CASE REPORT

# Unusual Skeletal Deformities in Calves in a Saskatchewan Beef Herd

J.P. ORR AND G.C. McKENZIE

*Department of Veterinary Pathology (Orr), and Department of Clinical Studies (McKenzie), Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N 0W0*

*Summary*

A very high incidence of posterior paresis in growing calves was casued by abnormal development of the vertebral column. The pathological changes were focal premature synostoses of vertebral growth plates with resulting reduction in size of the vertebral canal. Malacic changes were seen in sections from all areas of the spinal cord. Abnormalities in growth of the cranium and appendicular skeleton were also present. The growth plates in the cranial base were prematurely closed and the cranial cavity was shortened. Long bones were shortened and distorted. Possible causes include teratogenic plants or chemicals, mineral deficiencies, and hypervitaminosis A.

*Résumé*

**Difformités squelettiques inhabituelles, chez des veaux d'un troupeau d'animaux à boeuf de la Saskatchewan**

Plusieurs cas de parésie postérieure, chez des veaux en croissance s'avérèrent imputables à un développement anormal de la colonne vertébrale. Les lésions consistaient en des synostoses focales prématurées qui entraînaient une réduction de la lumière du canal vertébral. On décela de la malacie dans des coupes de toutes les régions de la moelle épinière. On nota aussi des anormalités dans le développement du crâne et du squelette appendiculaire. Les plaques de croissance de la base du crâne se fermaient prématurément et la cavité crânienne s'en trouvait raccourcie. Les os longs étaient aussi raccourcis et tordus. On attribua ces anomalies à des plantes ou à des substances chimiques tératogènes, à des déficiences minérales et à une hypervitaminose A.

*Introduction*

Generalized skeletal deformities are identified sporadically in one or in a few calves in any one herd, and the cause of these is usually not identified. A study of congenital defects in 476 calves over a 22 year period identified three types of systemic defect, namely dwarfism, osteopetrosis, and metaphyseal dysplasia (5).

Outbreaks of systemic skeletal deformities in calves have been attributed to teratogenic factors in plants (8) and to dietary deficiency of manganese (7).

The deformities described in this paper affected all or most of the calves born in 1979 in a herd of 18 Angus cows.

*History and Clinical Findings*

Eighteen purebred Angus cows were bred by a purebred Hereford bull in 1978. During the fall and till the end of January 1979, the cows were offered free choice barley straw and 6-8 lb (2.7-3.6 kg) of oat chop per head per day. Vitamin A injections were given in November, February and April. At the end of January the owner noticed that the cows were rubbing themselves with resulting loss of hair, and he substituted hay for the barley straw.

Calving extended from mid February till early April. A few calves were born a few days early and eight calves were stillborn or died within 24 hours of birth. Calves born weak could not stand up and died in spite of force feeding of colostrum. None of these calves were necropsied. The remaining ten calves appeared normal at birth but by the time they were one month old, they were unthrifty and "poorly shaped" and they had difficulty in walking. By two to three months they preferred recumbency and often could not rise without assistance. During this period the calves' appetites were normal. All of the calves had been given an injection of vitamin A at birth and a few neonates were treated with tetracycline boluses when they had diarrhea.

Physical examination revealed abnormalities of the skeletal and nervous systems. The calves had a dwarf-like appearance. They were smaller than normal, had a large, wide head with a snub-nose and short limbs which showed varying degrees of bow-leggedness. The carpal and tarsal joints appeared enlarged and were abnormal on palpation. When standing still, these calves shifted the weight on their hind limbs from side to side. The leg which became nonweight bearing was extended, and held out behind as is seen in spastic paresis. The tail heads of all calves were elevated. Occasionally the calves would "kneel" on their carpi as if to transfer weight from the hind quarters. The calves preferred to lie down or to remain in one place. When they did run or walk, their gait was stilted. Hindlimb ataxia and weakness were evident and knuckling of the fetlocks was noted occasionally.

Radiographic examination of several limbs revealed epiphyseal and metaphyseal malformations. Epiphyses appeared malformed or hypoplastic. Metaphyseal regions were flared and physes were highly irregular, cone shaped and difficult to distinguish. The spine of one animal was examined radiographically and showed similar changes to a milder degree.

Our clinical differential diagnoses

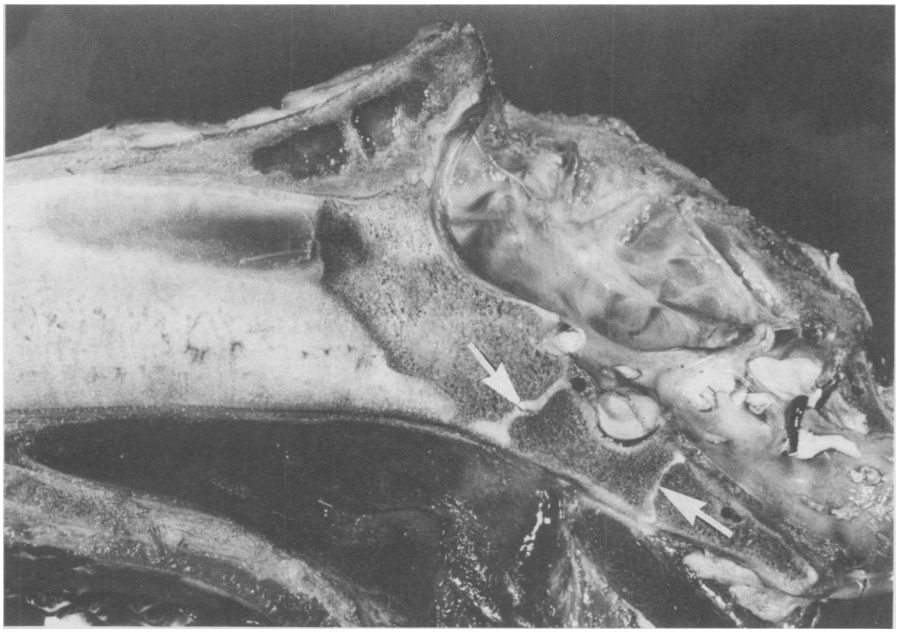

FIGURE 1. Sagittal section of cranium of a normal six month old calf for comparison with Figure 2. Growth plates are visible (arrows).

were hypovitaminosis D, dwarfism, hypervitaminosis A or toxicosis of unknown etiology.

Euthanasia and necropsy were performed on six calves, aged three to five months. Similar pathological changes were found in all six calves.

*Postmortem Findings*

*Gross Findings* — General body condition was good. The forelimbs and/or hindlimbs appeared shortened and distorted. Decubitus ulcers and subcutaneous abscesses were present over carpal joints and there was excess fluid and fibrin within these joints.

Long bones of the forelimbs were shortened and distorted. The shaft of the humerus and its cortical bone were thickened, and the proximal articular surface was flattened and angulated, with corresponding change in shape of the articular surface of the scapula.

The cranium was shortened and wider than normal, sometimes with a prominent midline ridge along the frontal suture. There were irregularities of the internal bony surfaces of the cranial cavity with sharp bony projections from the pons area of the floor. On midline sections, the basisphenoid bones were shortened, with shortening of the pituitary fossa. There was premature closure of the growth plates in the cranial base (Figures 1 and 2). The brain appeared longitudinally compressed due to abnormal shape of the cranial cavity. A mild degree of superior brachygnathia was noted.

Midline sagittal section of vertebrae revealed focal closures of the vertebral growth plates of the lumbar and thoracic regions (Figures 3 and 4). Affected vertebral bodies were abnormally shaped — "butterfly" shaped on sections. The vertebral canal was deformed and stenosed (Figure 3). Intervertebral discs were abnormally shaped having a central projection into the vertebral epiphyses on both sides or only on the caudal aspect (Figure 3). Some discs had central cavitation and degenerative change, and cavitations were sometimes present in the spongy bone of the vertebral bodies.

One calf had an enlarged heart with rounded apex.

*Histopathological Findings* — There were focal closures of vertebral growth plates and the gaps were bridged by trabecular bone. Cavitation in some intervertebral discs was accompanied by degeneration and necrosis. Large thick plugs of cartilage were seen in the physis at sites of premature closures. Trabeculae on the diaphyseal side of the physis were thicker and fewer than normal and alignment was poor. Organizing hemorrhages were present along the margins of the discs.

Numerous dilated axon sheaths were noted in white matter of the spinal cord. These were often empty or sometimes contained swollen axons, eosinophilic debris or phagocytic cells. Lateral and ventral columns were most severely affected. All regions of the cord were affected, but often the lesions were locally more severe.

Contracted pyknotic hyperchromatic neurones were more numerous

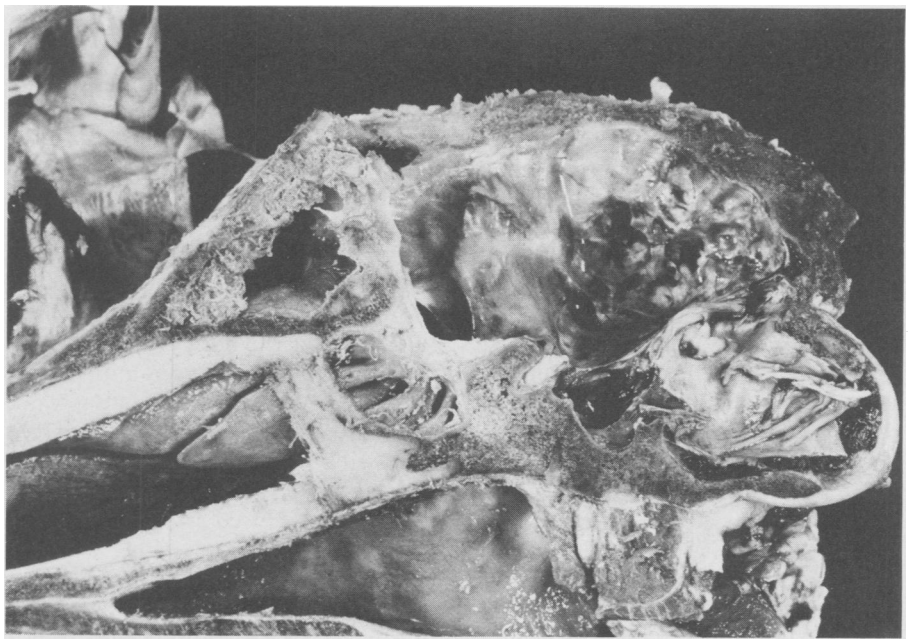

FIGURE 2. Sagittal section of cranium of an affected calf. Growth plates are absent from the cranial base which is also markedly shortened.

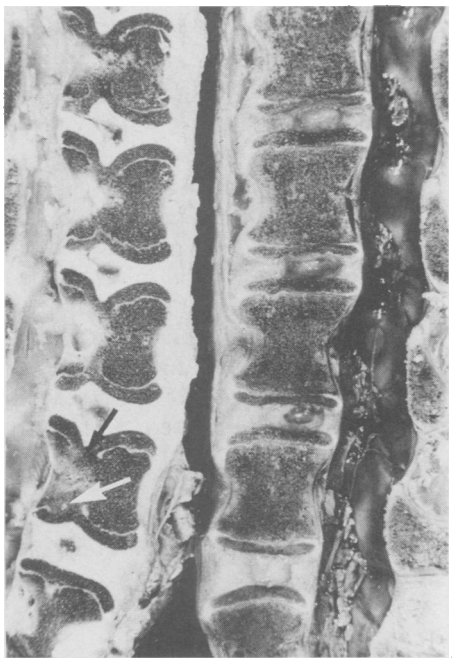

FIGURE 3. Sagittal sections of lumbar vertebrae of an affected (left) and a normal (right) calf of the same age. Focal synostoses of the epiphyseal growth plates (arrows), abnormal shape of vertebral bodies and intervertebral discs, and reduced diameter of the vertebral canal are evident in the affected calf.

than usual in the brain. Diffuse increase in glial nuclei was also apparent.

*Ancillary Laboratory Studies*

Hemogram and routine blood chemistry were normal.

The following analyses were performed:

Lead
- Blood = none detected
- Liver = 0.36 ppm
- Kidney = 0.41 ppm

Copper
- Blood = 0.83 to 0.97 ppm
- Liver = 8.70 ppm
- Kidney = 1.18 ppm

Zinc
- Serum = 0.93 to 0.95 ppm

Vitamin A
- Serum = 26 $\mu$g/dL

Organophosphates
- None detected

Organochlorines
- Kidney fat = 0.032 ppm H.C.B.
  = 0.038 ppm $\alpha$ B.H.C.
  = 0.007 ppm P.C.B.

All analyses were done on a wet weight basis. The lead and organochlorine levels detected are interpreted as normal background levels. Blood copper is within the normal range but the levels in liver and kidney tissues are low. The remaining parameters are normal.

*Notes on Development and Abnormalities of the Vertebrae*

The vertebral bodies and intervertebral discs are of mesodermal origin, and the nuclei of the discs are remnants of the notochord (2). During the early stages of ossification, the vertebral body shows a dorsal and a ventral ossification centre — these soon join to form a single ossification centre (2). The cranial and caudal end plates develop as secondary centres of ossification at a later stage. Each arch pedicle develops from a primary centre of ossification (1) (Figure 5).

Growth in size of the vertebral canal is mainly determined by the continued activity of the growth plates between the vertebral bodies and their arches, before and after birth (12).

Studies in prenatal and postnatal calves up to one month old have demonstrated growth plates between the vertebral body and each arch pedicle, and also between the vertebral body and its cranial and caudal epiphyses (9).

More extended anatomical studies in sheep (1) have shown that the vertebral bodies and arches fuse in the thoracic and lumbar regions at three to six months, and that the epiphyses of the end plates begin to fuse to the vertebral bodies at about 18 months of age.

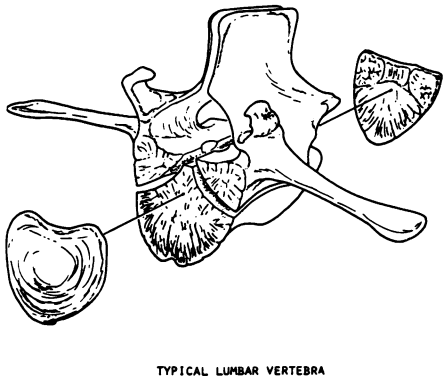


FIGURE 5. Drawing of a "typical" ruminant lumbar vertebra, exploded view.

Severe malformations of the spine are determined very early in prenatal life. Less severe anomalies of vertebrae and discs can be due to influences acting in later fetal stages (13). If there is secondary spinal cord disease, it is usually a result of compression (2). A wide variety of vertebral deformities have been described in humans. Some of these result from arrested regression of the notochord (12, 13), e.g. localized expansion of the discs in the area of the nucleus pulposus resulting in funnel shaped projections into the adjacent vertebral bodies. Cleft vertebrae in the sagittal plane are attributed to persistence of, or sagittal cleavage of, the

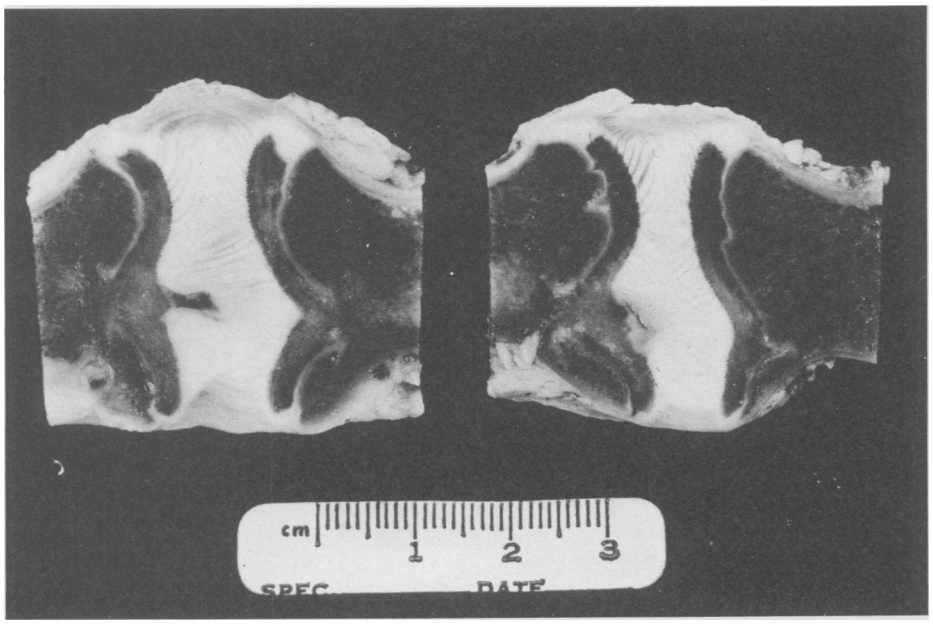


FIGURE 4. Fixed specimens from the vertebrae of an affected calf showing detail of the growth plate and disc deformities.

notochord; resulting vertebrae have a butterfly appearance on dorso-ventral radiographs.

*Discussion*

A consistent pattern of skeletal deformities involving the long bones, vertebrae, occipital bones and to a lesser extent the flat bones, was identified in the six calves necropsied. These deformities were characterized by focal premature closures of the physes, and resulted in restricted growth in size of the cranial cavity and spinal canal. Compression of the brain and spinal cord resulted in a paretic syndrome. Table I lists some known causes of developmental skeletal anomalies. Hereditary disease is not possible in this case for two reasons. First, the same bull sired a normal calf crop from the same dams in 1977-78. Secondly, the incidence of the disorder (10/18 or 56% if the neonatal deaths are counted as unaffected calves) is higher than would be expected even for a dominant factor.

Clinically the calves had some of the characteristics of dwarfs (5) and this was borne out at postmortem examination. The cranial deformities are very similar to those seen in dwarf cattle of the short-headed type (7). Also, complications affecting human achondroplastic dwarfs include spinal cord compression at the foramen magnum and in the lumbar region, and compression of emerging spinal nerve roots. These complications may cause paraparesis (13). But the vertebral abnormalities seen in these calves did not conform to those described for the achondroplastic dwarf, since in these calves premature closure of the growth plates was focal, leading to a different type of vertebral deformity (5).

No evidence of exposure to teratogenic plants (8) has been found, although we cannot entirely rule out that possibility. The cattle were possibly exposed to carbamates[1] as crop treatments. They were treated with fenthion[2] as a pour on insecticide. All of these are widely used chemicals and little is known of their potential as teratogens in ruminants.

Evidence of a serious mineral deficiency was not found, but studies in that area were not exhaustive.

Excessive intake of vitamin A has produced bone lesions in calves and pigs experimentally, characterized essentially by interference with endochondral and intramembranous ossification (6, 11, 14, 15). Similar naturally occurring bone lesions have been recorded in children and pigs (4, 10). The most significant changes recorded include dissolution and premature closures of growth plates of long bones, with cessation of longitudinal growth, and also osteoporosis. The vertebral column has received little or no attention in these studies. The deformities seen in the calves examined in this outbreak could have been caused by excessive vitamin A intake. The doses of vitamin A administered to the cattle are unknown. The cows received injections of vitamin A on three occasions during and immediately after pregnancy, and the calves each received one injection at birth.

A review of the teratogenic effects of excessive vitamin A in various species (3) describes abnormalities of many body systems including the skeleton. The effects depend on species, dose, and time of exposure. The resulting abnormalities seemed to be due to effects of vitamin A on mesoderm. Several research workers recorded growth disturbances manifested in postnatal life, although gross lesions were not identified at the time of birth. There is insufficient data to indicate the threshold of vitamin A for teratogenic effects. In most of the research, very high doses were used.

In conclusion, we feel that this disease problem was caused by a teratogen which we have failed to identify conclusively although hypervitaminosis A remains a possibility.

TABLE I
SOME KNOWN CAUSES OF DEVELOPMENTAL SKELETAL ANOMALIES

| | | |
|---|---|---|
| Hereditary | Dwarfism | Primordial |
| | | Chondrodystrophic |
| | Other e.g. | Osteopetrosis |
| | | Osteogenesis Imperfecta |
| Mineral Deficiency | Copper | |
| | Manganese | |
| Hypovitaminosis and Hypervitaminosis | | |
| | Vit A | |
| | Vit D | |
| Chemical Teratogens | | |
| Plant Teratogens | Locoweed | |
| | Lupin | |
| | Lathyrus | |
| Infectious Agents | Viruses | |
| | Bacteria | |

*Acknowledgments*

Our thanks to the Department of Physiology, Western College of Veterinary Medicine, Saskatoon and to the Alberta Provincial Veterinary Laboratory, Edmonton for performing the toxicological analyses. Also to Miss Leslie C. Lee for typing the manuscript.

*References*

1. AL-SHAIKHLY A.K.J. Growth and development of the vertebral column in the sheep. Ph.D. Thesis. Faculty of Medicine, Bristol. 1971.
2. BAILEY C.S. An embryological approach to the clinical significance of congenital vertebral and spinal cord abnormalities. J. Am. anim. hosp. Ass. 11:426-434. 1975.
3. GEELEN J.A.G. Hypervitaminosis A induced teratogenesis. C.R.C. Critical Reviews in Toxicology. pp. 351-375. November 1979.
4. DOBSON K.J. Osteodystrophy associated with hypervitaminosis A in growing pigs. Aust. vet. J. 45: 570-573. 1969.
5. GREENE H.J., H.W. LEIPOLD and K. HUSTON. Bovine congenital skeletal defects. Zentbl. VetMed. A 21: 789-796. 1974.
6. GREY R.M., S.W. NIELSEN, J.E. ROUSSEAU, M.C. CALHOUN and H.D. EATON. Pathology of skull, radius and rib in hypervitaminosis A of young calves. Path. Vet. 2: 446-467. 1965.
7. JUBB K.V.F. and P.C. KENNEDY. Pathology of Domestic Animals. 2nd Ed. New York: Academic Press. 1970.
8. KEELER R.F. Effect of natural teratogens in poisonous plants on fetal development in domestic animals. Adv. exp. Med. Biol. 27:107-125. 1972.

[1]Avadex — Monsanto Canada Ltd., Rexdale, Ontario.
[2]Spotton Cattle Insecticide — Cutter Animal Health, Bayvet Division Cutter Laboratories Inc., Mississauga, Ontario.

9. LINDSAY F.E.F. Observations on the loci of ossification in the prenatal and neonatal bovine skeleton III. The vertebral column. Br. vet. J. 128:121-128. 1972.

10. PEASE C.N. Focal retardation and arrestment of growth of bones due to vitamin A intoxication. J. Am. Med. Ass. 182:980-985. 1962.

11. PRYOR W.J., A.A. SEAWRIGHT and P.J. MCCOSKER. Hypervitaminosis A in the pig. Aust. vet. J. 45:563-569. 1969.

12. SCHMORL G. and H. JUNGHANNS. The Human Spine in Health and Disease. Second American Ed. Translated and edited by E.F. Besemann. New York and London: Grune and Stratton. 1971.

13. WARKANY J. Congenital malformations — notes and comments. Part XLV Malformations of the Axial and Appendicular Skeleton. Chicago: Year Book Medical Publishers Inc. 1971.

14. WOLKE R.E., H.D. EATON, S.W. NIELSEN and C.F. HEMBOLDT. Qualitative and quantitative osteoblastic activity in chronic porcine hypervitaminosis A. J. Path. 98:677-686. 1969.

15. WOLKE R.E., S.W. NIELSEN and J.E. ROUSSEAU. Bone lesions of hypervitaminosis A in the pig. Am. J. vet. Res. 29:1009-1024. 1968.

## BOOK REVIEW

*Canine Medicine, Fourth Edition. Volumes I and II.* Edited by E.J. Catcott. Published by American Veterinary Publications, Santa Barbara, California. 1979. 2038 pages. Price $92.50.

It has been ten years since the publication of the last edition of Canine Medicine, and as editor Catcott has written in the preface, the increase in size from one volume to two is remarkable. This edition is essentially a new book; a comprehensive text written by 59 authors, the vast majority of whom are working at universities in the USA. The quality of chapters varies as one would expect, but it is this reviewer's opinion that Canine Medicine, 4th Edition, is the best comprehensive text on the subject available.

The infectious and contagious diseases are described in the first four chapters and as in previous editions, canine distemper, infectious canine hepatitis and rabies are thoroughly described. Canine parvovirus disease was born after the book was printed. Mast cell sarcoma is discussed as a virus disease. The diseases caused by bacteria and rickettsia are described briefly and accurately, but Salmonellosis should receive more attention. The systemic mycotic diseases deserve a little more detailed attention in a new book. The internal metazoal and protozoal diseases are presented in detail with good photographs including heartworm disease.

The chapter on diseases due to chemical and physical agents is detailed and includes snakebite, seventeen pages on burns, a good section on electric shock as well as exposure, radiation and teratology.

The chapter on nutrition and disease is concerned with nutrition for health and the use of nutrition in the management of disease as well as nutritional dificiency. Chapter seven is a useful discussion of atopy, anaphylaxis, urticaria, contact dermatitis and food allergy. Food allergy is also described in the chapter on nutrition.

The digestive system is contained in 149 pages with many illustrations. It begins with an extensive description of dental diseases and diseases of the mouth. The diseases of the esophagus and stomach are well done and there is a review of gastric physiology and pathophysiology. The section on the small intestine contains good examples and the section on the large intestine is very thorough.

The section on the liver contains three useful pages of physiology, but the disease section is disappointing in the range and scope of the discussion of diseases. The section on the pancreas is a detailed and accurate description of signs and diagnosis, but the description of treatment is a general one and not categorized as to the severity of the disease. The advocacy of the injection of solutions of antibiotic into the pancreas lacks evidence of clinical science.

The peritoneum is discussed in Chapter 8 and the general description of peritonitis is acceptable, but the etiological agents ( example, nocardia species) are not mentioned.

Chapter 9, the urinary system begins with a perspective of urinary tract disease, including a definition of terms, followed by discussions of urinalysis, azotemia and the evaluation of the system, including of use of radiology, The chapter is more than adequate and is of practical use as well as a reference work.

The chapter on the genital system covers the topic well. The musculoskeletal system (Chapter II) is 145 pages and contains an excellent variety and number of photographs of various disease conditions, including some of the deficiency states described in an earlier chapter. It is a very thorough treatment of musculoskeletal disease.

Volume two begins with the nervous system, a 133-page chapter containing many line drawings and tables and an extensive discussion of spinal diseases as well as a competent coverage of all CNS problems, including electroencephalography and electromyology. Epilepsy receives extensive description, especially the author's methods of treatment.

The cardiovascular system is well organized and well illustrated in 156 pages. Radiology, physical examination, electrocardiography and cardiovascular pathophysiology are included.

The next three chapters, "Blood Cells in Disease", "The Respiratory System" and "The Endocrine System" are all excellent descriptions of the subjects and contain many illustrations.

The final three chapters, "The Skin", "The Eye", and "The Ear" are all very useful chapters. There are colour plates of skin and eye diseases and the diseases of the ear are illustrated with black and white photographs.

All in all, the fourth edition will be a little overwhelming to the student being asked to remember everything about dogs as well as cats, horses, cattle, sheep and goats, but it does demonstrate the advances made in veterinary medicine for the dog, and it is a very valuable collection of information for students of all ages. *J.H. Reed.*