duct obstruction/sperm stasis	Tr, Inf, H
Ultrasonography	Iat

E = endocrine, H = heritable, Inf = infectious, Tox = toxic, Tr = traumatic, Iat = iatrogenic, C = congenital, M = managerial, Neo = neoplastic, Nut = nutritional.

It is apparent there are many possible causes of testicular degeneration, with the prognosis for recovery, and management decisions, heavily dependent on the cause and duration of the insult. A major problem can arise in assessing the significance of "small testes", in bulls examined at puberty or later. If no history is available (i.e. whether the small testis was ever any larger), the differentiation of true hypoplasia from degeneration (i.e. atrophy) may be difficult, if not impossible to decide, even when histological examination is performed. This problem is further complicated by realisation that hypoplastic testes are, in any case, predisposed to degeneration. Although reaching a diagnosis and establishing the cause is difficult, a detailed history and careful physical examination are essential starting points. The requirement for further diagnostic tests will

often become apparent from information obtained from these initial investigations. The identification of freemartin co-twins is an example of the importance of history and accurate records. Otherwise the condition could mistakenly be identified as heritable, with the genetics of siblings unnecessarily put into question.

Semen collection and careful examination of the spermogram can be useful in providing a prognosis (Blanchard *et al.*, 1991).

#### *The Penis, Prepuce and Sheath*

##### **Persistent Penile Frenulum**

Persistent penile frenulum occurs when there is incomplete separation of epithelia of the penis and prepuce such that during erection the anterior penis cannot be extruded from the prepuce in a linear fashion. Caution is needed in differentiating true persistent frenulum from the balanopreputial fold in normal immature males. In the normal situation, the penis and prepuce begin to separate at about four weeks of age, with complete separation by eight to eleven months of age (Elmore, 1981). Persistent frenulum is considered to occur in approximately 0.3% to 0.5% of bulls (Carrol *et al.*, 1964).

The condition can be identified on routine breeding soundness evaluation in a large proportion of bulls if an assistant is available to pull back the sheath to expose the penis while rectal examination and massage is being carried out.

There is limited evidence on the heritability of this trait. However, the information that exists suggests it may be heritable as a simple autosomal recessive (Steffen, 1997). This suggests that affected stud bulls should not be used, and that dams and sires of affected bulls should be identified for future scrutiny as possible carriers.

Bulls for use in commercial herds can be treated by quickly clamping and severing the attachment while the penis is erect under the influence of electroejaculation. Alternatively, sedation of the bull with 20 to 30 mg of acepromazine, rectal massage, or the use of a pudendal nerve block may be used to assist with extension of the penis. Haemostasis is usually unnecessary.

It is emphasised that surgical correction of conditions that are considered heritable is not recommended. Progeny from these bulls should not find their way

<sup>3</sup> After Van Camp 1997.

back into the breeding herd. Specifically, care should be taken if commercial producers breed their own bulls such as littermates for joining to heifers, or in situations where clean musters for castration and branding are not possible.

#### Premature Spiral Deviation of the Penis

PSDP (= corkscrew penis) appears to be a heritable condition, however there is very little scientific data to confirm this suspicion (Steffen, 1997). There is anecdotal evidence that some cases may be the result of trauma, however it is difficult to differentiate between the two causes clinically. Therefore, the conservative recommendation is to consider cases of PSDP as heritable until further evidence is presented.

The condition is progressive, with the attachment between the dorsal apical ligament and the tunica albuginea of the penis becoming less intimate as the bull ages. An understanding of the progressive nature of this condition is useful in identifying affected bulls. Younger bulls with PSDP may only display the abnormality in less than 25% of services. Thus, affected bulls may go undetected until their second or third joining season. The condition commonly progresses until almost 100% of service attempts are affected by PSDP.

Diagnosis depends on visualisation of the condition, ideally during a 20 minute serving capacity test. No relationship has been identified between spiral deviation during electroejaculation and PSDP. Thus, observation of natural service is essential for the diagnosis of this condition.

The libido of affected bulls may progressively decrease as they become frustrated with the inability to gain intromission.

Stud bulls displaying PSDP should not be used for seedstock production. Commercial bulls where the condition is moderate to severe (more than 30% of service attempts are affected), should be culled where possible, or surgically corrected as a less desirable second option. The decision for surgery can be assisted by a cost-effectiveness evaluation.

#### Balanoposthitis

Inflammation of the glans penis (balanitis) is often accompanied by inflammation of the prepuce (posthitis). In considering balanoposthitis it is important to be mindful of the diversity and large number of commensal micro-organisms that reside in

the preputial cavity, and of local immune mechanisms that operate there. In the bull, the best studied specific cause of balanoposthitis is that caused by bovine herpesvirus 1 (IBR), in which clinically, there is a thin purulent preputial discharge evident (see IBR, above). Close examination reveals that at 2-3 days post infection, numerous white opaque foci of necrosis are present. These foci may coalesce to form larger lesions and there may be concurrent swelling of the penis and prepuce.

Unfortunately for diagnostic purposes, the typical herpetic foci are transient, existing for only 1-2 days. After this, they become indistinct then slough superficially to leave ulcers or erosions, especially in the area of the glans. Healing commences in 6-8 days and is complete after 2 weeks. Diagnosis is confirmed by virus isolation, ELISA or serum neutralisation test.

A posthitis in bulls, which in many respects parallels severe balanoposthitis (so-called "sheath rot") in wethers and sometimes rams, has been described as a herd problem in Australia (Nielsen, 1972), and Uruguay (Reit Correa *et al.*, 1979). In bulls, minor ulcers up to extensive ulceration of much of the preputial orifice occur, with bleeding, oedema and perhaps abscessation and myiasis. As in sheep, the cause is multi-factorial. Factors commonly associated with the condition include a high plane of nutrition (such as legume forage), and the presence of *Corynebacterium renale*. It was suggested that the occurrence of posthitis in bulls but not steers (in the same herds) might have been due to genital injury from homosexual activity in the bulls facilitating infection.

Treatments incorporating parenteral procaine penicillin or topical antibacterial ointments have been successful.

#### Penile Fibropapilloma

Penile fibropapillomas are caused by strain 1 of the bovine fibropapilloma virus. They are considered common in bulls between one and two years of age (Pearson, 1972, St Jean, 1995), with an incidence of 2.8% reported in yearlings (Bruner *et al.*, 1995). Rarely do they occur in bulls over three years of age (Van Camp, 1997). However, incidence can vary due to management and breed (Bruner *et al.*, 1995). They do not metastasise, are not locally invasive and are by far the most common tumours of the bovine penis (St Jean, 1995). They are capable of affecting

fertility by a number of mechanisms. Phimosis or paraphimosis can result from large lesions, or pain may reduce copulatory activity. Bleeding during copulation may reduce semen quality (Van Camp, 1997).

Fibropapillomas of the penis result from direct contact of the penis with body warts of other animals, or fomites. In particular, bulls can become infected when mounting other animals with warts around the perineal area (St Jean, 1995). In this regard, a higher incidence usually occurs in younger bulls due to the combination of reduced active immunity resulting from minimal previous exposure and the management requirement of maintaining groups of young bulls together, with resultant homosexual behaviour (St Jean, 1995). Animals with lesions on the nose may also be a source of spread for the virus (Van Camp, 1997).

Fibropapillomas of the penis usually regress with time, however regression is considered to take longer compared to body warts due to protection provided by the prepuce (St Jean 1995). The authors consider that the time for regression to occur may range from 2 to 8 months. Thus, the treatment options will vary depending on the time-frame for the bulls use. If time is not available to allow natural regression, the use of an autogenous vaccine or surgery will be necessary to remove the mass. The technique for surgical removal has been well described (St Jean, 1995), while the production and use of an autogenous vaccine will vary with local facilities and conditions (Noordsy, 1989).

Although controlled studies are not available, management strategies for this disease can be developed from the known epidemiology. Particular practices that would seem beneficial in the management of yearling bulls include isolation of bulls with body warts and avoiding coarse feed where possible. This may reduce the chance of the penile and preputial mucosa being penetrated by abrasive faeces during homosexual contact, therefore reducing sites for viral entry.

#### Preputial Injury and Prolapse

Preputial injury and prolapse is reported to most commonly occur during the breeding season (Memon *et al.*, 1988). However, in this study there were still 31% of cases that fell outside of the joining period. Yet, it is evident that regular observation of bulls during the season is necessary for early detection of

prolapse or injury at this critical time. Some factors involved in the aetiology of preputial prolapse have been identified (Venter and Maree, 1978). These include injury and infection, functional and anatomical abnormalities of the prepuce and preputial retractor muscle, inefficient preputial retractor muscle action, pendulous sheath (Wolfe *et al.*, 1983) size of the preputial orifice, longer preputial length (van den Berg, 1984), and genetic predisposition. It has been suggested that *Bos indicus* and polled breeds in general have increased susceptibility to the condition (Venter and Maree, 1978). In one study preputial prolapse was reported to occur in up to 30% of 4 year-old Santa Gertrudis bulls (Venter and Maree, 1978). Bulls of *Bos indicus* breeds are reported to have a significantly longer preputial length and a larger preputial orifice compared to *Bos taurus* breeds (van den Berg, 1984). Although *Bos taurus* breeds are considered to be more resistant to preputial prolapse compared to *Bos indicus*, the Hereford breed has been identified as the *Bos taurus* breed most susceptible to prolapse (Roberts, 1986). Preputial prolapse appears to be extremely rare in Shorthorns and their crosses (Lagos and Fitzhugh, 1970), with a significant reduction in preputial prolapse being recorded in cross-bred Santa Gertrudis bulls where the maternal grandmothers were Shorthorns (Venter and Maree, 1978).

It is interesting to note that the presence of preputial ulcers was found to be statistically unrelated to eversion (Long and Dubra, 1972).

The heritability of preputial prolapse is considered to be approximately 0.35. However, the standard error of this estimate is quite high (Lagos and Fitzhugh, 1970). Despite the large standard error, it can be concluded that the incidence of preputial prolapse can be reduced by culling affected bulls. Significant differences in the incidence of preputial prolapse in the progeny of different sires have been reported (Lagos and Fitzhugh, 1970) and confirm the potential effectiveness of this method of control. By applying accepted breeding principles, it would be expected more rapid reduction in the incidence of prolapsed prepuce could be made by actively selecting bulls with positive traits in sheath and prepuce structure.

From an individual case point of view, approximately 75% of bulls treated either medically, or medically combined with surgery, were returned

to breeding use in two studies (Baxter *et al.*, 1989, Memon *et al.*, 1988) and 70% successfully returned to breeding in another study (Desrochers *et al.*, 1995). Treatment success can be expected to drop to approximately 50% if associated abscessation is present (Memon *et al.*, 1988). It is apparent that improved surgical success is achieved if posthioplasty (resection and anastomosis with the penis in full extension = reefing) is performed in a surgical facility compared to circumcision (preputial amputation) in an hydraulic crush (Desrochers *et al.*, 1995).

The cost-effectiveness of treatment alternatives for bulls with preputial prolapse has been assessed (Kasari *et al.*, 1997). This model was based on 1997 data assuming the bull had two possible breeding seasons of use left when it was injured. The analysis found that if the replacement cost was less than \$2,500 it was more cost-effective to cull a bull with preputial prolapse than to treat either medically or surgically. It was suggested that only five pieces of information were necessary to provide a suitable cost-effectiveness analysis on the basis of annual depreciation cost. These are the expected cost of treatment, bull maintenance costs, salvage value, replacement bull price, and number of remaining breeding seasons. The authors emphasised the use of objective data (EBVs/EPDs) when assessing the replacement bull price.

Several, infrequent but important penile conditions that contribute to infertility, may not be apparent unless copulation is carefully observed. These are deviation of the penis and vascular shunts from the corpus cavernosum to the corpus spongiosum, thereby preventing effective erection.

#### **Additional Methods of Disease Investigation**

##### **Semen Culture**

Semen culture is traditionally used when physical examination of the reproductive tract identifies lesions, or when white cells are noted on the examination of a semen sample (Sprecher *et al.*, 1999). However, a recent study by these authors found that semen from 94% of bulls tested prior to the commencement of the breeding season were culture positive to a range of organisms including *Hemophilus somnus*, *Mycoplasma bovis genitalium*, *Arcanobacterium pyogenes* and *Ureaplasma diversum*. In particular, the prevalence of *H. somnus* was 68%. It was suggested that these organisms may normally colonise the urethra in contrast to a

pathological colonisation of testicular or epididymal tissue. The conclusion is that semen culture results may be clinically misleading and should be evaluated carefully in association with physical examination findings and herd reproductive data.

One organism which may be an exception is *Mycoplasma bovis*. This organism is not commonly cultured from bull semen and has proven pathogenicity for the reproductive tract of cows (Kirkbride, 1987). Thus, its presence in bull semen should initiate active treatment and control measures.

##### **Serving Capacity Testing**

The serving capacity test is designed as a quantitative assessment of the ability of a bull to successfully gain intromission and ejaculation after suitable pre-stimulation. Restrained females are used and the test is run for 20 minutes to ensure abnormalities such as PSDP are not overlooked.

The logistics of running this examination are explained in detail (McGowan *et al.*, 1995).

The ideal situation would be for bulls to have this test performed annually (or at least a capacity to serve) prior to the joining period. As a minimum, it could be argued that all bulls should undergo at least two serving capacity tests in their working life. An initial test prior to the first season of use will assist in identifying many congenital, heritable and physical abnormalities that may interfere with effective copulation, in addition to assessing libido.

A second test after two or three seasons of use will help to identify, degenerative and progressive defects such as PSDP and arthritic conditions.

##### **Karyotyping**

This procedure can be used to evaluate chromosomal abnormalities. It may be useful if a bull is suspected of being a twin to a female, if Jacksonian or Robertsonian translocation defects are suspected (McFeely, 1993), or if signs of intersex are present.

##### **Analysis of the Diet**

Dietary analysis can help to identify nutritional or trace element abnormalities that may interfere with reproductive function.

Conditions that may indicate the need for dietary analysis include a high incidence of morphological

abnormalities, poor testicular tone, or lameness in a group of bulls.

### Recommendations for the Control of Reproductive Disease in the Bull

#### Vaccinations and Control of Infectious Disease

The complete disease vaccination and control programme for bulls will depend on the specific diseases that are locally active. It is recommended that attention to disease control is maintained at all stages of the bull breeding and development process since diseases such as bovine pestivirus can assert their negative influence on bull health and fertility during foetal development.

It is recommended that all breeding bulls be vaccinated against campylobacteriosis and the five clostridial diseases.

General recommendations, which will vary depending on locality, include vaccinations against leptospirosis, BEF, botulism and tick fever.

Restricted breeding seasons should be used to allow a period of sexual rest for the breeder herd, where possible. This will provide an opportunity for infections of the female reproductive tract to be overcome and reduce exposure of bulls to infertility disease.

#### Regular Breeding Soundness Evaluation

Although an annual bull BSE is ideal, there is still producer resistance to annual assessment of each bull. As a compromise, once a herd is regularly using BSE and all bulls are being assessed prior to their first season, subsequent BSE every second year may be adequate, especially on extensive properties with plenty of "bull power". An assessment of the ability of a bull to complete natural service should be made annually, even if this only entails the producer observing the bull successfully serving a cow during the breeding season.

#### Maintain a Relatively Young Bull Herd

It is suggested that bulls be kept for a maximum of five breeding seasons. This recommendation is particularly relevant to the control of the venereally transmitted infertility diseases. Both *T. foetus* and *C. fetus* sequester in the epithelial crypts of the penis and prepuce of bulls. Since the crypts do not develop in most bulls until three to four years of age,

young bulls do not usually become permanent carriers (Ball *et al* 1983). Consequently, the use of young bulls can help in the control of these diseases, particularly *T. foetus*.

There is also less risk of degenerative changes, or the progressive deterioration of some heritable conditions, affecting herd fertility when younger bulls are used. Such conditions include degenerative joint disease, testicular degeneration and premature spiral deviation of the penis.

#### Keep Age Groups Separate

From weaning to three years of age, it is recommended to separate bulls by age group (Cain, 1987). Social dominance is negligible in bulls up to two years of age (Blockey, 1979), however these younger bulls may be dominated by older bulls if placed in the same joining paddock.

#### Acclimatise the Bull

Evidence on the effect of temperature, stress and nutrition on testicular function, coupled with current knowledge of spermatogenesis can be used to formulate recommendations for bull acclimatisation. Where major changes in climate or management are anticipated, or where long transport distances are necessary, it seems reasonable to ensure bulls arrive at their working environment approximately three months prior to the breeding season.

#### References

- Arbitt B, Fiske RA, Craig TM, Bitter JW (1995), Scrotal hydrocele secondary to ascites in 28 bulls., *J. Am. Vet. Med. Assoc.* 207: 753-756.
- Austin JW, Hupp EW, Murphee RL (1961), Effect of scrotal insulation on semen of Hereford bulls., *J. Anim. Sci.* 20: 307-310.
- Bagshaw PA, Ladds PW (1974), A study of the accessory sex glands of bulls in abattoirs in northern Australia., *Aust. Vet. J.* 50: 489-495.
- Ball L (1996). Thesis. The interstitial epididymitis in bulls. Colorado State University, Fort Collins.
- Ball L, Cheney JM, Mortimer RG (1983), Diagnosis and control of herd infertility in beef cattle., *Proc. of the Soc. for Therio. Ann. Meeting.* 22-30.

- Barling K, Wikse S, Magee D, Thompson J, Field R (1997), Management of beef bulls for high fertility., *Compend.Contin.Educ.Pract.Vet.* 19: 888-893, 903.
- Baxter GM, Allen D, Wallace CE (1989), Breeding soundness of beef bulls after circumcision: 33 cases (1980-1986), *J.Am.Vet.Med.Assoc.* 194: 948-952.
- Blanchard TL, Varner DD, Bretzlaff KN (1991), Testicular degeneration in large animals: Identification and treatment., *Vet.Med.* 537-541.
- Blockey MA (1979), Observations on group matings of bulls at pasture., *Appl.Anim.Ethol.* 5: 15-34.
- Blom E (1979), Studies on seminal vesiculitis in the bull II. Malformation of the pelvic genital organs as a possible predisposing factor of seminal vesiculitis., *Nord.Vet.Med.* 31: 241-250.
- BonDurant RH (1997), Pathogenesis, diagnosis, and management of trichomoniasis in cattle., *Veterinary clinics of North America.Food animal practice* 13: 345-361.
- BonDurant RH (1997). Personal Communication. The use of wetting agents to assist local treatment of the prepuce in bulls.
- BonDurant RH, Gajadhar A, Campero CM, Johnson E, Lun ZR, Nordhausen RW, Walker RL, van Hoosear KA (1999), Preliminary characterisation of a *Tritrichomonas foetus*-like protozoan isolated from preputial smegma of virgin bulls., *Bov.Pract.* 33: 124-127.
- Bouters R, DeKeyser J, Vandeplassche M, Van Aert A, Bronte E, Bonte P (1973), *British Veterinary Journal* 129: 52.
- Bruner KA, McCraw RL, Whitacre MD, Camp Sv, Van Camp SD (1995), Breeding soundness examination of 1,952 yearling beef bulls in North Carolina., *Theriogenology* 44: 129-145.
- Cain MF (1987). Developing and Managing Herd Bulls. University of Florida, Cooperative Extension Service, Institute of Food and Agricultural Sciences.
- Carrol EJ, Aanes WA, Ball L (1964), Persistent penile frenulum in bulls., *J.Am.Vet.Med.Assoc.* 144: 747-749.
- Cavalieri J, Van Camp SD (1997), Bovine seminal vesiculitis: A review and update., *Veterinary clinics of North America.Food animal practice* 13: 233-241.
- Chenoweth PJ, Burgess GW (1972), Mid-piece abnormalities in bovine semen following ephemeral fever., *Aust.Vet.J.* 48: 37-40.
- Clark BL (1974), Control of trichomoniasis in a large herd of beef cattle., *Aust.Vet.J.* 50: 424-426.
- Clark BL, Dufty JH, Monsborough Mary J (1968), *Aust.Vet.J.* 44: 530.
- Clark BL, Dufty JH, Monsborough Mary J, Parsonson IM (1979), A dual vaccine for vaccination of bulls against vibriosis., *Aust.Vet.J.* 55: 43.
- Clark BL, Dufty JH, Parsonson IM (1983), The effect of *Tritrichomonas foetus* infection on calving rates in beef cattle., *Aust.Vet.J.* 60: 71-74.
- Coulter GH (1986), Puberty and postpuberal development of beef bulls., in *Current Therapy in Theriogenology*, ed. Morrow DA, W.B. Saunders Company, Philadelphia p 142-148.
- Desrochers A, St Jean G, Anderson DE (1995), Surgical management of preputial injuries in bulls: 51 cases (1986-1994), *Canadian Veterinary Journal* 36: 553-556.
- Doig PA, Ruhnke HL, MacKay AL (1979), Bovine granular vulvitis associated with ureaplasma infection., *Canadian Veterinary Journal* 20: 89-94.
- Eaglesome MD, Sampath MI, Garcia MM (1995), A detection assay for *Campylobacter fetus* in

- bovine semen by restriction analysis of PCR amplified DNA., *Res.Comm.* 19: 253-263.
- Elmore RE (1981), Surgical repair of persistent penile frenulum., *Vet.Med.Sm.An.Clin.* 76: 701-704.
- Felleisen RSJ, Lamelet N, Bachmann S, Nicolet J, Muller N, Gottstein B (1998), Detection of *Tritrichomonas foetus* by PCR and DNA enzyme immunoassay based on rRNA gene unit sequences., *J Clin.Microbiol.* 36: 513-519.
- Heinemann MB, Garcia JF, Nunes CM, Gregori F, Higa ZMM, Vasconcellos SA, Richtzenhain A (2000), Detection and differentiation of *Leptospira* spp. serovars in bovine semen by polymerase chain reaction and restriction fragment length polymorphism., *Vet Microbiol.* 73: 261-267.
- Hum S, Brunner J, Gardiner B (1993), Failure of therapeutic vaccination of a bull infected with *Campylobacter fetus*, *Aust.Vet.J.* 70: 386-387.
- Hum S, Quinn C, Kennedy D (1994), Diagnosis of bovine venereal campylobacteriosis by ELISA., *Aust.Vet.J.* 71: 140-143.
- Hum S, Stephens LR, Quinn C (1991), Diagnosis by ELISA of bovine abortion due to *Campylobacter fetus*., *Aust.Vet.J.* 65: 272-275.
- Humphrey JD, Ladds PW (1975), A quantitative histological study of changes in the bovine testis and epididymis associated with age., *Res.Vet.Sci.* 19: 135-141.
- Irons P (2002). Personal Communication. Antibiotics for the local treatment of campylobacteriosis in bulls.
- Kasari TR, McGrann JM, Hooper RN (1997), Cost-effectiveness analysis of treatment alternatives for beef bulls with preputial prolapse., *J.Am.Vet.Med.Assoc.* 211: 856-859.
- Kastelic JP, Cook RB, Coulter GH (1997), Scrotal/testicular thermoregulation and the effects of increased testicular temperature in the bull., *Veterinary clinics of North America.Food animal practice* 13: 271-282.
- Kirkbride CA (1987), Mycoplasma, ureaplasma, and achleplasma infections of bovine genitalia., *Veterinary clinics of North America.Food animal practice* 3: 575-591.
- Kirkland PG, Richards SG, Rothwell JT, Stanley DF (1991), Replication of bovine diarrhoea virus in the bovine reproductive tract and excretion of virus in semen during acute and chronic infections., *Veterinary Record* 128: 587-590.
- Lagos F, Fitzhugh HA (1970), Factors influencing preputial prolapse in yearling bulls., *J.Anim.Sci.* 30: 949-952.
- Lander KP (1990), The development of a transport and enrichment medium for *Campylobacter fetus*., *British Veterinary Journal* 146: 327-333.
- Larson L (1980), Physical examination of the reproductive system of the bull., in *Current Therapy in Theriogenology*, ed. Morrow DA, W.B. Saunders, Philadelphia p 307-330.
- Long SE, Dubra CR (1972), Incidence and relative clinical significance of preputial eversion in bulls., *Veterinary Record* 91: 165-169.
- Mackerras IM, Mackerras MJ, Burnet FM (1940). Experimental studies of ephemeral fever in Australian cattle. Melbourne, Australia., CSIRO.
- Madrid N, Ott RS, Rao Veeramachaneni DN, Parrett DF, Vanderwert W, Willms CL (1988), Scrotal circumference, seminal characteristics, and testicular lesions of yearling Angus bulls., *Am.J.Vet.Res.* 49: 579-585.
- Marcus S, Shore LS, Perl S, Bar-el M, Shemesh M (1997), Infertility in a cryptorchid bull: a case report., *Theriogenology* 48: 341-352.
- McCool CJ, Ladds PW (1992), Impact of diseases on fertility., in *Bull Fertility - Proceedings of a Workshop held at Rockhampton*, ed.

- Holroyd RG, DPI Queensland, Rockhampton p 70-73.
- McCool CJ, Townsend MP, Wolfe SG, Simpson MA, Olm TC, Jayawardhana GA, Carney JV (1988), Prevalence of bovine venereal disease in the Victoria River District of the Northern Territory: likely economic effects and practicable control measures., *Aust.Vet.J.* 65: 153-156.
- McEntee K (1958), Pathological conditions in old bulls with impaired fertility., *J.Am.Vet.Med.Assoc.* 132: 328-331.
- McEntee K (1990), *Reproductive pathology of domestic animals.*, Academic Press, New York p 340-343.
- McFeely RA (1993), Chromosomal Abnormalities., *Veterinary clinics of North America.Food animal practice* 9: 11-22.
- McGowan MR, Galloway D, Taylor EG, Entwistle K, Johnston P (1995), *The veterinary examination of bulls.*, Australian Association of Cattle Veterinarians, Brisbane p 57-64.
- Mellick PW, Winter AJ, McEntee K (1965), Diagnosis of vibriosis in the bull by use of the fluorescent antibody technic., *Cornell Veterinarian* 55: 280-294.
- Memon MA, Dawson LJ, Usenik EA (1988), Preputial injuries in beef bulls: 172 cases (1980-1985), *J.Am.Vet.Med.Assoc.* 193: 481-485.
- Mossman DH (1984), The Effect of Calving Patterns on the Profitability of Beef Breeding Herds., in *Beef Cattle Production*, ed. Hungerford TG, The Post-Graduate Committee in Veterinary Science, Sydney p 216-222.
- Mossman DH, Hanly GJ (1977), A theory of beef production., *N.Z.Vet.J.* 25: 96-100.
- Nielsen IL (1972), An outbreak of bovine posthitis., *Aust.Vet.J.* 48: 39-40.
- Noordsy JL (1989), Surgically correcting penile and prepuccial conditions in the bull., in *Food Animal Surgery*, ed. Noordsy JL, p 218-220.
- Panangala VS, Winter AJ, Wijesinha A (1981), Decreased motility of bull spermatozoa caused by *Mycoplasma bovis* genitalium., *Am.J.Vet.Res.* 42: 2090-2093.
- Parsonson IM, Hall CE, Settergren I (1971), A method for the collection of bovine seminal vesicle secretions for microbiological examination., *J.Am.Vet.Med.Assoc.* 158: 175-177.
- Pearson H (1972), Surgery of the male genital tract in cattle: A review of 121 cases, *Veterinary Record* 91: 498-502.
- Queensland Department of Primary Industries (2002). Chilled tick fever vaccine. Tick Fever Research Centre, Queensland DPI, Brisbane.
- Rae DO (1989), Impact of trichomoniasis on the cow-calf producer's profitability., *J.Am.Vet.Med.Assoc.* 194: 771-775.
- Reit Correa F, de Freitas A, de Puignau MUR, Pedomo E (1979), Ulcerative posthitis in bulls in Uruguay., *Cornell Veterinarian* 69: 33-34.
- Riley DE, Wagner B, Polley L (1995), PCR-based study of conserved and variable DNA sequences of *Tritrichomonas foetus* isolates from Saskatchewan, Canada., *J Clin.Microbiol.* 33: 1308-1313.
- Roberts SJ (1986), *Veterinary Obstetrics and Genital Diseases (Theriogenology)*, S.J.Roberts., Woodstock, Vermont.
- Ross AD. Heat stress and testicular morphology in the bull. 1977. James Cook University of North Queensland.
- Spradbrow PB, Francis J (1969), Observations on bovine ephemeral fever and isolation of virus., *Aust.Vet.J.* 45: 525-531.
- Sprecher DJ, Coe PH, Walker RD (1999), Relationships among seminal culture, seminal white blood cells, and the percentage of primary sperm abnormalities in bulls evaluated prior to the breeding season., *Theriogenology* 51: 1197-1206.



- St George TD (1986), The epidemiology of bovine ephemeral fever in Australia and its economic effect., in *Arbovirus Research in Australia - Proceedings Fourth Symposium*, ed. St George TD, CSIRO, Brisbane, Australia. p 303-311.
- St Jean G (1995), Male reproductive surgery, *Veterinary clinics of North America. Food animal practice* 11: 55-93.
- St Jean G, Gaughan EM, Constable PD (1992), Cryptorchidism in North American cattle: breed predisposition and clinical findings., *Theriogenology* 38: 951-958.
- Steffen D (1997), Genetic causes of bull infertility., *Veterinary clinics of North America. Food animal practice* 13: 243-253.
- Uren MF (1989), Bovine ephemeral fever., *Aust.Vet.J.* 66: 233-236.
- Van Camp SD (1997), Common causes of infertility in the bull., *Veterinary clinics of North America. Food animal practice* 13: 203-231.
- van den Berg SS (1984), An investigation into the etiology of prolapsing lamina interna in the Brahman and Santa Gertrudis breeds and the correction thereof., *Proceedings 13th World Cong Dis Cattle* 2: 745-746.
- Vasquez LA, Ball L, Bennett BW, Rupp GP, Ellis R, Olson JD, Huffman MH (1983), Bovine genital campylobacteriosis (vibriosis): vaccination of experimentally infected bulls., *Am.J.Vet.Res.* 44: 1553.
- Venter HAW, Maree C (1978), Factors affecting prolapse of the prepuce in bulls., *J.South Afr.Vet.Assoc.* 49: 309-311.
- Weber JA, Hilt CJ, Woods GL (1988), Ultrasonographic appearance of bull accessory sex glands., *Theriogenology* 29: 1347-1355.
- Welles EG, Tyler JW, Wolfe DF, Moore A (1995), Eperythrozoon infection in young bulls with scrotal and hindlimb edema, a herd outbreak., *Theriogenology* 43: 557-567.
- Wolfe DF, Hudson RD, Walker DF (1983), Common penile and preputial problems of bulls., *Compend.Contin.Educ.Pract.Vet.* 5: 447-455.