CASE REPORT

Nitrate-nitrite toxicity in cattle and sheep grazing *Dactyloctenium radulans* (button grass) in stockyards

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Hungry cattle and sheep introduced to stockyards containing a dominant or pure growth of *Dactyloctenium radulans* (button grass) suffered acute nitrate-nitrite toxicity in four incidents in inland Queensland between 1993 and 2001. Deaths ranged from 16 to 44%. Methaemoglobinaemia was noted at necropsies in all incidents. An aqueous humour sample from one dead steer contained 75 mg nitrate/L and from one dead sheep contained 100 mg nitrate and 50 mg nitrite/L (normal = ca 5 mg nitrate/L). Both lush and dry button grass were toxic. The nitrate content of button grass from within the stockyards ranged from 4.0 to 12.9% as potassium nitrate equivalent in dry matter and from outside the stockyards ranged from <0.2 to 0.4%. These data suggest that urine and faeces in stockyard soil may boost the nitrate content of button grass to a concentration hazardous to hungry ruminants.

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Dactyloctenium radulans (button grass) (Figure 1) is a native, tufted, annual pasture grass confined to Australia and distributed widely in inland areas of all mainland states.[1, 2] It is the most common and widespread of the five species of *Dactyloctenium* known from Australia[2] and has been known as *Eleusine aegyptiaca* in early literature.[1] Everist[1] reports *D radulans* as toxic to ruminants and that the poisonous principal is "probably nitrate". He reported the first recorded case of poisoning by *D radulans* in sheep near Blackall on 24 February 1937 but could not establish the toxin responsible, obtaining a negative picric acid test result for cyanogenic glycosides from the plant at the site and on the day of the deaths.[3] We report here three fatal poisoning incidents in cattle and one in sheep that ate this grass at widely separate locations in inland Queensland. Our data support nitrate-nitrite as the cause of the deaths and suggest that its growth in stockyards, in particular, makes *D radulans* potentially lethal for hungry ruminants.

Laboratory methods

Plant and aqueous humour nitrate and nitrite concentrations were determined colorimetrically using nitrate and nitrite test strips (Merckoquant®; Merck, Darmstadt, Germany). The directions for analysis were carefully followed with the nitrate strip being assessed after exactly 1 min and the nitrite strip assessed after 15 s. To improve the consistency of visual interpretation the plant extracts were homogenised in water and, if required, diluted to provide a solution in the working range of the nitrate strips (25 to 250 mg/L) and the colour was assessed for all tests by one person. The precision of this semiquantitative method was regu-

Figure 1. *Dactyloctenium radulans* (button grass). Scanned specimen from the same collection as the Queensland Herbarium voucher specimen AQ551797.

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Table 1. Nitrate content of *Dactyloctenium radulans* in stockyards and elsewhere and in four associated livestock poisoning incidents in Queensland 1993-2001 compared with one historical record (1976).

Locality and date	State and location of plant sampled	%KNO_3 in plant dry matter[a]	Animals affected[b]	Aqueous humour nitrate/nitrite (mg/L)[c]	Data sources and herbarium voucher numbers
Roma, 1976	Plant state unrecorded; stockyards	5.0 to 6.5	Cattle; no mortality data available	Not sampled	SG Knott, unpublished data, Queensland Poisonous Plants Committee minutes 15 October 1976
Case A: Cloncurry, March 1993	Drying; stockyards	8.1	Cattle D15/34 (44%)	Not sampled	Department of Primary Industries, Queensland records
	[d]*Medicago sativa* (lucerne) hay fed to affected group	< 0.08, < 0.08, 0.20 (n = 3)	No adverse effects		Department of Primary Industries, Queensland, records
	[d]Dry; stockyards of neighbouring property	4.8	No animals exposed		Department of Primary Industries, Queensland, records
Case B: Kynuna, March 1993	Dry; stockyards	12.9	Cattle D13/80 (16%)	Not sampled	Department of Primary Industries, Queensland, records
	[d]Dry; *ca* 100 m outside stockyards	< 0.2	No animals exposed		Department of Primary Industries, Queensland, records
Case C: Richmond, May 1994	Dry; stockyards	4.8	Cattle D11/152 (7%)	nitrate: 75 (n = 1)	Department of Primary Industries, Queensland, records
Case D: Longreach, December 2001	Lush; stockyards	4.0, 4.0, 10.0 (n = 3)	Sheep D250/600 (42%)	nitrate: 100 nitrite: 50 (n = 1)	Department of Primary Industries, Queensland, records Queensland Herbarium AQ551792
Richmond, January 2002	[d]Lush, stockyards	1.2, 1.6, 2.4 (n = 3)	No animals exposed		Department of Primary Industries, Queensland, records Queensland Herbarium AQ551793, AQ551794 & AQ551795
	[d]Lush, roadside	0.2, 0.4 (n = 2)	No animals exposed		Department of Primary Industries, Queensland, records Queensland Herbarium AQ551796 & AQ551797

[a]Plant concentrations of nitrate hazardous to ruminants exceed 1.5-2.0% KNO_3 in dry matter.[7,13]
[b]D[ead] animals/number exposed
[c]Normal concentrations of nitrate in cattle aqueous humour are about 5 mg/L.[16]
[d]Not associated with stock deaths

larly assessed using standard nitrate solutions (50 and 100 mg/L) prepared from analytical grade KNO_3. Also, the test strip method was initially validated by comparing results with those obtained using a nitrate ion-specific electrode (Townson Nitrate Selective Ion Electrode, Townson & Mercer Pty Ltd, Lane Cove, Sydney) coupled to a pH meter (TPS Pty Ltd, Springwood, Brisbane). Plant material for analysis was dried at 80ºC for 4 h, then milled to pass through a 2 mm sieve. One gram of this material was then blended (Sorvall blender) with 100 mL distilled water for 1 min, then assayed using the test strips. Plant nitrate is reported as % KNO_3 in dry matter. Plasma cholinesterase activity was determined as acetylcholinesterase based on a method of Ellman.[4] Clinical chemistry serum profiles were obtained using an automated analytical system (Olympus Reply®). Calcium, magnesium, total protein, total bilirubin, creatinine, urea, γ-glutamyl transferase, creatine kinase and aspartate aminotransferase were assayed using commercial kits (Thermo Trace, South Oakleigh, Victoria) and albumin and glutamate dehydrogenase with other kits (Randox Laboratories Ltd, Antrim, UK). Haematological profiles were obtained by microhaematocrit and automated electronic examinations for haemoglobin, packed cell volume, erythrocyte and leucocyte counts and erythrocyte indices. Tissue samples were fixed in 10% formalin, processed by routine methods and haematoxylon-eosin sections were examined by light microscopy.

Case histories and laboratory findings

Case A: Cloncurry

A group of 34 Brahman-cross steers, 2 years old, in strong store condition, purchased at Charters Towers saleyards on 8/9 March 1993, were trucked 635 km to Cloncurry. There they were found to be infested with cattle ticks (*Boophilus microplus*) and dipped in a mixture of cypermethrin and chlorfenvinphos (Barricade S® Cattle Dip, Fort Dodge, Australia) at 1500 h on 13 March, having been fed meanwhile on average quality grass hay. Then they were trucked to a nearby property and unloaded into the stockyards at 2030 h, about 20 h after last being fed. The soil surface of the yards was almost covered by dry (yellow) and drying (green) *D radulans* in equal proportions. Subsequent inspection revealed no other plant nitrate sources, such as *Portulaca oleracea* (pigweed). Lucerne hay (*Medicago sativa*) was offered as feed. Early next morning, 15 were found dead or dying, with dyspnoea, pale mucous membranes, tachycardia (110 to 160 beats/min), fine muscle tremors, staggering and collapse. Venous blood samples were collected from five affected cattle for clinical chemistry profiling and cholinesterase assays and from four of these for haematological profiling. No significant abnormality was found in the profiles. Serum cholinesterase activities ranged between 117 and 177 IU/L (normal > 100 IU/L). Necropsy of two recently-dead cattle revealed chocolate-brown discoloration of

blood but no other abnormalities. Liver and kidney specimens from each were fixed in formalin. No lesion was detected histologically. A sample of whole *D radulans* plants from the stockyards, a second, similar sample from stockyards at a neighbouring property and three samples of the lucerne hay fed to the affected group were collected on 15 March for nitrate assay. Results are given in Table 1.

Case B: Kynuna

A group of 80 Brahman cattle, 8 to 10 months old, of both sexes had been held in bare stockyards near Kynuna (180 km south-east of Cloncurry) for 1.5 days before being released into another yard, containing dry *D radulans* and *Cenchrus ciliaris* (buffel grass), on 25 March 1993. Four to six hours later some became ill, with staggering, muscle tremor, slight salivation, dyspnoea and collapse. Thirteen died soon afterwards. Necropsy revealed chocolate-brown discoloration of blood. Venous blood samples were collected on 26 March for clinical chemistry profiling from six surviving cattle, including two that had been affected and recovered. No significant abnormality was found. A sample of whole *D radulans* plants was collected from the yard containing the dead cattle and a second sample was collected some 100 m outside the yards. The nitrate content of these is given in Table 1.

Case C: Richmond

A group of 152 Brahman steers, 1 to 2 years old, from a property near Charters Towers were trucked 400 km west to Maxwellton and dipped there in a mixture of cypermethrin and chlorfenvinphos (Barricade S®) before being trucked into the cattle tick-free area. They were then trucked to a nearby property in the Richmond area and unloaded into stockyards with the soil surface covered by dry *D radulans* on 10 April 1994 after about 2 days without being fed. Early on the morning of 11 April five were found dead and six others were ill, two of which died within 2 h. The manager released the surviving cattle from the yards into a large paddock making them unavailable for inspection. Necropsy of the five dead steers revealed chocolate-brown discoloration of blood. Liver, kidney, lung, heart and spleen specimens from one steer were fixed in formalin. No lesion was detected histologically. An aqueous humour sample was collected for nitrate assay. Samples of whole *D radulans* plants were collected from the yards containing the dead cattle. Results of plant and aqueous humour assays are given in Table 1.

Case D: Longreach

A group of 600 Merino wethers, 4 to 6 months old, was trucked to a property near Longreach from New South Wales, arriving on 14 December 2001 after being on the trucks for over 48 h without feed. They were unloaded into stockyards with the soil surface covered by a thick growth of lush *D radulans* which they consumed avidly. The area had received 150 mm of rain three weeks before. Within about 3 h of arrival 250 wethers were dead. Necropsy of two about an hour after death revealed cyanotic mucus membranes, congested tissues, brown discoloration of blood and rumens distended with *D radulans*. One sheep had very pink skeletal muscles. Liver, kidney, heart, lung, spleen, skeletal muscle and cerebral cortex samples were fixed for histopathological examination. No lesion was detected. An aqueous humour sample was collected from one dead sheep and rumen samples from two dead sheep for nitrate and nitrite assay. A rapid qualitative assessment, using test strips directly, strongly indicated the presence of both nitrate and nitrite in one of the two rumen samples. A specimen of grass from the ungrazed area of the stockyards was identified as *D radulans* (Queensland Herbarium voucher AQ551792). Three, air-dried, whole plant samples of the grass from the same area were assayed for nitrate. Results of plant and aqueous humour assays are given in Table 1. A few small pigweed plants *(P oleracea)* were present when the stockyards were inspected later in the investigation, but their biomass was insignificant compared with that of *D radulans*.

Comparative button grass samples

Whole plant samples of *D radulans* from the stockyards of a grazing property near Richmond (different from Case C) (Queensland Herbarium vouchers AQ551793, AQ551794, AQ551795) and from a roadside elsewhere on the property (Queensland Herbarium vouchers AQ551796, AQ551797) were collected on 15 January 2002 and submitted immediately for nitrate assay to compare the nitrate content of the grass from environments with and without heavy manure loads, respectively. The results are given in Table 1.

Discussion

These findings confirm the toxic potential of *D radulans* for ruminants when it grows in stockyard soil heavily manured with nitrogen-rich urine and faeces. They establish beyond reasonable doubt that nitrate-nitrite is responsible for this toxicity. Nevertheless, *D radulans* is regarded as a valuable ephemeral, summer-growing pasture grass of inland regions, being highly palatable to sheep.[5,6] But this characteristic meant that the hungry sheep in Case D were uninhibited in their consumption of the plant on offer in the stockyards, producing a very rapid intake. This was also the case in the 1937 sheep mortality reported by Everist.[3] Nitrate-nitrite poisoning in ruminants[7] follows conversion by ruminal bacteria of nitrate to nitrite, then absorption of nitrite into the blood and its reduction of haemoglobin to non-oxygen-carrying methaemoglobin, thus producing profound tissue hypoxia and rapid death. Important factors predisposing to toxicity include rapid intake of the hazardous plants so that the ruminal microbial capacity to convert nitrite to ammonia is grossly exceeded.

D radulans has been suspected of toxicity for hungry ruminants since at least 1937 when three field cases in hungry sheep were described briefly by Queensland Government botanists[3,8] and experimental feeding of sheep was conducted at Yeerongpilly, Brisbane, under the aegis of the Queensland Poisonous Plants Committee using plant collected in the Clermont district (unpublished records 1938). One sheep was dosed by stomach tube with a watery extract from 0.9 kg of the plant daily for 5 days with the addition on days 4 and 5 of 0.23 kg and 0.68 kg, respectively, of the extracted grass. During the following 5 days the sheep was dosed with a total of 3.4 kg of whole plant. No ill effects were produced. A second sheep was dosed with 14 kg over 3 days, again without ill effect. In further experiments in the same year in Townsville, neither 2.3 kg of locally-collected plant dosed to a sheep over 14 days, nor the grazing of sheep on a local pure sward of the plant produced signs of intoxication. Nitrate in plants was not then generally recognised as hazardous to hungry ruminants and the experimental conditions used appear, in retrospect, to be probably incapable of inducing nitrate-nitrite intoxication. Either the dose and rate of intake of plant fed or its nitrate content was too small. The discovery that plant nitrate could be converted to nitrite and thus become toxic for grazing ruminants was made in South Africa during *Tribulus terrestris* toxicity research[9] and confirmed in North America during investigations

of poisonous oaten hay[10] and in Queensland during investigations of poisoning incidents with cattle consuming *Salvia reflexa*.[11]

Published data on the nitrate content of *D radulans* is scanty. McBarron[12] reported weak reactions in the diphenylamine spot test in two of six samples from New South Wales. Everist[1] cited a report by SG Knott (unpublished data from Department of Primary Industries, Queensland, records quoted in the minutes of the Queensland Poisonous Plants Committee 15 October 1976) of 5.0 to 6.5% KNO_3 in dry matter of samples from Roma associated with *P oleracea* (6.8 to 8.7% KNO_3) and *Urochloa panicoides* (liverseed grass) (5.5% KNO_3) in deaths of cattle starved for 3 days before eating the plants. These data are included in Table 1 for comparison with our cases. The contrast in nitrate content of *D radulans* (Table 1) collected from stockyards (mean 5.4% KNO_3 in dry matter; n = 12) and *D radulans* collected elsewhere (mean 0.3%; n = 3) is striking. The almost two-fold difference between the nitrate content of *D radulans* from the site of Case A and that from the neighbouring property's stockyards may reflect the greater numbers of cattle handled through the former yards.

Nitrate concentrations in plant dry matter greater than 1.5 to 2% KNO_3 are widely regarded as potentially hazardous to grazing ruminants.[13, 7] These guideline values were based on an oral LD_{50} estimate for cattle of about 0.3 g/kg body weight for nitrate drenched as an aqueous solution.[13] The oral LD_{50} in cattle for nitrate fed in plant material has been estimated as 1.0 g/kg body weight.[14] This difference largely reflects the lower rate of intake of plant material and its slower microbial digestion compared with that of an aqueous drench. Calculation from the LD_{50} value for fed plant material suggests that the guideline value for nitrate concentrations in plant dry matter above which plants may be hazardous to ruminants is 5% KNO_3. However, the issue is complicated by factors influencing the rate of microbial metabolism of nitrate and nitrite in the rumen, including recent feeding history (fasting animals are more susceptible), prior exposure to dietary nitrate, intake of readily-available carbohydrate and, particularly, the rate of intake of the plant.[7,15] Emphasising the imprecision of plant nitrate toxicity guidelines is the finding from the original investigation of nitrate-nitrite poisoning that batches of *T terrestris* containing 1.2 to 3.39% KNO_3 in dry matter were toxic to sheep.[9] The association of greater nitrate concentrations with plants growing in and around stockyards was noted originally for *T terrestris*.[9]

Aqueous humour nitrate concentrations in normal cattle are about 5 mg/L, rising to 100 to 150 mg/L in poisoned cattle.[16] Microbial action after sampling can decrease its nitrate content, so samples should be chilled for transport to a laboratory.[17] Aqueous humour may be tested for nitrate in the field using paper urine test strips[18] or nitrate test strips (Merckoquant®). The diagnosis of nitrate-nitrite poisoning in ruminants is based on rapid death, methaemoglobinaemia (chocolate-brown blood), access to plants with hazardous concentrations of nitrate, and significant concentrations of nitrate, nitrite or both in aqueous humour, serum or urine.[19,20,21] Methaemoglobin progressively reverts to haemoglobin and may only be recognisable within a few hours of death, but when present in grazing ruminants, it is characteristic of nitrate-nitrite poisoning. [19,20,21] Poisoning of ruminants by the herbicide sodium chlorate or by the silo or silage gases nitrous dioxide or nitric oxide will also produce methaemoglobinaemia,[19,20] but poisoning by these compounds is rare and access to them is readily identifiable in the field. Assaying methaemoglobin as a postmortem diagnostic tool is much less practical than measuring nitrate in aqueous humour because the latter persists much longer in carcases and necropsy samples with concentrations remaining potentially significant for 60 h after death.[16,20] Necropsy sampling of aqueous humour is preferred to serum because of a reduced likelihood of postmortem bacterial decomposition degrading the samples.[16]

Cyanogenic glycosides were reported in samples of *D radulans* (as *E aegyptiaca*) from New South Wales before 1913[22] but not subsequently.[12] Neither the plant from the first recorded case of *D radulans* toxicity in 1937[3] nor a sample of the plant from Clermont assayed in 1938 yielded cyanide[23] and White[8] states that "repeated tests of button grass for a prussic acid-yielding glucoside have always [*sic*] given negative results". Recently, *D radulans* from stockyards near Aramac (120 km east-north-east of Longreach) yielded 7.0% KNO_3 on a dry matter basis, but no cyanide (BR Burren, unpublished data, 28 March 2003). Recent experience of a case of poisoning of cattle by *Dysphania glomulifera* containing hazardous concentrations of nitrate and yielding dangerous amounts of cyanide suggests that affected animals are likely to die of cyanide poisoning before lethal methaemoglobin concentrations occur (RA McKenzie, BR Burren and JW Noble, unpublished data). Cyanide toxicity produces sudden death after access to dangerous plant species, but does not produce methaemoglobinaemia, yielding cherry-red blood instead.[19,20] Consequently, we believe that the nitrate concentrations in *D radulans* and the consistently-seen methaemoglobinaemia support nitrate-nitrite toxicity rather than cyanide toxicity in all our cases. The pink discoloration of skeletal muscles in one sheep from Case D was consistent with the presence of nitric oxide haemoglobin reported after prolonged decomposition in animals dead from nitrate-nitrite poisoning.[19]

There is anecdotal evidence that several incidents of sudden death in hungry sheep off-loaded into railway trucking yards in western Queensland were caused by access to abundant *D radulans*. This and the occurrence of these four cases highlight the imperfect nature of knowledge transfer from the scientific literature to graziers faced with critical livestock management decisions. The risk of ruminant poisoning by *D radulans* is clearly recognised in several currently available books on Queensland pasture plants written for the layman, but cases continue to occur. Improving the recognition of this serious hazard from an apparently excellent source of feed may need better targeting of conventional extension publications and seasonal reminders using electronic media. The Queensland Animal Care and Protection Act 2001 places a duty of care on those responsible for animals that may extend to preventing poisoning by known hazardous plants.

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References

1. Everist SL. *Poisonous Plants of Australia*. 2nd edn. Angus & Robertson, Sydney, 1981:313-315.
2. Sharp D, Simon BK. *AusGrass: Grasses of Australia*. CD-ROM. Australian Biological Resources Study, Canberra and Environmental Protection Agency, Brisbane. 2002.

3. Everist SL. *Dactyloctenium radulans* (button grass). *Annual Report of the Department of Agriculture and Stock, Queensland 1936-37*:113-114.
4. Ellman GL, Courtney KD, Andres VA Jr, Featherstone RM. A new and rapid colorimetric determination of acetylcholinesterase activity. *Biochem Pharmacol* 1961;7:88-95.
5. Roberts BR, Silcock RG, Scott G. *Western Grasses. A Grazier's Guide to the Grasses of South-West Queensland.* Darling Downs Institute Press, Toowoomba, 1982:50-51.
6. Tothill JC, Hacker JB. *The Grasses of Southern Queensland*. University of Queensland Press, St. Lucia, 1983:178-179.
7. O'Hara PJ, Fraser AJ. Nitrate poisoning in cattle grazing crops. *NZ Vet J* 1975;23:45-53.
8. White CT. Is button grass poisonous to sheep? *Queensl Agric J* 1937;47:502.
9. Rimington C, Quin JI. Studies on the photosensitisation of animals in South Africa. II. The presence of a lethal factor in certain members of the plant genus *Tribulus. Onderstepoort J Vet Sci Anim Indust* 1933;1:469-489.
10. Bradley WB, Eppson HF, Beath OA. Nitrate as the cause of oat hay poisoning. *J Am Vet Med Assoc* 1939;94:541-542.
11. Williams CH, Hines HJG. The toxic properties of *Salvia reflexa*. *Aust Vet J* 1940;16:14-20.
12. McBarron EJ. *The Nitrate and Cyanogenetic Status of Certain Plants in New South Wales.* Science Bulletin No 83. NSW Dept Agriculture, Sydney, 1972:24.
13. Bradley WB, Eppson HF, Beath OA. Livestock poisoning by oat hay and other plants containing nitrate. *Wyoming Agric Exp Sta Bull* 241, 1940 [cited by Crawford *et al* 1966].
14. Crawford RF, Kennedy WK, Davison KL. Factors influencing the toxicity of forages that contain nitrate when fed to cattle. *Cornell Vet* 1966;56:3-17.
15. National Research Council Committee on Nitrate Accumulation. *Accumulation of Nitrate.* National Academy of Sciences, Washington, DC, 1972.
16. Boermans HJ. Diagnosis of nitrate toxicosis in cattle using biological fluids and a rapid ion chromatographic method. *Am J Vet Res* 1990;51:491-495.
17. Carlson MP, Schneider NR, Harris B, Erickson ED, Nabity MB. Alteration of vitreous humour nitrate and nitrite content by *E coli*. In: *Proceedings of the 15th Annual Western Food Animal Disease Research Conference,* Lincoln, Nebraska, 1994 [Abstract].
18. Montgomery JF, Hum S. Field diagnosis of nitrite poisoning in cattle by testing aqueous humour samples with urine test strips. *Vet Rec* 1995;137:593-594.
19. Jones TO. Nitrate/nitrite poisoning in cattle. *In Practice* 1988;10:199-203.
20. Osweiler GD, Carson TL, Buck WB, van Gelder GA. *Clinical and Diagnostic Veterinary Toxicology*. 3rd edn. Kendall/Hunt Publishing Company, Dubuque, Iowa, 1985:460-467.
21. El Bahri L, Belguith J, Blouin A. Toxicology of nitrates and nitrites in livestock. *Compendium on Continuing Education for the Practicing Veterinarian: Food Animals* 1997;19:643-649.
22. Petrie JM. Hydrocyanic acid in plants. Part II. Occurrence in the grasses. *Proc Linn Soc NSW* 1913;38:624-638.
23. Gurney EH. Queensland Department of Agriculture and Stock, unpublished report, 11 March 1938, in Queensland Herbarium files.

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BOOK REVIEW

Crocodiles Biology, Husbandry and Diseases. *Huchzermeyer FW, CABI Publishing, Oxon, (Available from DA Information Services, Melbourne) 2003, 337 pages. Price AUD268.75. ISBN 0 85199 656 6.*

According to the author this is the first comprehensive book on diseases of crocodiles. It is promoted as a reference work on the biology, management and health of crocodiles, alligators and gharials, applicable to both farmed and captive animals. The introductory chapter describes crocodilian anatomy, physiology, biochemistry and behaviour. Subsequent chapters include the examination, both clinical and postmortem, diseases of hatchlings and eggs, transmissible and non-transmissible diseases and diseases classified according to organ systems. Fritz Huchzermeyer is the founder of the veterinary group within the Crocodile Specialist Group (CSG) chaired by Professor Harry Messel, the writer of the foreword. Originally a poultry pathologist, Dr Huchzermeyer comments that his avian and crocodile farming background enables him to understand and explain crocodilian disease from a herd health viewpoint. The book has been written for veterinarians, scientists, wildlife officials, students and crocodile farmers. The chapters are well set out and illustrated with black and white photographs. There is also a set of colour plates at the beginning of the book. Most of the photographs add to the understanding of the text, however some are poor quality, over or under-exposed and occasionally out of focus. Similarly, the hand-drawn diagrams are at times useful but mostly badly drawn and somewhat baffling. For example, a schematic drawing of a smiling crocodile with six arrows pointing in different directions is used to explain the physical, chemical and biological challenges to a captive crocodile. Another interesting illustration involves a dustpan being held over a hatchling about to be caught. Both diagrams seem superfluous to the text.

The book is easy to read and has a strong emphasis on husbandry and pathology. Crocodilian anatomy and physiology is explained in a simple and effective manner. The headings in this section follow a functional approach. Particularly appealing is the description of locomotion in young crocodilians, divided into swimming, sliding, walking, running and jumping. Another valuable feature of this book is the description of the postmortem examination, providing useful and practical information on necropsy and other laboratory techniques. However some of the information presented, especially the clinical procedures, is not of the same standard. Some sections on anaesthesia and surgery are best omitted as they are out of date clinically and ethically. For example, the section on surgical intervention describes laparotomies performed on adult crocodiles using local anaesthesia and manual restraint and another using only manual restraint. There is no accompanying comment by the author comparing these techniques with current trends in reptilian analgesia and anaesthesia and modern standards of animal welfare. Laparoscopy of 23 mature Johnston's crocodiles is also described. Animals were strapped to a board to prevent movement. The abdominal cavity was inflated with air and a blunt stylet and trocar were inserted through a ventral abdominal incision. Admittedly the author declares in the introduction that he is not a clinician, however better analysis and criticism of these techniques would have been apposite.

The work is well referenced and the enthusiasm of the author for his subject is obvious. This book would have a limited readership in Australia but would enhance the library of anyone interested in the veterinary aspects of captive crocodile management.

R Johnson

Dr Robert Johnson owns a first opinion and referral practice in reptiles and exotic pets in South Penrith, New South Wales.