Stress and Postcapture Myopathy in Red Deer

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Abstract

The neuroanatomy and physiology of stress and shock are briefly considered. Clinical, biochemical, gross anatomical, and histological changes of postcapture myopathy of red deer (*Cervus elaphus*) in New Zealand are discussed, and these changes are compared with the clinical, biochemical, and histological changes associated with postcapture myopathy of wild animals and the porcine stress syndrome. The physiological responses to the stressors of postcapture myopathy in deer and other wild animals and the porcine stress syndrome are considered to fall within the changes described by Selye for the stress condition.

Both stress imposed by capture and stressors other than capture (e.g. cold, low nutrition) can alter the responses of the thymico-lymphatic system in red deer leaving them susceptible to disease.

Keywords: Cervus elaphus, *stress, postcapture myopathy, porcine stress syndrome*

Introduction

Rather than giving an overview of stress and shock in animals, this paper reviews these conditions particularly as they apply to red deer (*Cervus elaphus*) in New Zealand.

In 1936 Hans Selye published in *Nature* a paper entitled "A syndrome produced by Diverse Nocuous Agents"; the syndrome, which later became known as the general adaptation syndrome, was divided into 3 parts (Selye 1936, 1946):

(i) general alarm reaction,
(ii) phase of resistance (phase of adaptation),
(iii) phase of exhaustion.

Selye had formulated the description of this syndrome from his experiments with mice in which he used physical and pharmacological insults; the essential feature was that the response of the animals to the various insults was always similar. The general alarm reaction occurred in Selye's experiments 6 – 48 hours after the initial insult. There was a rapid decrease in the size of the thymus, spleen, lymph nodes, and liver, disappearance of adipose tissue, oedema, loss of muscular tone, drop in body temperature, formation of acute erosions in the digestive tract (particularly in the stomach, small intestine, and appendix), loss of cortical lipoids and chromaffin substance, and increased lachrymation and salivation.

The second stage began after about 48 hours, when the adrenals enlarged greatly and regained their lipoid granules, oedema began to disappear, the thyroid showed a tendency towards hyperplasia, general body growth ceased, the gonads became atrophic, and milk secretion stopped, i.e. there was reduction in the production of gonadotropic hormones and prolactin, and an increase of the thyrotropic and adrenotropic hormones. As the insult continued the various organs became practically normal. In the third stage the animals lost their resistance and died with symptoms similar to those of the first stage.

The terms "shock" and "stress" are now commonly used, though often misused. A definition of each should clarify the meanings used in this paper:

- *Stress* may be considered as a homeostatic or physiological mechanism in response to a stimulus or succession of stimuli; this enables the organism to best cope with or adapt to the changed environment.
- *Shock* is a clinical manifestation of acute peripheral vascular collapse. A widespread dilatation of the blood vessels institutes a series of circulatory changes resulting in hypotension, weak pulse, tachycardia, pallor, and diminished urinary output.

Shock and stress are difficult to separate as both are initiated from the same area of the brain, the hypothalamus.

Shock

The hypothalamus is closely involved in the control of the autonomic nervous system. It receives input

from various parts of the brain subserving autonomic functions and is concerned with regulation of body temperature, endocrine control of the metabolic rate, and control of water balance via neuro-endocrine reflexes. The signs of increased autonomic activity associated with stress appear to be mainly due to activation of the sympathetic (adrenergic) division resulting in increased production of catecholamines, namely adrenaline by the adrenal medulla and noradrenaline by the sympathetic nerve terminals. This results in an increased rate of heart beat, increased contractility of cardiac muscle with increased cardiac output and arterial pulse pressure, constriction of arterioles in some regions, e.g. skin and kidneys, and dilatation in others such as muscle, the general effect being a shunting of blood to the muscle beds.

As a result of these responses the animal is better equipped to handle an emergency situation. However, if the stimulus is too great and there is an excessive secretion of catecholamines, a haemorrhagic enteropathy and death may result, a finding which has been reported in red deer (McAllum 1981). In the same way, dogs injected with catecholamines suffered ulceration of the gastrointestinal tract and haemorrhagic enteropathy (Penner and Bernheim 1939), while a similar condition (neurogenic shock) occurs in man and is believed to be caused by the same mechanism (Robbins and Cotran 1979).

Shock may be caused by other mechanisms including hypovolaemic shock, the endotoxic forms of septic shock as found in bacterial and other diseases, and anaphylactic shock.

Stress

Stimulation of the pituitary arises from the activation of particular groups of neurosecretory cells in the hypothalamus, which secrete releasing or inhibiting hormones. The hormones diffuse into the capillary loops and are carried in the portal blood to the cells of the adenohypophysis, where they stimulate or inhibit production of the appropriate adenohypophyseal hormone. Although many hormones are secreted by the anterior pituitary, adrenocorticotrophin (ACTH) acting upon the cortex of the adrenal gland is the most important in the manifestation of stress.

Stress has been studied in a number of animals since the initial investigations conducted by Selye: e.g. studies in fish (Wedemayer and Yasutake 1977), pigs (Topel *et al* 1971, 1975), and deer (McDonald 1979) have related stress to the changes in cortisol levels. Two particular stress conditions in animals have been the subject of research in recent years, namely postcapture myopathy (PCM) in wild animals and the porcine stress syndrome (PSS) in domestic pigs. The important features of these conditions are described here preliminary to the description of a study of a myopathic condition seen in red deer after capture from the wild.

Stress in wild animals

Studies of capture-associated stress in wild animals have provided important insight into the particular stress condition associated with capture in red deer. For example, in a study of feral horses, plasma corticosteroid levels were greatly elevated just after capture; 2 horses which had very high levels also showed clinical signs of stress and shock (Kirkpatrick *et al* 1979).

The postcapture myopathy syndrome as it affects wild animals in South Africa has been described by Basson and Hofmeyer (1973) as being characterised by "ataxia, paresis or paralysis with the excretion of brown urine. Associated with these clinical changes were asymmetrical muscular and myocardial lesions".

Harthoorn *et al* (1974), immobilising zebras manually without drugs, found that when the animals were given extensive exercise over short distances and the clinical parameters measured shortly after capture, respiratory and cardiac rates and rectal temperatures were elevated. Despite a period of complete rest 1 animal's heart rate rose to 360 beats/min and the respiratory rate remained rapid (56/min), and another animal's temperature rose to 43°C. The zebras were disinclined to move when placed on their feet and 30 minutes after capture were almost incapable of rising. Death occurred from 30 minutes to 12 hours after capture.

Investigation of the blood biochemistry of these zebras revealed that the animals chased to exhaustion before darting had irreversible biochemical changes (Hofmeyer *et al* 1973). Analysis of the plasma included determinations of plasma glucose, lactate, creatinine kinase, and plasma osmolality. Not only were these levels greatly elevated, but severe muscle lesions were described from an animal from the exercised group that died. These workers concluded that the changes were due to stress associated with the chase and capture.

Stress susceptibility in pigs

The porcine stress syndrome (PSS) is a stress condition seen in domestic pigs and has been described as "an inability of susceptible swine to endure the usual environmental stressors. associated with normal management" (Williams *et al* 1975). Such stressors include handling, castration, vaccination, movement, high ambient

temperature, breeding activity, etc. These workers suggest that it is a condition identical to fulminant hyperthermia in humans, swine, cats, dogs, and horses.

PSS is considered to be due to a genetic error of metabolism inherited as a dominant gene or genes in which there appears to be a deficiency of monoamine oxidase, resulting in an excess of noradrenaline activity leading to the observed pathophysiological responses. Pretreatment of susceptible animals with α-methyl DOPA or reserpine has been shown to prevent the development of PSS (Williams *et al* 1975).

Topel *et al* (1975) have described the clinical picture of PSS thus: "Early signs of stress adaptation difficulty in the pig are muscle and tail tremors. Further stress can result in marked dyspnoea, irregular breathing, alternating blanched and reddened areas of the skin, rapid increase in body temperature, cyanosis and development of an extreme acidosis condition. The next stage results in total collapse, marked muscle rigidity, hyperthermia and death".

Serum enzyme levels are difficult to study in stress-susceptible pigs and similarly the adrenal corticoid levels give variable results. However, Marple and Casseus (1973) measured the cortisol turnover rate and found it to be 3 times faster in stress-susceptible than in normal pigs and that stress-susceptible pigs also cleared plasma of cortisol approximately 5 times faster. Changes were found in the histology of the zona reticularis of stress-susceptible pigs; they often had large mitochondria with elaborate cristae and an adjacent smooth-surfaced endoplasmic reticulum. These animals also had high levels of adrenal corticoids in the blood prior to slaughter (Ball *et al* 1972; Cassens *et al* 1965).

Other changes reported include higher ACTH levels in stress-susceptible pigs (Marple *et al* 1972), very rapid development of rigor mortis (15 – 30 minutes after death) caused by denaturation of muscle protein due to high temperature, high lactic acid, and low pH in the muscle (Nelson *et al* 1974), and fibrosis and acute degeneration in the muscles (semitendinosus, semimembranosus) of pigs dying from PSS (Invermay Animal Health Laboratory, unpubl.). In the latter condition, a vitamin E/selenium deficiency was excluded as the cause of the changes.

Capture Stress in Deer

The capture of wild red deer is mainly by helicopter operations using either a tranquilliser gun or a net. Netting is quicker than the pharmacological method, but the stress imposed by the 2 methods may be similar. Other capture methods include tranquillising by hunters on foot, and bush trapping. In the capture operation, the alarm reaction is probably first elicited when deer hear the sound of the approaching helicopter or, in the case of deer caught in bush traps, when men approach the pen.

This study

Data for this study were collected from deer tranquillised from helicopters in the southern back country of the South Island of New Zealand (McAllum 1978). The samples collected from captured deer were divided into 2 groups, namely those from animals which lived, known as "captured deer", and those from deer which died, defined as "myopathic deer". Affected deer varied in age and size. The myopathic syndrome was found to be the same, irrespective of whether the animals were captured with or without the use of drugs, as has been shown by later studies (McAllum unpubl.).

Clinical signs

Parameters were first recorded within 30 minutes of capture and thereafter at intervals. The respiratory rate ranged from 36 to 160/min within 30 minutes of capture for both the captured and myopathic groups, returning to normal levels within 24 hours. The heart rate was also higher in the myopathic group particularly at 30 minutes. In general, an increase in heart and respiration rates, its magnitude depending on the work intensity, may be expected in studies of the response of animals to exercise. In horses a linear increase in heart rate to 240 beats/min was related to greater levels of work (Lindholm and Saltin 1974). Similarly, heart rates of 250 beats/min were recorded in blesbok when run over distances of about 2 km (Harthoorn and van der Walt 1974).

In the blesbok study those that ran the greatest distances also had the highest rectal temperatures. In this respect most of the red deer studied were considered to have run over short distances and generally heart rates of 80 – 90 beats/min were recorded within 10 minutes of capture. It is, however, interesting to note that the female with the highest heart rate (380/min) had run a long distance of 3 – 4 km and was in an excited state from the time she was first seen. This hind, which was very distressed and warm to the touch, suggesting a condition similar to malignant hyperthermia, died within 30 minutes of capture; the rectal temperature reached the limit of the thermometer (43 °C). Although the rectal temperatures of both groups of deer were high

immediately after capture, the values tended to return rapidly to normal.

Although some of the animals, both captured and myopathic, were alert in appearance after capture, many were very exhausted. A "wry" neck often developed within 45 – 60 minutes after capture. Some animals showed either knuckling over of the fore or hind legs, dragging a leg, or reluctance to move within hours. Paresis developed within 24 hours and progressed to a general paralysis. Even when assisted to stand, lack of control of the legs was apparent. In acute cases the deer became recumbent after capture and died within a short time.

Deer often urinated soon after capture, with the urine being a very dark, almost black, coffee colour. Such dark brown urine has also been reported to occur in PCM of African wild animals (Basson and Hofmeyer 1973). Microscopic examination of the deer urine revealed the presence of red blood cells, haemoglobin, and other debris, but the presence of myoglobin was not confirmed.

Blood from captured and myopathic animals was frequently found to be very dark. After separation the serum was pink indicating that either haemolysis of the red blood cells had occurred or that myoglobin was present in the blood.

Haematological and biochemical parameters

Data for the captured and myopathic groups are presented in Table 1. Although very few haematological readings were taken, the myopathic animals had high haemoglobin and PCV levels which were still apparent at 24 hours post capture, probably indicative of dehydration. The total white blood cell count was very low in the myopathic group 30 minutes after capture but had returned to within the normal range by 24 hours. In contrast, the white cell count of the captured group was within or above the normal range at both times.

Biochemical parameters have been compared with normal values and also compared between the 2 groups of deer. The most marked effects are evident in lactate, SGOT (serum glutamicoxaloacetic transaminase), urea, phosphate, glucose, and potassium.

Lactate levels were high by 30 minutes after capture, with the level at that time being about 4 times normal in the captured group and about 7 times normal in the myopathic group. Levels were still elevated the day after capture. The increase in lactate levels indicates that a high level of anaerobic glycolysis occurred and still persisted 1 day after capture. Although the number of animals was small, it appears that levels in myopathic deer were higher than those in captured deer.

High levels of lactate after exertion have also been recorded in man (Osnes and Hermansen 1972), in pigs (Jorgensen and Hyldgaard-Jensen 1975), and in horses (Lindholm and Saltin 1974). The time for lactate levels to return to normal may relate to the fitness of the animal. Basson and Hofmeyer (1973) and Harthoorn *et al* (1974) concluded that fear and shock contributed to the acidotic state, while the severity of the acidosis may

Table 1: Haematological and biochemical parameter mean values from venous blood for "captured" and "myopathic" deer compared with normal values

	Captured		Myopathic		Normal
Time post capture (h):	0.5	36	0.5	24	±s.d.[1]
No.	3	2	4	5	
Haemoglobin	–	14.5	–	19.4	
PCV	–	42	–	54	
White cells ($\times 10^9$/l)	4.3	9.7	0.5	3.9	3.5 ±1.5
Blood pH	7.29	7.51	7.17	7.37	7.27 ± 0.77
pCO_2	30.0	38.6	81.4	51.1	
Bicarbonate	–	27.8	–	34.5	
Total protein	65.2	61.0	62.4	71.5	
SGOT (SFU)	233	83	1424	7352	
Blood urea (mmol/l)	6.2	12.7	5.6	15.3	
Phosphate (mmol/l)	0.8	1.3	0.8	2.8	
Glucose (mmol/l)	8.6	6.3	7.8	4.7	9.4 ±1.6
Sodium (mmol/l)	180	152	178	163	
Potassium (mmol/l)	9.6	4.1	19.1	12.1	
Calcium (mmol/l)	2.2	2.0	2.4	1.8	
Magnesium (mmol/l)	1.0	0.7	1.1	1.0	
Lactate (mmol/l)	7.9	4.5	13.6	3.8	1.9 ±1.0

[1] Normal range data

be controlled by the fitness of the animal. Jorgensen and Hyldgaard-Jensen (1975) showed a difference between unfit and fit pigs, the latter being able to cope more than adequately with the increased lactic acid in the blood. From this it may be suggested that wild animals are not necessarily very fit and that the most intense exercise would be of short duration when escaping from a predator. Long arduous runs would be uncommon. Thus the high levels of lactate on the day after capture may indicate that the deer were unfit or that there was a slow release of lactate from the muscles.

Lactate influences the hydrogen ion concentration of the blood (Henry *et al* 1974) so that pH levels normally fall when lactate levels rise. However, the mean pH values obtained in this study did not conform to this pattern of lactate levels. This lack of correlation between lactic acid rise and pH decline may have been due to the compensatory effects of respiration or to the mean values used representing small numbers of animals which have run different distances. Some individual animals had an alkalosis initially and this on a group basis may have obscured an initial acidosis in those animals forced to run rapidly over short distances without prior warning. It may also be that the alkalosis arose in the counter-shock state.

The partial pressure of carbon dioxide (pCO_2) in the blood was higher for the myopathic group than the captured group, and the bicarbonate levels tended to be higher in the myopathic group. There appears to be no such correlation between the rise in lactate levels and the changes in pCO_2 as Milne (1974) found in horses. However, this may have been due in part to the temperature of the animal's body, particularly after a run.

The SGOT level, an index of muscle damage, rose to very high levels in the myopathic group compared with the captured group. In the former the levels were very high at 24 hours suggesting an ongoing necrosis of muscle, whereas levels were near normal after 36 hours in the captured group of deer (data not presented).

Potassium levels were about twice the normal value in the captured deer at 30 – 60 minutes after capture but had fallen to within the normal range by the day after capture. In the myopathic group a potassium level of up to 28 mmol/l or nearly 4 times normal was recorded in the first half hour after capture. Such high levels probably contributed to the sudden deaths. Death would result from the extracellular fluid potassium rising and water passively entering the cells causing cell swelling and degeneration. The peak in potassium occurred before the peak in SGOT possibly because potassium is a smaller, more mobile ion and therefore more easily released from damaged cells. As well there is a mitochondrial GOT enzyme fraction which would not be released until all necrosis had occurred. On the day following capture, potassium levels were lower probably because of the effect of aldosterone control. However, there are a number of other possible causes of elevated serum potassium levels including an efflux of K+ ions from within the cell in exchange for H+ ions during a metabolic acidosis, a diffusion of K+ ions from the intracellular space when muscle and liver glycogen is reduced, and release of K+ from red blood cells as a result of intravascular haemolysis (Bergstrom *et al* 1971; Gilligan *et al* 1943; Kjellmer 1965).

Blood urea levels in both groups were intially below those of normal animals and of normal Fentaz (Ethnor Pty Ltd) treated animals. However, the increased levels in both captured and myopathic groups indicated that tubular damage was occurring, particularly in the myopathic group. Both myoglobin and haemoglobin are known to cause tubular epithelial cell degeneration (Heptinstall 1966). The faint bluish tinge with the Perl's Prussian blue stain in the histological sections of the kidney may indicate this excretion of the myoglobin. It is possible that the degeneration of the kidneys in this manner could have led to kidney failure, uraemia, and eventual death.

Calcium levels of both groups were perhaps slightly higher than normal. This may have been due to a shift by the calcium from the muscle cells to the serum as a result of the acidosis (Rose *et al* 1970).

Serum inorganic phosphate levels were initially depressed compared with normal animals; the depression was greater than could be attributed to the effects of Fentaz. However, it appears that levels recovered within 1 – 1.5 hours. Adrenalin and muscular work could have been involved in the depression in serum phosphate (Codazza *et al* 1974).

Histopathology

Representative samples from each myopathic animal were examined, and descriptions, except for skeletal muscle, are presented in the Appendix to this paper.

For examination of skeletal muscle, bilaterally paired samples were taken from Muscularis cleidobrachialis, the long and short heads of M. triceps brachii, M. longissimus lumborum, and M. semimembranosus (Fig. 1). Sections were also taken from other muscles where pale areas were observed. The lesions could be described as either acute or chronic.

In acute lesions, the fibres appeared distorted,

swollen, disrupted, and clumped with a loss of striations. The nuclei were swollen, there was separation of the myofibrils, and macrophages were occasionally present. Many of the less affected fibres had a granular degeneration of the cytoplasm and large numbers of red blood cells were present between the fibres. In chronic lesions, the sarcolemmal cells were well rounded and tended to form lines, and in places many more macrophages were present. Both regeneration and fibrous tissue were present together.

On an anatomical basis it was found that muscles on 1 side of the neck were often more affected than the other. This was most pronounced in animals with "wry" neck, where muscles on the concave side were less affected than those on the convex side. Generally the back muscles were less affected than the neck muscles, which were less affected than the shoulder or leg muscles.

Postcapture myopathy in deer

The foregoing findings strongly suggest that the changes associated with postcapture myopathy of deer have a great similarity to the biochemical and histological lesions found in wild animals in South Africa and to those of the porcine stress syndrome. It is therefore unfortunate that analyses for the catecholamines and the adreno-corticosteroids were not made.

Experimental myopathy has been produced in rats from an excess of acetyl choline (Fenichel *et al* 1974). It is unfortunate that histological examination of muscle was not undertaken by Selye and that when muscle lesions were found they were considered to be a result of vitamin E/selenium deficiency.

The changes in the biochemical parameters observed have obviously resulted from the stress before capture and organ damage prior to and during capture. Some of the variation observed was probably due to the variation in the speed and duration of the chase.

Stress and the Thymico-lymphatic System

Stress in the myopathic deer was severe: it caused the white cell count to be greatly reduced (Table 1), the germinal centres of the spleen to be deficient in leucocytes, and the lymph nodes themselves although not deficient of leucocytes suffered congestion and some haemorrhage (*see* Appendix).

Other forms of stress in deer may not cause such spectacular changes, but nevertheless prolonged exposure to a stressor, e.g. cold or nutritional stress, suppresses the immune response to invading organisms because of the involution of the thymico-lymphatic system. As well there would be an anti-inflammatory effect of the glucocorticoids which would reduce localisation of infections and impair wound healing.

Conclusion

The state of stress is a physiological response to adverse environmental factors and involves essentially a mobilisation of energy reserves to fuel whatever specific adaptive reactions may be necessary to cope with the adverse factor or stressor (McDonald 1979). The nature of the stress response is always the same regardless of the nature of the stressor (Selye 1946): the only correlation between stress and stressor is quantitative.

The maintenance of a state of stress has a

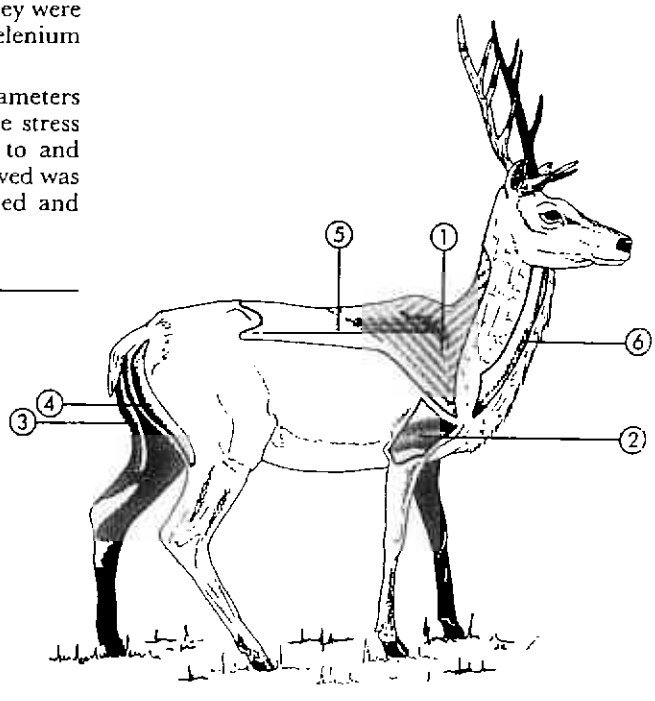

Fig. 1: Muscles most frequently affected with gross myonecrosis—1. M. trapezius; 2. M. tricepsbrachii; 3. M. semitendenosus; 4. M. semimembranosus; 5. M. longissimus dorsi; 6. M. cleidobrachialis.

metabolic cost which may ultimately lead to a wide variety of disease states consequent upon protein wastage, suppression of the immune system, suppression of the anti-inflammatory response to tissue damage, and induced abnormalities of organ function; for example, the prevalence of yersiniosis and malignant catarrhal fever in deer in winter (McAllum 1980) may be due to prolonged exposure to the stressors cold and low nutrition. Physiological changes due to stress may also confuse the responses from animals and therefore the results in experimental situations.

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Appendix

Histopathology

Representative samples from each myopathic animal were examined:

• *Lung:* Severity of the lesions varied. Nearly all lungs studied showed a generalised mild oedema, congestion, and focal alveolar emphysema. Sections of adult and larval forms of the lungworm *Dictyocaulus viviparus* were found in the alveoli and bronchi. Eosinophils were commonly found around blood vessels and were probably related to the lungworm infestation.

• *Liver:* Animals which survived for a few hours showed large amounts of oesinophilic granular material in the hepatic sinusoids. There was generally a mild congestion of the central veins and sinusoids. In other cases many of the hepatocytes showed eosinophilic degeneration through to a coagulative necrosis; these resembled necrobiotic cells, but increased in number from the periphery of the lobule to the central vein. Occasional animals had central hepatic veins thickened with collagen fibres.

• *Kidney:* The following lesions, varying in severity and extent, were commonly observed: the glomeruli were congested. The juxta-glomerula apparatus was occasionally prominent. In severe acute cases the glomerula fronds appeared contracted leaving an enlarged uriniferous space which contained either globules or larger coalescing amounts of proteinaceous-like material. Staining with Perl's Prussian blue stain for iron gave a light blue reaction in contrast to the more intense blue expected for haemosiderin. In severe cases where dying was prolonged there was often a pronounced nephrosis. The proximal tubules were always involved and there was often a fine vacuolation and/or a granular degeneration in the distal tubules.

• *Adrenal glands:* The medulla appeared less basophilic than normal and contained areas of congestion. Granular degeneration was present in cells of the zona fasciculata which also contained numerous diffusely distributed necrobiotic-like cells. There were fewer cells with fat globules in the congested areas of the z. fasciculata and z. reticularis than elsewhere.

• *Thyroid glands:* Generally the thyroids were normal, i.e. a cuboidal epithelium surrounded a well filled acinus of well stained colloid. Occasional thyroids showed a thickened interstitium which was due to marked congestion and the presence of some colloid. Others showed signs of having recovered from thyroid hyperplasia, the epithelial cells being cuboidal or columnar and with involution of the walls still present. However, the colloid was well stained and homogeneous. Exhaustion was suggested in a few by the presence of macrophages in poorly stained ragged colloid.

• *Gastrointestinal tract:* Sections examined from the abomasum, duodenum, jejunum, ileum, and caecum showed some autolysis. No abnormalities were seen.

• *Lymph nodes:* All lymph nodes examined had congested blood vessels and occasionally red blood cells were present in the sinuses. All nodes had macrophages containing large numbers of greenish brown granules (Perl's positive) in the medullary sinuses. The germinal centres appeared normal, but the nodes associated with the intestine (hepatic, mediastinal, and ileo-caecal) all contained numerous eosinophils.

• *Spleen:* The 3 examined were all heavily congested. The germinal centres were small and with a few lymphocytes present.

• *Brain and cervical cord:* No changes were seen in the 7 brains or cervical cords examined. A few lymphocytes were present in the meninges and there was a suggestion of satellitosis in the white matter.

• *Myocardium:* Transverse and longitudinal sections of the interventricular septum showed pathological changes in 4 cases. There were occasional swollen fibres, loss of striations and a mild granular degeneration, focal Zenker degeneration, oedema, sarcolemmal cell enlargement depending on whether the condition was acute or not, and enlargement of the centrally placed nuclei. Sections from the other walls of the ventricles showed similar lesions. It was noticed in more than 1 heart of animals with the acute syndrome that the Purkinje fibres appeared ragged at the edges and the cytoplasm depleted and granular.