impairment of vascular supply to the terminal small colon and proximal rectum, as no macroscopic evidence of infarction was present at necropsy.

The mare's progressive deterioration in condition following resection and anastomosis resulted from leakage of faecal material through the anastomosis, into the peritoneal cavity. All anastomoses leak to some extent (Peacock 1984), but in the majority of cases containment and resolution occurs with few systemic signs. In this mare, sufficient leakage occurred to result in acute, diffuse peritonitis with concomitant signs of septic shock. Therefore, a diverting colostomy was performed to reduce peritoneal contamination.

Colostomies have received little attention from equine surgeons in the past. Stashak and Knight (1978) reported the successful management of a grade 3 rectal tear with an end colostomy. Post-operative management included intra-colostomy enemas, regular cleaning and application of protectant ointment to the stoma, antibiotics, and modification of the diet. Herthel (1974) used a similar surgical technique with success in treating a grade 4 rectal tear. In both reports, atrophy of the distal stump is mentioned as a complication which makes closure difficult. Herthel (1974) stated that it was necessary to leave 60 cm of bowel proximal to the rectal tear to facilitate closure of the colostomy.

Azzie (1975) and Spiers et al (1980), in treating horses with rectal tears, performed loop colostomies. Azzie (1975) treated 2 cases, with Case 2 dying from haemorrhage associated with prolapse of the colostomy. In order to avoid this, nylon tape tension sutures were placed in the skin across the colostomy site in Case 1. In the latter, no prolapse occurred and treatment was successful. Spiers et al (1980) performed loop colostomies on 3 horses. Two of the 3 had complications associated with the colostomies (severe mucosal oedema at the colostomy site, with herniation through the colostomy stoma in one case and mucosal prolapse in another).

Mucosal prolapse was the major complication associated with colostomy in this mare. Birnbaum and Ferrier (1952) list 7 factors involved in the prolapse of a colostomy. Of these factors, an excessively large incision in the abdominal wall was considered the most important in this case. However, the relative contribution of other factors is difficult to determine in the absence of sufficient case reports in horses. Saha et al (1973) stated that in man, a colostomy wound which admits more than 2 fingers is liable to prolapse. We are not able to state with certainty, but it is possible that mucosal prolapse is more likely to occur in horses than man. If this is true then further work is required to develop optimal techniques for the creation of colostomies in horses. The progression from acute, diffuse peritonitis on presentation to a localised, fibrinous peritonitis at necropsy and the absence of faeces passed via the anus confirmed that the colostomy diverted faecal material from the anastomosis.

At necropsy intestinal obstruction, secondary to entrapment of a loop of small intestinal by fibrin in the region of the anastomosis, was the cause of acute abdominal pain, which necessitated destruction of the animal. Of the factors listed by Peacock (1984) infection was regarded as the major cause of adhesions. Thus, a colostomy performed earlier may have reduced the tendency to form adhesions.

In conclusion, the prognosis for types 3 and 4 rectal prolapse remains poor. Successful treatment relies on an intact vascular supply to the remaining small colon and rectum following resection and anastomosis of the compromised portion. In addition, colostomies will be useful in the short term to divert faecal material from compromised bowel or to enable healing of the anastomosis, once techniques for creation and management are improved.

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Blue heliotrope (Heliotropium amplexicaule) poisoning in cattle

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Heliotropium amplexicaule (blue heliotrope, wild verbena) is a native of South America which is naturalised in southern Queensland, northern New South Wales and South Australia (Everist 1981, Jacobs and Pickard 1981). A perennial plant which dies back during winter, it grows along roadsides, in cultivated ground and in pastures. It grows especially well in red loam soils and can be a serious pest of pastures and crops (Kleinschmidt and Johnson 1979; Everist 1981). Indicine, a pyrrolizidine alkaloid, has been isolated from H. amplexicaule (Bull et al 1968). The plant was suspected in one instance of causing toxica jaundice in sheep but very small amounts of alkaloids were demonstrated in samples (Everist 1981).

The south Burnett district of southern Queensland had red loam soils. Over the last 8 years several cases of poisoning with liver pathology consistent with pyrrolizidine alkaloid poisoning have occurred in cattle in this area. In some of these cases H. amplexicaule was suspected as the source of intoxication. This paper records 4 of these cases.

Case 1: A 4-year-old Murray Grey bull from a herd of 40 cattle was sent to the abattoir in winter (July) following chronic wasting together with weakness and incoordination. During the preceding 2 years this bull had been grazing a 4 ha paddock containing kikuyu, clover, native grasses and H. amplexicaule. H. amplexicaule dominated the pasture during the summer and autumn. A botanical survey of the paddock was carried out and of 38 plant specimens identified only H. amplexicaule was recognised as a possible toxic species containing pyrrolizidine alkaloids.

Case 2: A 4-year-old Hereford crossbred cow from a group of 5 cattle died in winter (August) following signs of compulsive walking into and through obstructions as if blind. The farm was small (3.3 ha) and the cattle grazed in a 2 ha paddock adjoining the farm of Case 1. Pasture conditions were similar. Four cattle had died with similar signs on this farm in the previous 2 years.
Case 3: this occurred on the same farm as Case 2 but 2.5 years later in summer (February). Of 14 crossbred beef cattle 12 to 15 months of age, 5 were scouring profusely, with wasting and photosensitisation. Three died after 7 days and 2 survivors were sent to the abattoir but were condemned as unfit for human consumption. These cattle had been brought onto the farm 4 months previously. H. amplexicaule dominated the pasture and cattle were seen grazing the plant.

Case 4: Two 4-month-old Friesian calves became severely ill in summer (February) and after 3 days showed signs of anorexia, ruminall atony and tenesmus without passing faeces. The injurious area was infected and jaundiced. Two steers and 2 cows in the same 2.3 ha paddock were unaffected. The calves had been weaned at 6 weeks of age, with the aid of a supplement, onto pasture which consisted of a luxuriant growth of kikuyu, white clover, native grasses and H. amplexicaule. The calves were observed to eat H. amplexicaule as well as the other pasture species. The paddock was regularly fertilised with urea after rainfall. The calves were killed and post mortem examinations carried out.

Livers of affected cattle varied from pale with nodular surface and fibrous texture (Case 1) to yellow with rounded edges and firm texture (Case 2). Urethra and bladder wall of Case 1 had fibrous nodular thickening. Jaundice was seen in Case 2 and Case 4 and ascites was present in the yearlings and calves of Cases 3 and 4.

Histologically livers of all cases had extensive hyperplasia of bile ducts and a variable degree of periporal and centrilobular fibrosis with fibrous dissection of the lobules. Hepatocytes showed pronounced megalocytosis. Focal fatty degeneration and haemorrhage were present in Cases 3 and 4 and regenerative nodules of normal hepatocytes in Case 1. Megalocytosis of renal cortical epithelium was evident only in Cases 1 and 2 and was most prominent in Case 2. Cerebral cortex of Case 2 had large vacuoles in the neuropil of the deep laminae of the grey matter (status spongiosus).

Plant material was assayed by a modification of the method of Culvenor and Smith (1955). Duplicate plant samples consisting of stems with attached leaves and flowers were collected from the farm of Case 4 in January 1986 and immediately immersed in ethanol. A third sample collected at the same time was used to confirm the botanical identity. The plant material for alkaloid assay was macerated twice with ethanol and finally extracted with hot ethanol for 2 h. Alkaloid content of the first sample was 0.16% free base and 4.22% N-oxide. For the second sample, only total base content was determined, and this was 4.60%.

Thin layer chromatography (methanol on silica gel) and mass spectrometry showed that the alkaloid was essentially pure indicine, with a trace of an unknown alkaloid. The crude alkaloid was purified from achromic material to give colourless prisms, m.p. 97-98° (lit. 97-98°, Mattocks et al. 1961).

The variable clinical signs observed in cattle in this series of cases are similar to those described by Blood et al. (1983) for pyrrolizidine alkaloid toxicity in domestic herbivores. Neurological signs, photosensitisation, scouring and tenesmus were seen. Depressive and staggering gait were seen in Case 1 and in Case 2 the more severe "walkabout" syndrome occurred. Examination of records of cattle deaths on the farm of Case 2 showed that several cases with signs of depression and shuffling gait or circling with limb stiffness and blindness had occurred in the past. Because of these clinical signs sporadic (chlamydial) bovine encephalitis and lead poisoning had been suspected but in retrospect these cases were probably H. amplexicaule poisoning.

The microscopic pathology of the liver and kidney seen in affected cattle was consistent with pyrrolizidine alkaloid poisoning (Hooper 1978; Jubb et al. 1985). The vaculation of the cerebral cortex seen in Case 2 also is consistent with hepatoxic encephalopathy caused by pyrrolizidine alkaloids. (Hooper et al. 1974).

The concentration of indicine present in H. amplexicaule from Case 4 (0.16% free base, 4.22% N-oxide) is substantially higher than that detected in a previous analysis of this plant (0.10% free base, 1.50% N-oxide; C C J Culvenor and L W Smith unpublished). The alkaloid concentration is also higher than in most other species containing pyrrolizidine alkaloids; the total alkaloid concentration in H. europaeum is commonly in the range 1 to 2%, although 3.1% has been recorded (Crowley and Culvenor 1956). The frequent application of nitrogenous fertiliser to the pasture may have contributed to the high plant alkaloid concentration (Everist 1981). The immediate collection of samples into ethanol in Case 4 maximised preservation of alkaloids whereas drying of the plant in the earlier study may have caused alkaloid loss (Bull et al. 1968). Everist's report (1981) of trace amounts of alkaloid in H. amplexicaule associated with a case of toxicity in sheep in New Zealand was possible due to an alkaloid assay which did not detect N-oxide. The free base and N-oxide contents of ingested material are both relevant to toxicity, since N-oxides are largely reduced in the rumen of sheep and cattle (Dick et al. 1963; Lanigan 1970). Indicine and its N-oxide are both of low acute toxicity in laboratory animals. Indicine did not induce liver lesions in the rat at a dose level of 1000mg/kg intraperitoneally (Schoental 1968; Mattocks 1972), whereas the acute lethal dose of indicine N-oxide is about 2,400mg/kg intraperitoneally in mice and 1200mg/kg intravenously in dogs (Castles et al. 1976). Chronic effects have not been reported for indicine although the feeding of Heliotropium indicum, another species known to contain indicine, produced liver lesions in rats (Schoental 1968). A mixture of the closely related isomeric alkaloids, intermediode and lycopsamine, (an extract of Ammernia intermedia seeds) was reported to give liver lesions in rats 4 to 6 months after a single oral dose of 500mg/kg (Fowler and Schoental 1967). In general terms cattle are 10 times as susceptible as mice and 4 times as susceptible as rats to pyrrolizidine alkaloids (Hooper 1978).

H. europaeum has been reported to cause pyrrolizidine alkaloid poisoning in cattle (Bull et al. 1961; Wiltjer and Walker 1974). Cattle forced to eat the plant under drought conditions can die within a few weeks. H. amplexicaule has rarely been suspected of causing poisoning in domestic herbivores. Like H. europaeum it was considered to be seldom eaten because of low palatability (Everist 1981). However, in 3 of the circumstances described alternative feed was available and only in one was there evidence of grazing pressure due to overstocking. Young cattle were affected in Cases 3 and 4 after only 4 and 2.5 months of exposure, respectively. These cases occurred in January-February when H. amplexicaule growth is most luxuriant.

Young cattle may be less discriminating as well as more susceptible to toxicity. Cases 1 and 2 which involved adult cattle may have been cases of cumulative poisoning over more than one season. All 4 cases occurred on red soil headlands which had been cultivated in the past. H. amplexicaule is an aggressive coloniser on similar neglected cultivation through-out the south Burnett district and deaths in cattle attributed to H. amplexicaule are reported almost annually. The evidence presented now supports the claim that H. amplexicaule toxicity can be of significance.

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References


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Medical therapy of urinary incontinence in ovarioctomised bitches: a comparison of the effectiveness of diethylstilboestrol and pseudoephedrine

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Urinary incontinence is a troublesome condition in bitches and the problem is more prevalent in ovarioctomised bitches (Ruckstuhl 1978; Thrusfield 1985). Affected bitches show dribbling incontinence which may be continuous or intermittent but is often more noticeable when they are recumbent or asleep (Holt 1985).

The etiology of the condition is not clear but it has long been recognised that the condition improves when bitches are oestrogen deficient and the term ‘oestrogen-responsive’ incontinence has been used to describe this disease (Osborne et al 1980). Urethral pressure profile studies have shown that many incontinent bitches have lowered maximum urethral closure pressures and incontinence is considered to be due to primary sphincter incompetence (Richter and Ling 1985; Holt 1985).

Treatment of incontinent bitches with oestrogens has been poorly documented. In a series of cases this treatment has been standard therapy for many years but the effectiveness of the therapy has been poorly documented. In a series of 39 cases treated with oestrogen therapy Holt (1985) reported that 23 showed no improvement, 9 partially responded and 7 dogs became continent.

The use of α-adrenergic drugs to treat urinary incontinence has been described (Rosin and Ross 1981; Adams and DiBartola 1983). In a series of 11 clinical cases treated with phenylpropanolamine Richter and Ling (1985) reported that the condition resolved in 10 dogs and was considerably improved in the other.

This paper reviews the results of medical therapy for urinary incontinence in bitches. This study used the case records of ovarioctomised bitches treated at Murdoch University Veterinary Hospital for urinary incontinence. Cases which had a history of dribbling incontinence and no abnormalities involving the urinary tract were included in the study. Bitches with other signs of urinary tract disease such as dysuria, haematuria or change in frequency of urination were excluded.

Details of therapy and response to therapy were extracted from the case records and the owners were contacted by telephone if the record did not contain adequate information on the effectiveness of the treatment, or the occurrence of side-effects. The shortest follow-up time was 9 months and the longest was 6 years. Over 6 years, 45 bitches were treated and the results of therapy were known for 40 cases.

Two treatments were used. Diethylstilboestrol was given orally at a dose of 1 mg daily for 3 to 7 days followed by a maintenance dose of 1 mg weekly. Although all bitches received the same initial dose, some owners increased the frequency of the maintenance dose when they found the prescribed dose was inadequate. In bitches weighing over 25 kg, 30 mg of pseudoephedrine was given thrice daily, or for those bitches in which the owners reported the problem was only nocturnal, 30 mg was prescribed only at night. Bitches weighing less than 25 kg were given 15 mg thrice daily. Eight of the bitches which were initially treated with stilboestrol were subsequently treated with pseudoephedrine. In 7 cases this was due to a poor response to stilboestrol therapy and in one case it was due to the bitch attracting male dogs.

Urinary incontinence mainly affected large dogs. Twenty-five dogs weighed more than 25 kg, 10 weighed between 12.5 and 25 kg and 5 weighed less than 12.5 kg. The distribution of the weights of the dogs was similar in the groups treated with stilboestrol and pseudoephedrine.

The age of onset of the disease ranged from 1 to 14 years (mean 7.6) for the group treated with stilboestrol and 1 to 15 years (mean 6.1) for the group treated with pseudoephedrine.

The duration of clinical signs prior to treatment was known for 34 dogs and it ranged from <1 month to 48 months. Eight bitches had a history shorter than 2 months, 17 had a history longer than 2 months but less than 12 months and 9 dogs had had the condition for over 12 months. The mean duration of clinical signs for the group treated with stilboestrol was shorter than for the group treated with pseudoephedrine (8.6 y 14.8 months).

The response to therapy was graded as totally effective if urinary incontinence was restored and partially effective if the condition significantly improved without reaching total continence. The results of treatment are given in Table 1.

<table>
<thead>
<tr>
<th>Response</th>
<th>Diethylstilboestrol</th>
<th>Pseudoephedrine</th>
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</thead>
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<tr>
<td>Totalley effective</td>
<td>20/31 (64.5%)</td>
<td>14/17 (82.4%)</td>
</tr>
<tr>
<td>Partially effective</td>
<td>7/31 (22.6%)</td>
<td>9/17 (52.9%)</td>
</tr>
<tr>
<td>Not effective</td>
<td>4/31 (12.9%)</td>
<td>3/17 (17.6%)</td>
</tr>
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</table>

Side-effects were not a major problem for most owners. One owner discontinued the administration of stilboestrol because the bitch was attracting male dogs. None of the dogs developed clinical problems with bone marrow suppression despite the fact that some owners administered doses of stilboestrol in excess of those prescribed. No adverse responses were noted with the use of pseudoephedrine although care was taken not to give the drug to dogs with signs of cardiovascular disease.

Normal urethral function is necessary to maintain urinary continence in bitches. This involves urethral muscle tone sufficient to occlude the lumen and counteract pressure within the bladder. The mechanisms controlling urethral function are incompletely understood but it is recognised that oestrogen and α-adrenergic innervation via the hypogastric nerves have important actions on the canine urethra (Cred 1983).

Urinary incontinence is recognized as being more common in ovarioctomised bitches but Richter and Ling (1985) measured plasma oestrogen levels and found no difference between a group of incontinent ovarioctomised bitches and a group of continent entire bitches in anoestruum.

Medical therapy with diethylstilboestrol or with pseudoephedrine produced a satisfactory response in the majority of cases. The results of oestrogen treatment were better than those reported by Holt (1985). However he indicates that many of his cases were referred after failure of therapy. The results of