POST CAPTURE MYOPATHY SYNDROME IN RED DEER (*CERVUS ELAPHUS*)

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ABSTRACT

The capturing of red deer from the wild to stock deer farms has brought with it problems of stress. Large numbers of deer have died due to poor catching techniques and inappropriate drugs. Efforts to minimize these deaths require an understanding of the physiology, pathology and epidemiology of the changes occurring within the animal and during capture.

To assist in the correct interpretation of the data collected, normal haematological and biochemical parameters had to be established. This was carried out on deer of different age groups and sex from deer farms. In addition the effects of the commonly used capture drugs on the biochemical parameters were established.

Blood and serum were obtained from captured animals at the site of capture and where possible further samples were obtained from these animals at set intervals.

The biochemical parameters found to vary from the normal in captured animals were pH, Pco_2, lactate, SGOT, (Aspartate aminotransferase), blood urea nitrogen, and potassium. The changes in these parameters clearly indicated a profound acute or delayed lactic acidosis and severe muscle damage both skeletal and cardiac. The captured animals were divided into those which survived (captured) and those which died (myopathic). It was found that the changes in the 'myopathic' group were more profound than in the 'captured' group.

The rising blood urea nitrogen levels and damaged cardiac muscle may account for the delayed deaths from uraemia due to a severe nephrosis and cardiac failure.

The clinical effects on captured animals were recorded and those that died in both the acute and delayed form were necropsied. The gross and histological lesions were described. The most obvious clinical changes in addition to temperature, respiration and heart rates were lameness, recumbency and the wry neck. Histologically, the muscle changes resembled those found in white muscle disease of domestic ruminants in this country.

The epidemiological studies suggested certain simple measures could be taken to reduce the effects of the respiratory depression resulting from the drugs and transportation, to reduce the stress of capture and to allow acclimatisation to the new conditions. These were (1) that less or no Nalorphine be used, (2) that the animals were caught early in the year, (3) that young smaller females were preferred to males (4) that a loose bag totally enclosing the animals was used, (5) that darkened conditions helped keep the animals quiet and (6) all captured animals should be retained in a dark house for two or more days before release into the paddocks.

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POST CAPTURE MYOPATHY SYNDROME IN RED DEER (CERVUS ELAPHUS)

INTRODUCTION.

The post capture myopathy (PCM) syndrome as it affects wild animals is described by Basson and Hofmeyr (1973) as being characterized by ataxia, paresis or paralysis with the excretion of brown coloured urine. Associated with these clinical changes are asymmetrical muscular and myocardial lesions. Other synonyms for the condition are white muscle disease, white muscle stress syndrome, stress myopathy, overstraining disease, and capture myopathy.

Species of animals and birds affected are listed in Table I. A similar syndrome has been observed in red deer (Cervus elaphus) in New Zealand.

The condition is usually associated with the capture of wild animals but a similar syndrome occurring spontaneously has been reported in the Peking duck (Rigdon 1961). Also the post capture myopathy syndrome has clinical and pathological similarities to the white muscle disease syndrome of calves (Fairlie 1964, Jarret et al 1964), paralytic myoglobinuria (azoturia) of horses (Young and Bronkhurst, 1971) and paroxysmal myoglobinuria of man (Seibold et al 1971). In addition muscle lesions found in the porcine stress syndrome and malignant hyperthermia of Landrace and Pietrain pigs, are similar to those found in PCM (Ludvigsen 1953, Briskey et al 1959a).

TABLE I

List of species affected by Post Capture Myopathy

Roebuck	Capreolus capreolus	Fairlie 1964
Hunter's antelope	Beatragus hunteri	Jarrett et al 1964
Red hartebeeste	Alcelaphus buselaphus	Young 1966
Springbok	Antidorcas marsuplialis	" "
Blesbok	Damaliscus dorcas phillipsi	"
Bantebok	D. dorcas dorcas	Young "
Eland	Taurotragus oryx	" "
Gemsbok	Oryx gazella	Ebedes 1969
Giraffe	Giraffa camelopardalis	" "
Roan antelope	Hippotragus equinus	Basson et al 1971
Sable antelope	H. niger	" " "
Burchell's zebra	Equus birchelli	Young & Bronkhurst 1971
Mountain zebra	E. zebra hartmannae	Basson et al 1971
Kudu	Tragelaphus strepsiceros	" " "
Nyala	T. angasi	" " "
Steenbok	Raphicerus campestris	" " "
Blue wildebeeste	Connochaetes taurinus	" " "
Black rhinoceros	Diceros bicornis	" " "
Elephant	Loxodonta africana	Young 1971
Oribi	Ourebia ourebi	" 1972
Tsessebe	Damaliscus lunatus	" "
Greater flamingo	Phoenicopterus ruber roseus	" 1967
Lesser flamingo	Phoeniconaias minor	Siebold et al 1971
Mountain goats	Oreamnos americanus	Hebert & Cowan 1971
Virginia deer	Odocoileus virginianus	Wobeser et al 1976

Very little has been published on the post capture myopathy syndrome as it affects the red deer in New Zealand. This review therefore discusses the various factors which may contribute to the syndrome in various species of wild game and birds throughout the world and where applicable draws comparisons with other similar disease syndromes. In addition current knowledge of clinical signs, pathological and biochemical changes which are associated with the disease are detailed.

PREDISPOSING FACTORS

It would seem that capture and transportation methods play an important role in predisposing animals to the post capture myopathy syndrome.

In their introduction Hofmeyr et al (1973) stated: "The recent increase in capture and transport operations involving large game has focused attention on a characteristic shock syndrome. This syndrome ... occurs most frequently when animals are manhandled or restrained for several hours or more without judicial use of suitable tranquillizers or immobilising drugs ...".

Capture methods

Pharmacological -

Most methods have entailed the use of a drug. They are many and varied and a particular choice is usually made with respect to size and species of animal to be captured. For example, etorphine a derivative of thebaine an alkaloid occurring in opium, has been successfully used in conjunction with acetylpromazine

in capturing and controlling large species of animals such as the giraffe, elephant and rhinoceros (Harthoorn 1973). Other drugs used, have been xylazine (Rompun*) and fentanyl hydrochloride/azaperone mixture (Fentaz**). The use of morphine derivative drugs such as Fentaz has enabled the use of antagonists such as nalorphine (Lethidrone***) which give an almost immediate reversal of the drug action. These are usually injected before the animals are carried in large canvas bags, in a dog sitting position to some restricted area. A disadvantage of nalorphine is that it is a respiratory depressant.

In many instances the administration of drugs has entailed a period of chasing which may have initiated an acidosis (see Biochemical changes p.11). The chasing has usually been done on foot, vehicle or helicopter with the drug being administered by dart gun or cross bow (Pienaar 1973). An excellent review of the drugs used with their pharmacological properties is given by Harthoorn (1973).

Mechanical

In some species, because of drug intolerance, mechanical methods of capture have been found to be more suitable. Examples of such methods are spotlighting with the blinding of the animals, lassooing from fast vehicles, nets, (either drop nets in the bush or from helicopters), and chasing animals along converging

* Bayer (Henry H. York)
** Ethnor Pty. Ltd.
*** Wellcome N.Z. Ltd.

fences which lead to a large pen (Pienaar 1973). In New Zealand individual deer have been captured in pens where a trip wire released a spring gate behind the animal (R. Brooks pers. comm.). A jump fence is often used. This is where a fence of a deer paddock is made to appear low, approximately 1 m, from the outside but because of the excavation at the foot it is at least 2 m on the inside. This has made it easy for animals to enter but difficult to escape. The use of a turnip crop has been used to attract them.

The dropping of nets from helicopters has been tried during the winter capturing periods but was unsuccessful. Bulldogging i.e. the dropping of a man from a helicopter has been used for calves, hinds and yearlings, but this has required heavy falls of snow. The snow slows or immobilises the animal (Wallis and Faulks 1976).

Transportation

A full description of the problems of transportation for individual or groups of animals has been given by Hirst (1973). He found that "under field conditions the operator normally has little option but to transport stressed animals, however, he can minimise the effects by efficient handling and loading with a minimum of disturbance and noise."

It has been well documented that in domesticated animals transportation with its associated stresses can cause many problems. A good example is the transport tetany in feed lot lambs (Pierson and Jensen 1975).

Transportation methods differ for each species of animal. For wild deer transport has usually been by motorised vehicle with a subdivided form of loose box which may confine two or three animals in each compartment.

There are two stages of transportation:

(i) Transport from the site of capture to the central collection point.

(ii) Transport from the central collection point to holding pens on the farm.

The first stage, depending on the terrain, sometimes required the use of a helicopter (Wallis and Faulks 1976). A flying time of up to 30 minutes was reported.

Transport from the central collection site has usually been by motorised vehicle and often over rough roads. It was found essential that each animal had enough room to avoid being trampled when lying recumbent.

For short distances feeding and watering facilities have not been found necessary. Floor covering in the form of straw or sand to allow a good footing and a tarpaulin spread over and around the top to protect the animals from wind and sun have been recommended (Hirst 1973).

Tranquillization

Tranquillizers given prior to transport have allowed easier and safer handling of fractious animals but have not been considered a substitute for efficient handling and desirable facilities (Hirst 1973). With tranquillizers animals have been more likely to become recumbent; this has tended to reduce the stress of transport but may lead to greater

danger of trauma from trampling.

The tranquillizers most frequently used are acetylpromazine and chloropromazine. The benzodiazepine derivatives (e.g. Valium*) have been found useful for more fractious animals (Hirst 1973).

Traumatic injuries are a common sequel to tranquillization and should be treated as soon as possible (Hirst 1973). Tranquillizers have also affected the heat regulatory mechanisms and hypothermia during transport has often been a complication. Another complication has been pneumonia due to inadequate protection from wind; the occurrence of such infections has been reduced by the administration of antibiotics (Hirst 1973).

It was also recommended by Hirst (1973) that ungulates on long journeys should not be allowed to remain recumbent for more than one hour. Regular disturbance from recumbency reduced problems of circulation in the limbs.

The release site should be clear of dangers such as wire fences, holes, large stones etc. and should be level. Hirst (1973) recommended that animals be released with little fuss or noise from the transporter into a small enclosure which is boarded up, to allow the animals to recover from tranquillization and become calm after their ordeal. The exit gate should then be opened gently and the animals allowed to venture forth at their leisure.

* Roche Products (N.Z.) Ltd.

THE DISEASE PROCESS

Clinical signs

The clinical disease varies according to whether the case is 'acute' or 'chronic' i.e. whether the animal dies within 30 minutes to 48 hours after capture or whether it lives for several days or weeks. Signs have appeared irrespective of the methods or drugs used for capture.

In chronic cases, death from heart failure may occur at any time up to one month after capture without prior warning signs (Young 1966). However, it is more usual to observe some symptoms. These may include a clinical stiffness and lameness due to the improper functioning of muscle groups. This may be bilateral, but the development of the signs may progress asymmetrically. If the neck muscles are affected torticollis and opisthotonus may be seen. Depression, dehydration and/or myoglobinuria may occur. In the acute case laboured respiration and tachycardia occur, along with pain, muscle tremors and often tetraplegia (Young 1972).

In addition to the above symptoms, Wobeser et al (1976) found that a white-tailed deer (Odocoileus virginianus) on the day following capture, appeared oblivious to humans and offered no resistance to handling. The rectal temperature was 37.8°C, heart rate 98/min and the respiratory rate was irregular. The eye-body attention and swallowing reflexes were absent and there were no withdrawal reflexes in any limbs. However, the patellar reflexes were brisk and the anal reflex was normal.

In the experimental condition induced by chasing zebras over short distances Hofmeyr et al (1973) found the cardiac and respiratory rates to be elevated and that a moderate but significant increase in rectal temperature also occurred (Table II). These changes were considered to represent normal physiological adjustments to strenuous exercise but it was considered that the differences between the experimental group and the controls showed the former to be under considerable stress. However, M99 Etorphine hydrochloride, the immobilising drug used causes tachycardia and respiratory depression. Because of this no definite conclusions were drawn.

Harthoorn et al (1974) also experimented with zebras but immobilised the animals manually and not by drugs. The animals were given extensive exercise over short distances and the clinical parameters were measured shortly after capture. They also found that respiratory, cardiac rates and rectal temperatures were elevated. Although different distances were run the parameters' values generally appeared to be higher than those recorded by Hofmeyr et al (1973). Despite complete rest one animal's heart rate rose to 360 beats/min. and the respiratory rate remained rapid (56/min). Another animal's temperature rose to 43.0°C. Clinically Harthoorn et al (1974) found 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 min to 12 hours after capture.

Heart rates of 112-125/min, respiratory rate 40 to 96/min. and rectal temperatures 41°C to 42°C were

TABLE II

Comparison of rectal temperatures, cardiac and respiratory rates between control and chased zebras (Hofmeyr et al 1973).

	Control n = 5	S.D.	Experimental n = 6	S.D.
mean cardiac rate/min	68.0	± 13.4	152.3	±44.3
mean respiratory rate/min	13.7	± 4.6	24.7	± 5.8
mean rectal temperature (^{o}C)	38.73	± 0.49	40.04	± 0.39

obtained by Harthoorn and Van der Walt (1974) from Blesbok after chasing them over various distances and catching them manually.

Physiological and biochemicl changes

An apprehensive animal can suffer physiological and pathological changes which according to Basson and Hofmeyr (1973) result from a massive release of adrenaline as part of the reaction to fright and fear and to an acidosis as a result of profound biochemical changes.

Hofmeyr et al (1973) investigated the blood biochemistry in zebras and found that animals chased to exhaustion before darting had irreversible biochemical changes. By contrast, animals usually survived when there was minimum alarm and disturbance. Hofmeyr and co-workers noted a similarity between the blood chemistry of chased animals and one that had died from the condition on a previous occasion. It was concluded that the biochemical changes were typical of capture myopathy and that because of the persistence of such constituents as lactate in the blood, such changes were prolonged.

Harthoorn et al (1974) also found that animals pursued intensively over short distances are the most susceptible to capture myopathy. In affected zebras they observed changes in blood pH, base excess, buffer base, plasma and total bicarbonate; the degree of change was consistent with an acute and profound acidosis.

In a study of nine selected animals, they estimated

the distance each animal ran before capture and measured the respiration rate, pulse rate and temperature. Six animals which received treatment (see p.19) survived, whereas the untreated zebras died thirty minutes to 12 hours post capture. The blood pH in these latter animals dropped to about 6.5 and the heart and respiration rates remained rapid despite complete rest. Pulmonary oedema occurred in the untreated animals within thirty minutes to 12 hours post capture. It was believed by Harthoorn et al (1974) that fear and shock, also contributed to the acidotic state.

Variation in species susceptibility has been reported. Harthoorn and Van der Walt (1974) found that the blood pH fell more in blesbok (Damaliscus dorcas phillipsi) than in the zebra (Equus sp.) after animals were intensively chased over short distances of about two kilometers. Animals chased over longer distances suffered a less severe acidosis probably because of compensatory mechanisms coming into play.

It appears that the myopathy syndrome with its consequent biochemical changes is more severe following a short period of maximal exercise. Acid-base studies in some detail have been done on humans. Osnes and Hermansen (1972) found that short periods of maximum exercise in humans resulted in an acidosis, mainly resulting from a high lactate level (32.1mmol/ℓ) low plasma bicarbonate (2.6 m Eq/ℓ), pCO_2 (14 mm Hg) and a base excess of -34.0 m Eq/ℓ. There was a high negative correlation between lactate and bicarbonate

levels. The main conclusions made by Osnes and Hermansen (1972) were that the work intensity, duration and type of work, and the amount of muscle mass involved control the level of lactate attained in the blood and that this level was higher following maximal intermittent exercise than during maximal continuous exercise.

Another possible factor affecting the production of lactate is the animal's degree of fitness. It has been shown by Jorgensen and Hyldgaard-Jensen (1975) that unfit pigs when exercised build up a high level of lactate in the blood which is slow to return to normal. In contrast pigs which were trained on an ergometer for a period of one year and similarly exercised, had a brief initial rise of lactate which dropped below the normal resting level during the exercise. Histochemical studies of the muscle fibres indicate that the main differences between the fit and unfit pigs were the higher level of myoglobin and the 5th isoenzyme of lactate dehydrogenase (LDH_5) present in the fit animals. The most intense exercise required by wild animals would be of short duration when escaping from a predator. Long arduous runs would be uncommon.

The role of adrenaline

An animal in a state of fright releases adrenaline into its circulatory system. The effect of adrenaline has been studied in pigs which have a stress adaptation difficulty and show the charateristic signs of muscle tremor and reluctance to move. These clinical signs may progress to marked dyspnoea, irregular alternating blanched and reddened areas of skin, increased body

temperature, cyanosis and development of an acidosis condition. It may be followed by total collapse and death. These pigs are referred to as "stress susceptible" pigs (Topel et al 1968). Post mortem changes show a variety of muscular changes including those closely resembling post capture myopathy.

The levels of adrenaline secretion following stimulation of pigs by electrical probe have been studied (Van der Wal 1970, Topel et al 1974). It was found that there was a considerable and immediate elevation of adrenaline in both normal and stress susceptible pigs. The level in the stress susceptible group was 8-10 times the base level and in normal pigs 4-5 times. Thirty and sixty minutes after stimulation the level of adrenaline was higher in the stress susceptible group than in the normal.

Mechanisms as to how high lactate levels occur have been studied in stress susceptible pigs. It is possible that similar mechanisms operate in the post-capture myopathy syndrome, although no stress susceptibility such as occurs in pigs has been shown to occur in wild animals. Adrenaline stimulates glycogen breakdown with resultant elevation in blood glucose levels (Krebbs et al 1966). In stress susceptible pigs high levels of blood glucose are formed. When muscle cells reach their metabolic capacity, nicotinamide-adenine dinucleotide (NAD) which is required for glycogen breakdown is regenerated from the reduced form by converting pyruvate to lactate (White et al 1968). The lactate produced diffuses rapidly from the muscle cells into the blood (de Duve and Hers 1957). In stress susceptible pigs blood lactate levels can increase from 1.57 mmol/l

to 19.04 mmol/l in a few minutes when stress such as forced exercise is applied (Weiss et al 1974). The rate of pH decrease is more rapid than in normal pigs and takes nearly twice as long to return to normal (Topel et al 1974). The rapid accumulation of blood lactate, along with the high muscle lactate is a major factor in muscle necrosis (Topel et al 1974). These changes in conjunction with the extreme acidotic condition which develops, appear important in explaining the high incidence of deaths in these animals.

Hofmeyr et al (1973) found that blood glucose levels in chased zebra had risen markedly an hour after immobilization with an etorphine hydrochloride (M99)/acetylpromazine mixture. An adrenaline role was postulated or a depletion of oxygen reserves and a switch to anaerobic metabolism. There was also a marked rise of blood glucose in an immobilised standing group of animals and it was possible that this could have been due to the drug. Lactate levels also rose markedly in the chased group.

Adrenaline also plays a role in respiration. Following a large intravenous dose of adrenaline there is a period of apnoea (Lewis 1965). This is a reflex due to the effect of the high arterial blood pressure upon pressure receptors in the carotid sinuses and aortic arch. Smaller therapeutic doses stimulate respiration, increasing rate and depth. Pulmonary ventilation is increased, as is the consumption of oxygen. One of the physiological roles of adrenaline is probably to increase pulmonary ventilation and hence to increase the efficiency at which blood can be

oxygenated (Lewis 1965).

Some other effects of adrenaline are:-

(a) A rise in serum potassium and cholesterol and a fall in serum organic phosphate levels.

(b) An eosinopenia due to a non-specific action on the anterior pituitary adrenocortical system.

(c) An increased rate of secretion of hormones from the adrenal cortex via the anterior pituitary. The effects of the anterior pituitary are accompanied by a fall in ascorbic acid and cholesterol in the adrenal glands.

In conditions of stress it is likely that adrenaline is responsible for the rapid response by the adrenal cortex. If the stress condition persists then some other mechanism must take over (Lewis 1965).

Miscellaneous biochemical changes

While much of the later biochemical work in post capture myopathy has been directed towards the study of acid/base mechanisms (Harthoorn and Young 1974: Harthoorn and Van der Walt 1974: Harthoorn et al 1974), some biochemical measurements were made earlier. The first of these was by Young (1966) in Red Hartebeeste. He measured the serum glutamic oxalo acetic transaminase (SGOT): serum glutamic pyruvic transaminase (SGPT) and blood urea nitrogen (BUN) in five captured hartebeeste 2-3 days post capture and again seven days later. These values were compared with another hartebeeste which had been captured a few weeks previously. It was found that the SGOT, SGPT and BUN values for the first group of measurements were very much higher than the control. The levels for the survivors were much

closer to those of the control one week later. These levels were taken to indicate muscle damage rather than other organ damage. Hofmeyr et al (1973) measured, in addition to glucose and lactate, plasma creatine phosphokinase (CPK) and plasma osmolality in chased zebra. The CPK levels rose to a level four times that of the resting value but returned to normal within one hour.

In animals with severe lactic acidosis and muscle damage the levels of SGOT, CPK and lactate dehydrogenase (LDH) were found to be high and this was thought to indicate impending heart failure and shock (Harthoorn and Van Zyl 1972). The red discolouration of the plasma could be seen in some affected animals and was presumably myoglobin (Harthoorn and Van der Walt 1974). Levels of potassium have not been published in affected antelope and zebra but in stress susceptible pigs they have risen to levels of four times or more the normal level.

Haematological and urine changes

Siebold et al (1971) measured the haematological parameters of the Macaca monkey prior to and during the idiopathic myonecrosis and myoglobinuria afflicting it. They found that the haemoglobin, haematocrit and white cell counts rose on the first day of illness and then began to return to normal. The rise in white cells (mainly neutrophils) was supposedly due to muscle necrosis. The slight dehydration was treated with intravenous fluids. Urine examination on the second day of illness revealed cloudy yellow urine with a pH of 6.0 and positive values of albumin and sugar. Microscopically red blood cells could be seen in the urine along with granular casts.

It was found by Hofmeyr et al (1973) that animals suffering from more prolonged stress had increased

haematocrits which were significantly greater in the experimentally chased animals as compared with the experimental standing group ($P < 0.01$). Values had returned to normal within one hour. Changes in haematocrit and osmolality readings along with cardiac and respiratory rate changes were thought to be brought about by a reaction to catecholamines such as adrenaline which were released as a result of alarm and stress. In nearly every case, particularly the acute, myoglobin and haemoglobin were present in the urine.

Pathology

The first full description of the syndrome both macroscopic and microscopic was made by Young (1966). The main gross lesions were present in the skeletal and cardiac musculature. The lesions described in the liver, lungs, kidney and digestive system were mainly congestion with some nephrosis present in the kidneys. The muscle lesions appeared pale and necrotic with extensive and severe haemorrhages frequently being present. Similar lesions were present in the muscles of the auricles and ventricles.

Microscopically, Young (1966) described the skeletal and myocardial muscles as "a homogeneous mass with loss of striations which stained a deep pink with eosin". The sarcoplasm of some fibres were calcified and where severe changes had occurred the nuclei showed degeneration. Connective tissue replaced necrotic fibres, and Von Kupffer cells contained yellow-brown haemosiderin-like material. There was tubular nephrosis, congestion and focal haemorrhage in the kidney.

Later reports on the pathological changes of post

capture myopathy were similar to those of Young (1966). Basson and Hofmeyr (1973) noted that most lesions were in the muscles of the limbs and back. Although the muscles were bilaterally affected the lesions were not symmetrically distributed. Fibrotic areas were present in the myocardium of apparently healthy animals that died approximately one month after capture. In their detailed description Basson and Hofmeyr (1973) described the adrenals as sometimes being congested and haemorrhagic and the cortex atrophied. Eosinophilic globules were sometimes present in the cortical zona glomerulosa and the outer zone of the medulla. The spleen and lymph nodes were relatively small and atrophied in cases with adrenocortical atrophy.

THERAPY

From the careful work on acid/base imbalance which occurs in the post capture myopathy, Harthoorn and associates (1974) derived a successful treatment for affected animals. They infused 1000 m Eq of sodium bicarbonate dissolved in a solution containing the following ions Na^+, 140; K^+,5; Mg^{2+},3; Cl^-,98; acetate, 27; gluconate, 23m Eq/1 with an approximate pH of 7.4. All six animals treated after experimental stress survived whereas three untreated, which underwent the same stress, died within twelve hours (see page 12). About thirty minutes after capture the salt solution was administered over a period of 5-10 minutes resulting in the immediate cessation of clinical symptoms. Young (1966) suggested that treatment with vitamin E and selenium, systemic antibiotics, corticoids, antihistamines, methionine, vitamin B complex and urinary alkalizers may be of

value. Since then these have been tried and found to be of only limited value. Young (1972) later suggested that perhaps prophylaxis may be of greater use than treatment.

SUMMARY

The post capture myopathy syndrome has been described in many species of animals. It is similar to azoturia in horses, white muscle disease in cattle and sheep and paralytic myoglobinuria in man. All have in common the clinical signs of ataxia, paralysis and occasionally myoglobinuria.

Both the pharmacological and mechanical means of capture cause this condition. It is related to stress, particularly to forced excessive exercise over a short period of time.

The clinical signs may be acute or chronic and one of the commonest manifestations is the 'wry' neck.

Hofmeyr and his co-workers (1973) found the clinical and physiological changes represented physiological adjustments to exercise. Harthoorn et al (1974) in their series of experiments with zebra and blesbok found increased respiratory and cardiac rates and rectal temperatures, changes in pH, base excess, buffer base and bicarbonate consistent with an acute and profound acidosis.

Lactate levels were found to be the most reliable indicator of the degree of anaerobic respiration. The ability to cope with the lactate may relate to the fitness of the animal as found in pigs by Jorgensen and Hyldgaard-Jensen (1975), the unfit animal being unable to reduce the levels of lactate rapidly.

Blood lactate levels increase when the metabolic potential of aerobic respiration is reached, and if the

stress continues, adrenaline continues to stimulate the formation of blood glucose. After the change over to anaerobic respiration occurs the NADH utilizes energy to form NAD from the conversion of pyruvate to lactate, thus contributing further to the blood lactate levels. This mechanism is suggested from the studies on stress susceptible pigs.

In addition to the blood lactate levels rising, it seemed the muscle lactate levels also rose, to such an extent that degeneration of the muscle fibres occurred.

Adrenaline affects the respiratory system dilating the respiratory tubules and increasing the tidal volume which in turn increases the supply of oxygen and the excretion of carbon dioxide. The drugs used for capture are commonly morphine related compounds which with their antagonists are respiratory depressants and thus may assist in increasing the acidosis.

Hofmeyr et al (1973) found that the haematocrit and osmolality rose in stressed animals as well as cardiac and respiratory rates which they suggested were a response to catecholamines. The later biochemical analyses were directed toward the elucidation of the acid/base mechanism whereas the earlier work was directed towards the changes accompanying the condition. The main parameters which changed were SGOT, CPK, BUN, potassium and lactate.

The main gross pathological changes were found in cardiac and skeletal muscles particulary those muscles used in locomotion. Other organs were affected but the changes were moderate and non-specific. The affected muscles were bilateral but asymmetrical. It was found that muscle fibres appeared pale and necrotic in the acute cases but in the chronic cases regeneration and fibrosis had

occurred. Basson and Hofmeyr (1973) also described the adrenals as having atrophic cortices.

Harthoorn et al (1974) published a treatment found to be experimentally successful in stressed zebra counteracting the acidosis and reducing myonecrosis.

MATERIALS AND METHODS

INTRODUCTION

It is apparent from the literature review that there is no information on the clinical, biochemical and pathological changes that occur in the post capture myopathy syndrome in red deer. The available information refers to other species of animals and it has been assumed that similar changes occur in deer.

The experimental approach to this condition was fourfold.

1. To obtain haematological and biochemical data from clinically normal farmed animals.
2. To determine the effect of capture drugs on the various haematological and biochemical parameters in the undisturbed normal farmed animals.
3. To observe the capture and post capture methods used in the handling of red deer and where possible monitor the biochemical and haematological parameters during this period.
4. To observe as in (3) above and to subsequently perform a detailed pathological examination on deer which were affected by the post capture myopathy syndrome.

ANIMALS AND BLOOD SAMPLE COLLECTION METHODS.

The following animals provided material for clinical haematological and biochemical data.

Normal animals

Three groups of farmed animals were used.

(a) Normal data for respiratory rates, cardiac rates and temperatures were recorded from a group of 30 animals not used for the haematological and biochemical data, quietly herded into the Invermay deer farm yards, into a large familiar pen and left as a group for two or more hours. Groups of 6 or 7 were herded in succession into a small pen and the parameters were recorded.

(b) Thirty females from the Invermay deer farm, these were 11 at 9 months of age and 19 at 14 months of age. They were handled in similar fashion to (a) and then were individually held while blood was collected under sterile conditions from the jugular vein.

Occasional EDTA bloods were found to have clotted and were discarded.

(c) Eightysix slaughtered male farm animals of varying ages of two years and older.

These were herded into holding yards on the evening prior to slaughter and allowed to stand and quieten. On the following morning each in turn was confined in a small holding pen and stunned with a 0.22 bullet and exsanguinated. Blood samples were collected immediately after the initial strong flow.

Capture and myopathic animals

Twentythree animals were studied.

A series of blood samples were taken. beginning as soon after capture as possible usually within 30 minutes to 1 hour, and at 1 to 2 hourly intervals thereafter. These time intervals depended on the rate and number of animals captured.

Blood samples and handling procedures

Wherever possible a standard procedure for the collection and subsequent handling of blood samples was adopted. This standard procedure was decided upon after a literature survey and pilot experimentation. (Schmidt et al 1953, Pennock and Jones 1966, Anderson 1969, Clark et al 1970, Lewis and Stoddart 1971, Manston et al 1974, Gregoryand Reid 1976).

Blood samples

Blood was collected into 3 types of containers:- Two 10 ml glass vacutainers(1) with heparin as anticoagulant. This blood was used for pH determination and biochemical tests which did not require the use of an autoanalyser (see under Analytical Methods).

Two 15 ml. glass vacutainers for serum collection. The harvested serum was used for biochemical measurements by the autoanalyser. One polystyrene plastic irradiated container(2) containing dipotassium salt of ethylenediamine tetra-acetic acid (EDTA) as an anticoagulant was used for haematological examinations.

(1) Becton Dickinson Co. Ltd.

(2) Tasman Vaccine Laboratory

Blood handling procedure

Normal Animals

The standard procedure for the handling of blood samples collected from the normal farmed deer at Invermay was as follows:-

Each container was labelled with the animal's ear tag number.

The blood samples were placed in a polystyrene foam holder. This held them in the upright position and protected them from breakage. A smear of the EDTA blood was made within 5-30 minutes of the sample being taken. Blood pH values were determined from one of the heparinized samples as soon as possible; normally within 10-15 minutes.

All bloods were stored within a cool polystyrene bin to prevent temperature changes and consequent haemolysis (Manston et al 1974).

The heparinized and clotted blood were centrifuged at the laboratory within 1-2 hours after collection. The plasma and serum were transferred into four appropriately labelled 5 ml Bijou bottles and frozen until required for analysis.

Immediately after centrifuging and before freezing, one bottle of serum from each animal was used for the determination of total CO_2.

The haemoglobin (Hb), packed cell volume (PCV) and white cell count (WCC) were determined as soon as possible on returning to the laboratory. This was normally within one hour.

Duplicate samples of blood collected into EDTA were often taken for comparison studies.

The handling procedure for blood collected from the normal stags was similar except that because of distance

from the laboratory total CO_2 determinations and freezing of serum and plasma samples were delayed from up to 4 to 6 hours after collection. They were, however, stored in a chilled Dewar flask until the laboratory was reached.

Captured and myopathic animals

The method of preparing the blood samples from these animals differed slightly from those for the normal samples due to the isolation of the capture sites from the laboratory.

The capturing of animals extended over a period of 3-5 days at any one time and usually 200 to 250 miles from the laboratory, often in very isolated mountainous areas.

The pH levels were determined in the field within 5-10 minutes of the blood being taken or as soon as possible using the same method as for the normal bloods. Blood smears were also made within 15 minutes.

All bloods were kept in the polystyrene holders in polystyrene foam plastic bins until return to the nearest source of electricity: this usually being the farm homestead.

Blood samples were then centrifuged, and the Bijou bottles containing serum or plasma were stored in a heavily insulated Dewar flask containing frozen ice packs. The samples were frozen on arrival at the laboratory.

EPIDEMIOLOGICAL DATA COLLECTION

Captured animals were studied at Mesopotamia Station, Rangitata Valley (2 occasions); Papamoa deer farm Glenavy (3 occasions); St. Bathans Station, St. Bathans (2 occasions); and Haldon Station McKenzie Country (2 occasions) (fig.1). Periods of working with the capturing teams took place on the latter two stations,

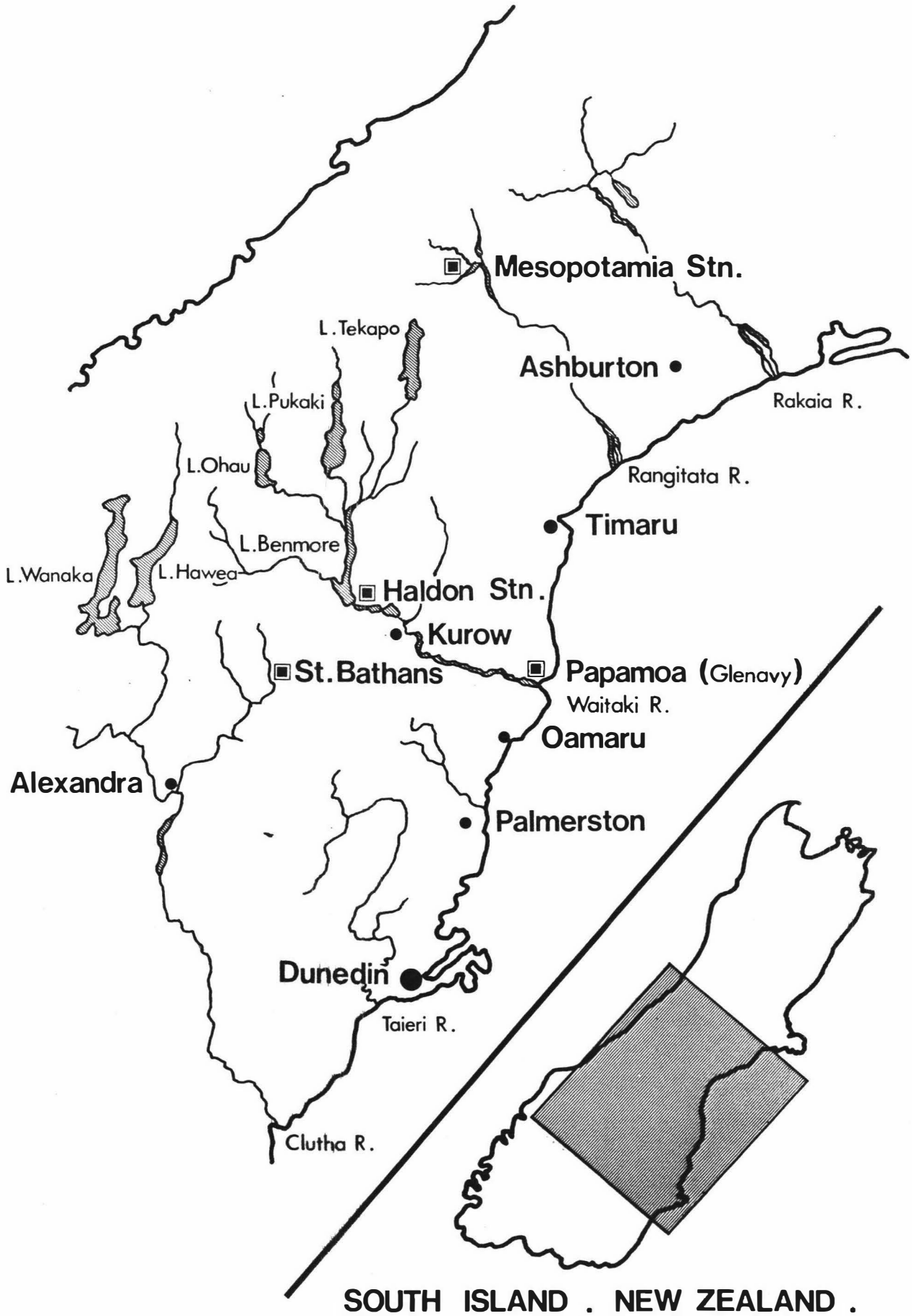


Fig. 1. Locations where capture of deer was studied

from September to December 1976.

The data record sheet used is given in Table III. As it was impossible to observe from the helicopter at the time of tranquillization much of the data had to be recorded from the observations of the helicopter pilots.

POST MORTEM PROCEDURES

Necropsies were performed on deer that died of the myopathic syndrome.

Gross pathological findings were verbally recorded on tape as the necropsy proceeded. Selected samples of lung, myocardium, liver, kidney, adrenals, thyroids, abomasum, duodenum, jejunum, ileum, caecum, retropharyngeal lymph nodes, tonsils, spleen, brain, and meninges, cervical cord, and skeletal muscle from the neck, foreleg, back and hing leg were preserved in 10% formalin. Where possible urine was taken. In later cases, samples from liver, kidneys and skeletal muscles only, were selected. Photographs were taken of any gross lesions.

Tissues for histopathology were paraffin embedded using standard techniques and were stained with Harris's haemotoxylin and eosin. In addition skeletal muscles were stained with phosphotungstic acid haematoxylin and the kidneys with Perl's Prussian Blue.

ANALYTICAL METHODS

Haematology

Standard techniques were used.

Haemoglobin (Hb) concentrations were estimated by the cyanmethaemoglobin method. Commercially prepared Drabkins diluent (3) was used.

(3) Medi-Chem Inc.

TABLE III

Epidemiological data record sheet.

Date:

Geographic position:

Climatic conditions:

Capture with or without drugs:

Drugs used:

Volume:

Strength:

Site of injection:

Delay before going down:

Delay before antidote given & quantity:

Approximate distance run:

Behaviour of animal:

Recovery rate:

Respiratory rate:

Heart rate:

Rectal temperature:

Age if possible:

Sex:

Nutritional state:

Transportation (a) from capture site

(b) to farm

The optical density of the test dilution was read on a Perkin-Elmer 124 Spectrophotometer at 545 nm and compared with standard solutions(4).

Packed cell volumes (PCV) were measured by the microhaematocrit method. Blood was centrifuged for 5 minutes in a Hawksley(5) microhaematacrit centrifuge.

Mean corpuscular haemoglobin concentrations (MCHC's) were calculated from the formula $MCHC\ (g/dl) = \frac{Hb\ (g/dl)}{PCV}$

Total white cell (WCC) numbers were counted in a Hawksley(5) counting chamber with an improved Neubauer ruling. Blood was mixed with 2% acetic acid at a dilution of 1 in 20.

Blood smears were stained with May-Grunwald-Giemsa. Differential counts were performed using the standard battlement technique (Schalm et al 1975).

Biochemistry

Blood pHs were measured in the initial stages of this study using the I.L. 217/317 Blood Gas Laboratory(6) but this was discontinued due to difficulties of access to this equipment. All blood pHs were subsequently measured with a Transistorized Analytical Pocket pH meter No. 101(7) by inserting the pH electrode into the vacutainer of heparinized blood.

Before use standard phosphate buffer solutions of

(4) Dade Division American Hospital Supply Corpn.

(5) Hawksley & Son Ltd.

(6) Instrument Laboratory Inc.

(7) Analytical Measurements A.D.D. Middlesex.

6.0 and 7.0 were used to calibrate the instrument. The meter was allowed to equilibrate with the blood and the scale read to the nearest half division (equivalent to 0.05 units). The electrode was washed clean with distilled water and dried before re-use. The measurement was then repeated.

Total Carbon Dioxide (CO_2) was measured with a Harleco Total CO_2 Apparatus(8) according to the method described by the manufacturers. The method is a simplified version of, and has been found to be comparable to, the classical Van Slyke method (Peters and Van Slyke 1932, Gentry and Black 1975). Sera were allowed to equilibrate with room temperature (20-25°C)before determination.

Partial pressure of carbon dioxide (PCO_2) and Base Excess were calculated from the known pH and total CO_2 values, using a Siggard-Andersen alignment nomogram (Henry et al 1974).

Lactate levels were determined in the supernatant fluid after 2 mls of phosphoric acid were added to whole blood, and by the ultra-violet method using Boehringer(9) kits. Readings were made on a Perkin-Elmer 124 double beam spectrophotometer using ultra-violet light at a wavelength of 366 nm.

Duplicate samples and controls were tested in each batch of blood.

(8) Harleco, A Division of American Hospital Supply Corporation.

(9) Boehringer Mannheim G.M.B.H.

Urea was measured in serum using the Auto-analyser method N-1e. The urea reacts with diacetyl monoxime (2,3 - butane dione-3-oxime) in the presence of thiosemicarbazide in acid (Marsh et al 1965).

Serum Glucose was estimated by an Autoanalyser II method which is based on the reduction of copper neocuproine chelate (Bittner and McCleary, 1963).

Glutamic oxaloacetic Transaminase (GOT) (Aspartate amino-transferase.) in serum was estimated by the method of Reitman and Frankel (1957) using a Sigma Kit(10).

Calcium and Magnesium in serum were estimated by an atomic absorption spectrophotometer using disodium E.D.T.A. diluent (Willis 1960).

Sodium and Potassium in serum were estimated by the standard technique of flame emission spectroscopy using a caesium chloride diluent.

Phosphate was estimated in serum using the Autoanalyser II molybdenum blue method using stannous chloride reductant (Kraml 1966).

Total Protein in the serum was estimated using the biuret reaction.

The albumen/globulin ratio was calculated following electrophoretic separation on cellulose acetate plates. After buffering the plates were placed in an electrophoretic bath(11) containing a Tris barbital buffer (pH8.6). Following application of the serum sample to the strip an operating voltage of 250 volts was applied for 20 minutes.

(10) Sigma Chemical Co.

(11) Shandon Southern Instruments Ltd.

Plates were scanned by a Kipp and Zonen[12] densitometer after staining with Penceau O.S. stain.

Urine Analysis

When possible the urine was collected in clean sterile glass bottles. The specific gravity was measured with a specific gravity refractometer[13]. Tests for urine pH and the presence of blood, glucose, protein and ketones were assessed with bililabstix[14].

The presence of myoglobin was determined using a salt precipitation technique. This depended on myoglobin being soluble in 70% ammonium sulphate whereas haemoglobin was not (Blondheim et al 1957).

STATISTICAL ANALYSES

The mean, standard deviation, standard error and co-efficient of variation for each group of data were calculated by the standard formulae (see appendix P. 85).

Data comparisons between different groups of animals were analysed by Students 't' test and standard methods for the analysis of variance. The method and procedures for these analyses are described in the appendix P. 86.

The probability of significance was taken at the 5% level ($p < 0.05$).

(12) Kipp and Zonen Co. Ltd.

(13) Uricon-Atago Optical Works Co. Ltd.

(14) Ames Co. A Division of Miles Laboratory

RESULTS

NORMAL CLINICAL, HAEMATOLOGICAL AND BIOCHEMICAL PARAMETERS

Clinical

Data for respiratory rate, heart rate and the rectal temperatures in normal resting one year old male and female deer are presented in Table IV.

There were no apparent differences between sex for these clinical parameters.

Haematological

Histograms of the haemoglobin values for 86 males and 25 of the 30 females are shown in Figs. 2 and 3 respectively. Mean and standard deviation values are also shown. Both sex groups showed a normal Gaussian distribution in the values. There were no significant differences between the values for females and males or between the six/nine months old and the 19/14 months old female group.

The values for packed cell volume (PCV) in males and females are shown in Figs 4 and 5. Within the females there was no significant difference between the two age groups according to the analysis of variance but there was according to the Students 't' test: female PCV levels differed significantly ($P < 0.05$) from that of the males.

The total leucocyte counts (Figs. 7 & 8) of the 14 month old females were significantly higher than each of the other two groups, according to the analysis of variance but not with the Students 't' test.

The differential count showed a reversal of neutrophil; lymphocyte ratio, changing from low:high to high:low respectively with increasing age.

TABLE IV

Respiratory rates, heart rates, and rectal temperatures in clinically normal 1 year old red deer

Parameter	Sex	No. of Animals	$\bar{X}$	S.D.	S.E.	C.V.%
Respiratory rate (/min)	M	12	51.5	16.27	4.697	31.59
	F	15	49.2	15.7	4.054	31.9
	All	27	50.2	15.7	3.02	31.3
Heart rate (/min)	M	12	79.5	14.5	4.186	18.24
	F	15	81.2	12.4	3.20	15.27
	All	27	80.44	13.1	5.19	16.28
Rectal Temperature (^{o}C)	M	12	40.13	0.28	0.08	.698
	F	15	40.23	0.67	0.173	1.665
	All	27	40.18	0.53	0.10	1.32

$\bar{X}$ = Mean
S.D. = Standard deviation
S.E. = Standard error
C.V. = Coefficient of variation

Fig 2 : Distribution of haemoglobin levels (g/100ml) in 86 adult males

$\bar{X}$ = 17·15 g/ml.

S.D = 1·5 " "

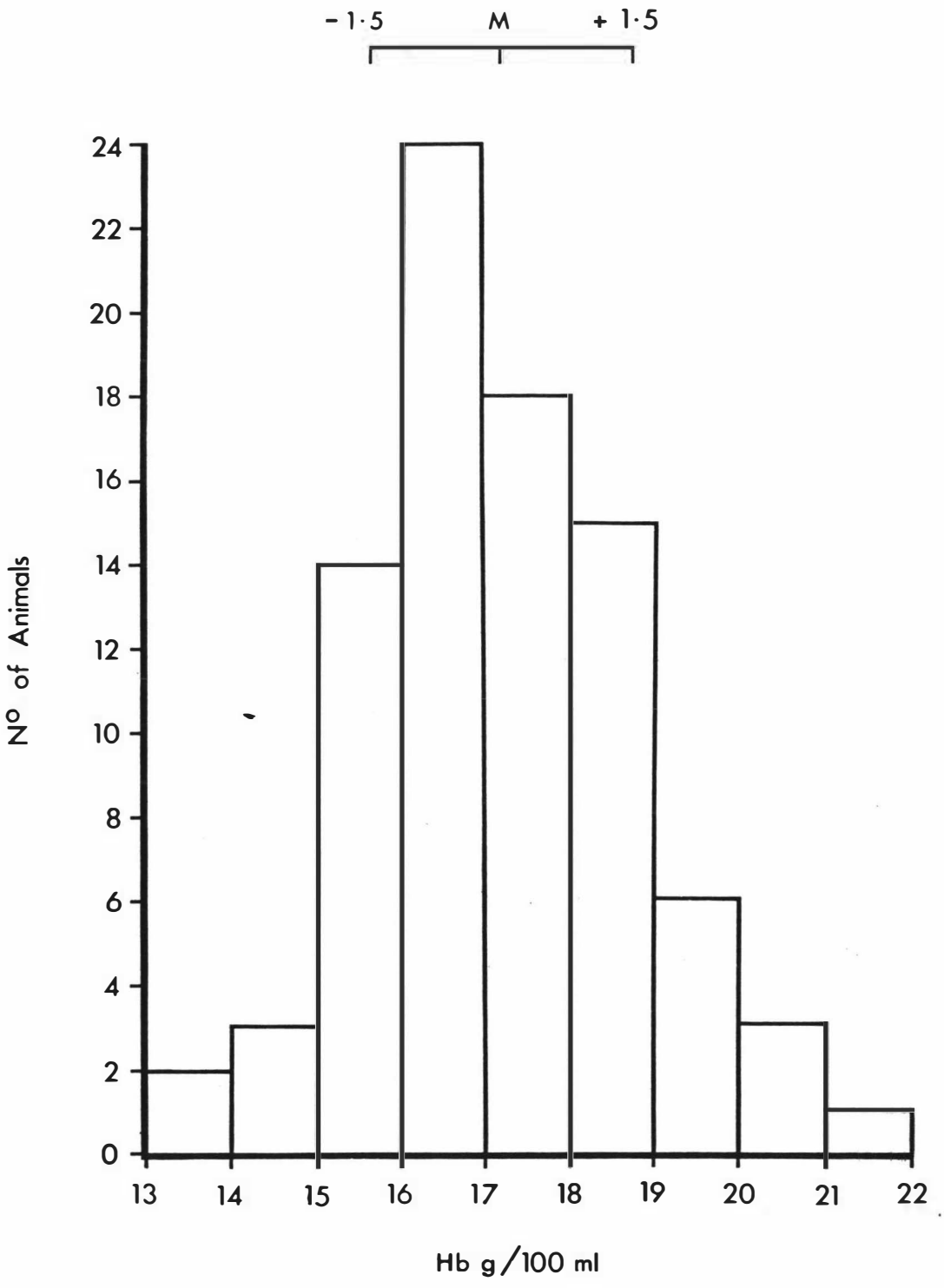


Fig 3 : Distribution of haemoglobin levels (g/100 ml) in 25 females

$\bar{X}$ = 17·03 g/100 ml

S.D = 1·45 " "

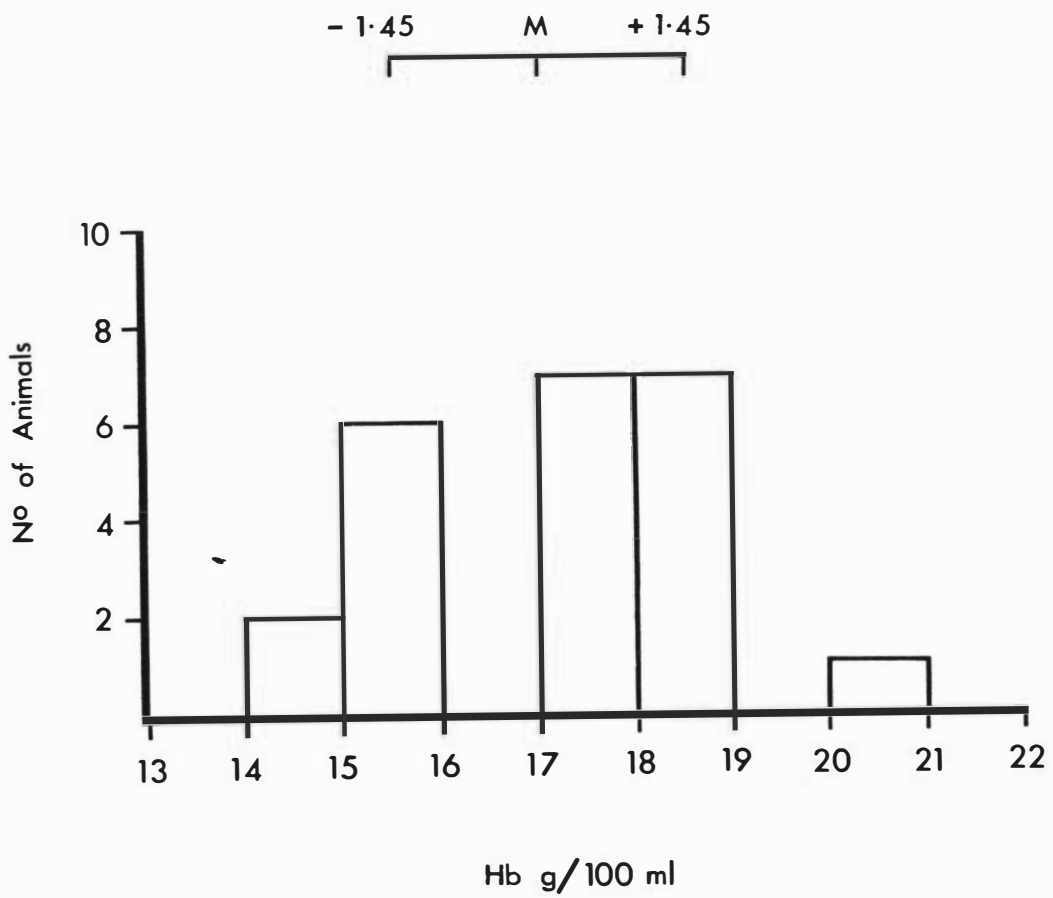


Fig 4 : Distribution of P.C.V. in 86 males

$\bar{X}$ = 0·487

S.D = 0·04

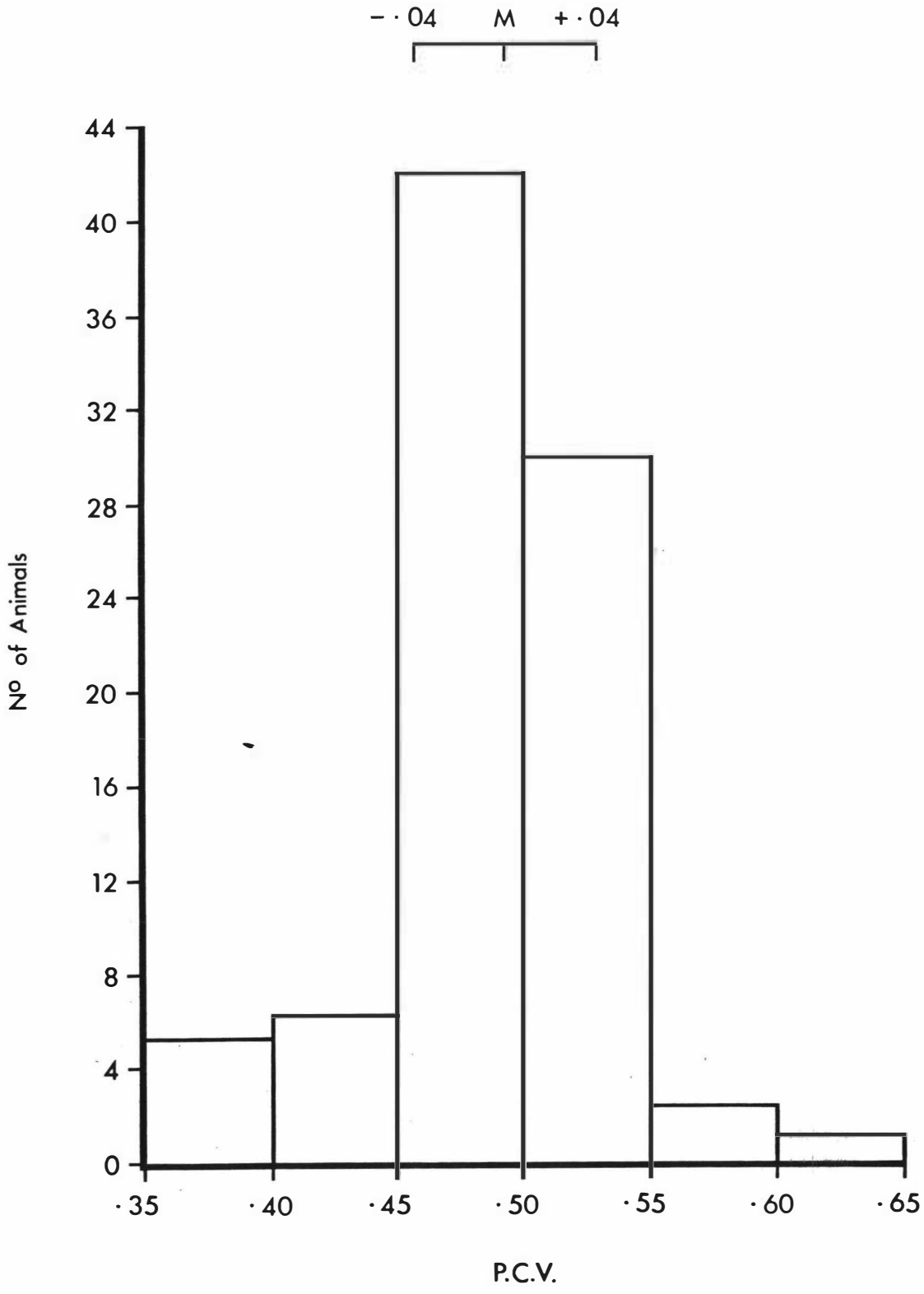


Fig 5 : Distribution of P.C.V. in 24 females

$\bar{X}$ = ·44

S.D = 0·03

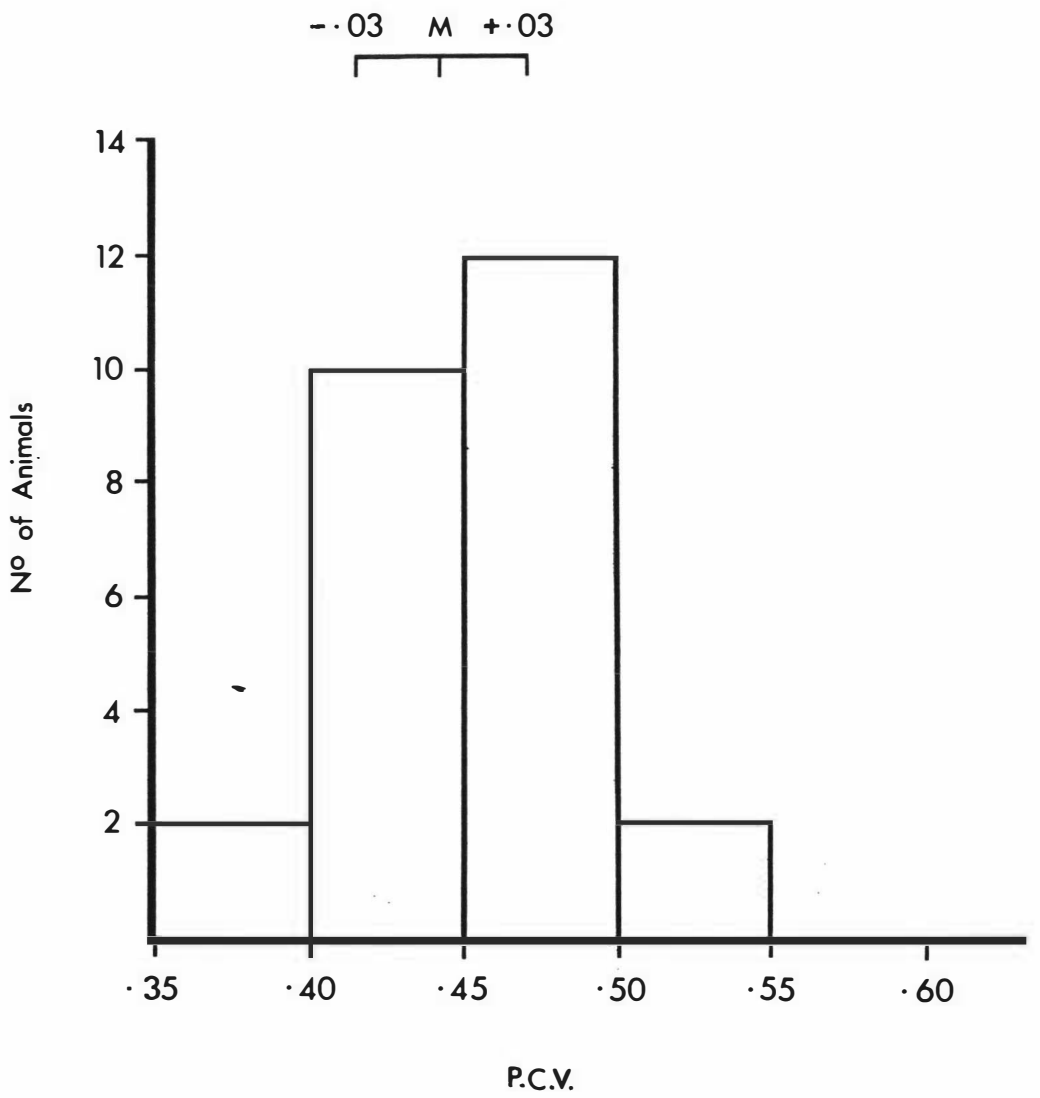


Fig 6 : Distribution of W.C.C. in 27 females

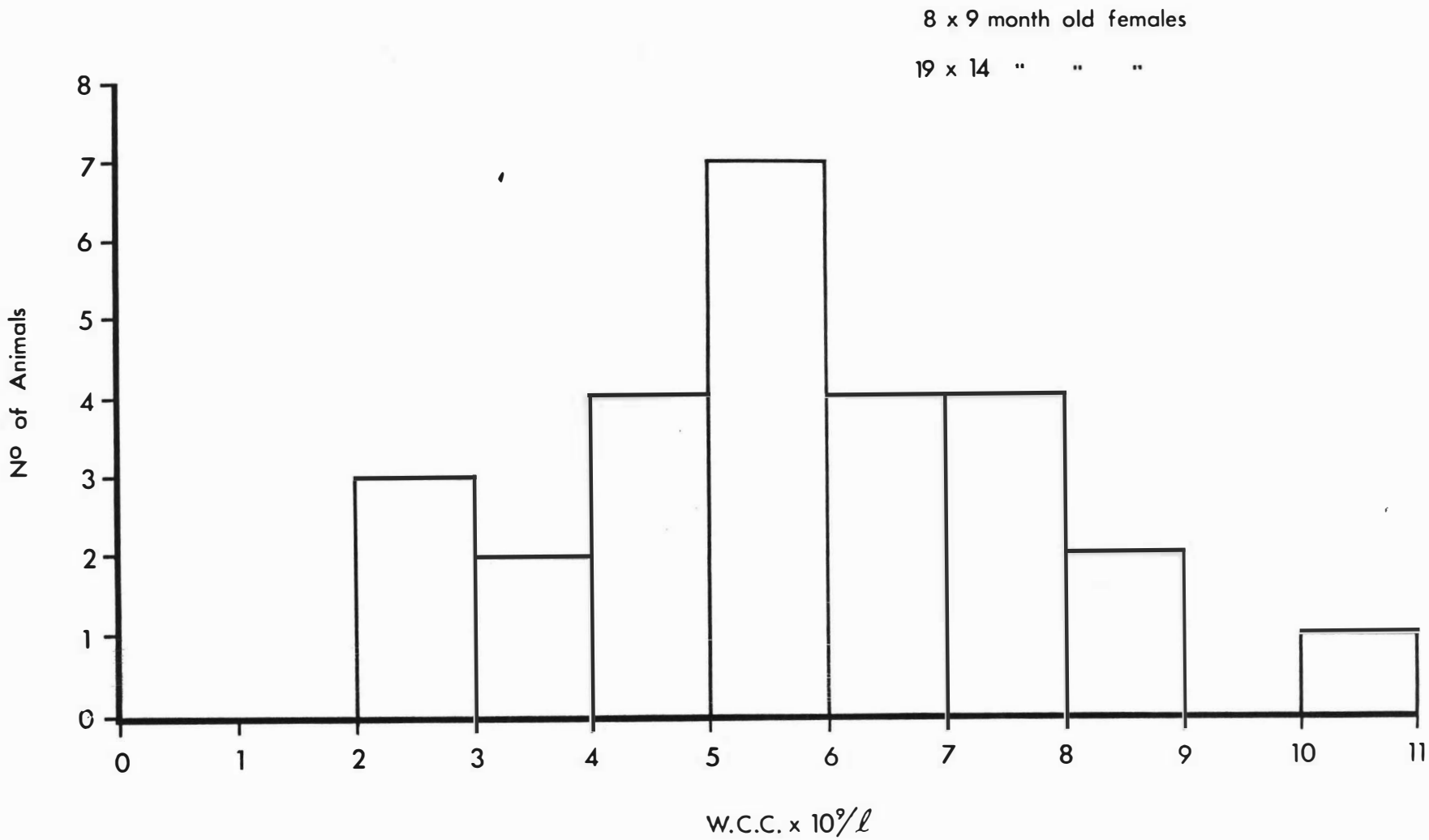


Fig 7 : Distribution of W.C.C. in 19 x 14 month old females

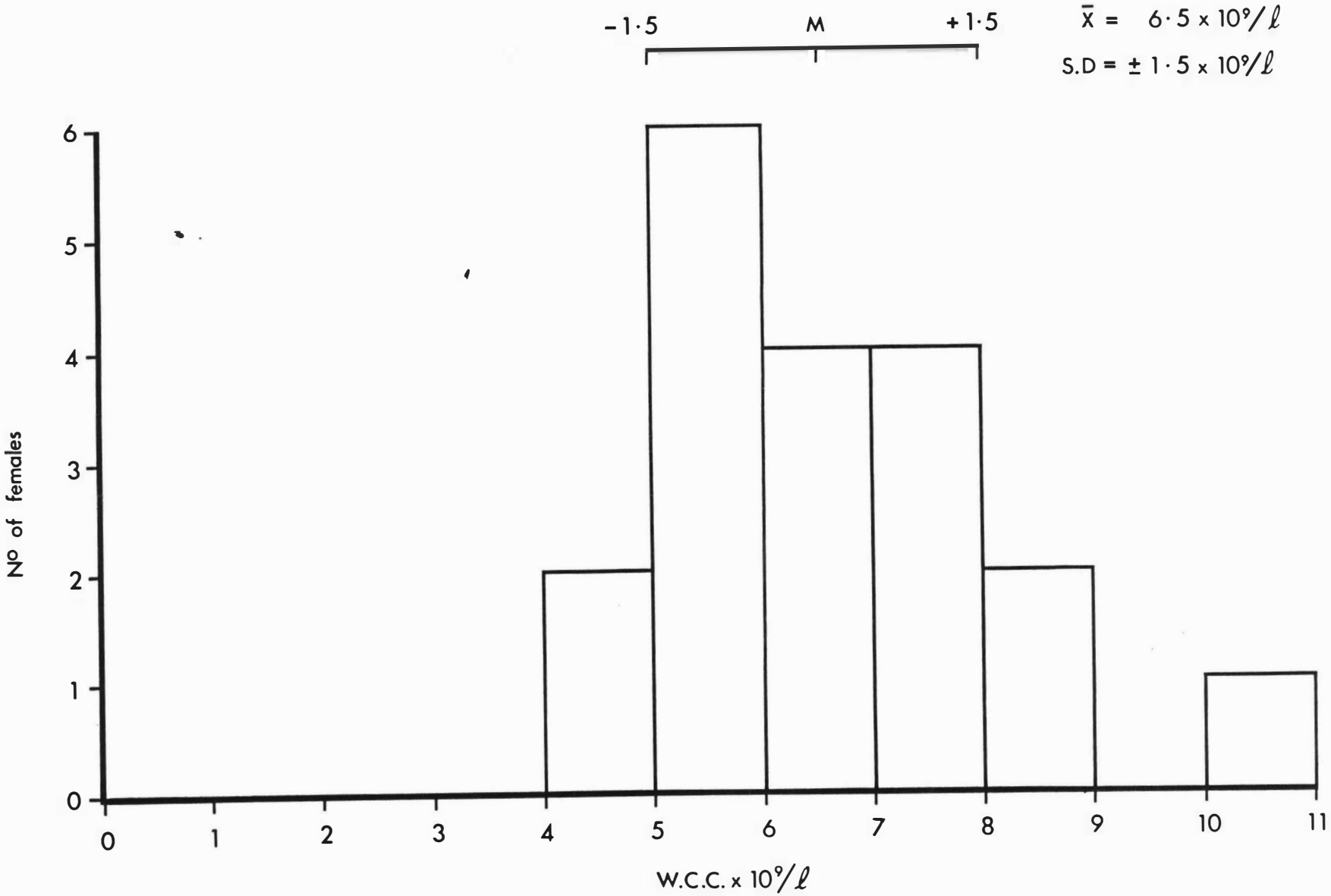
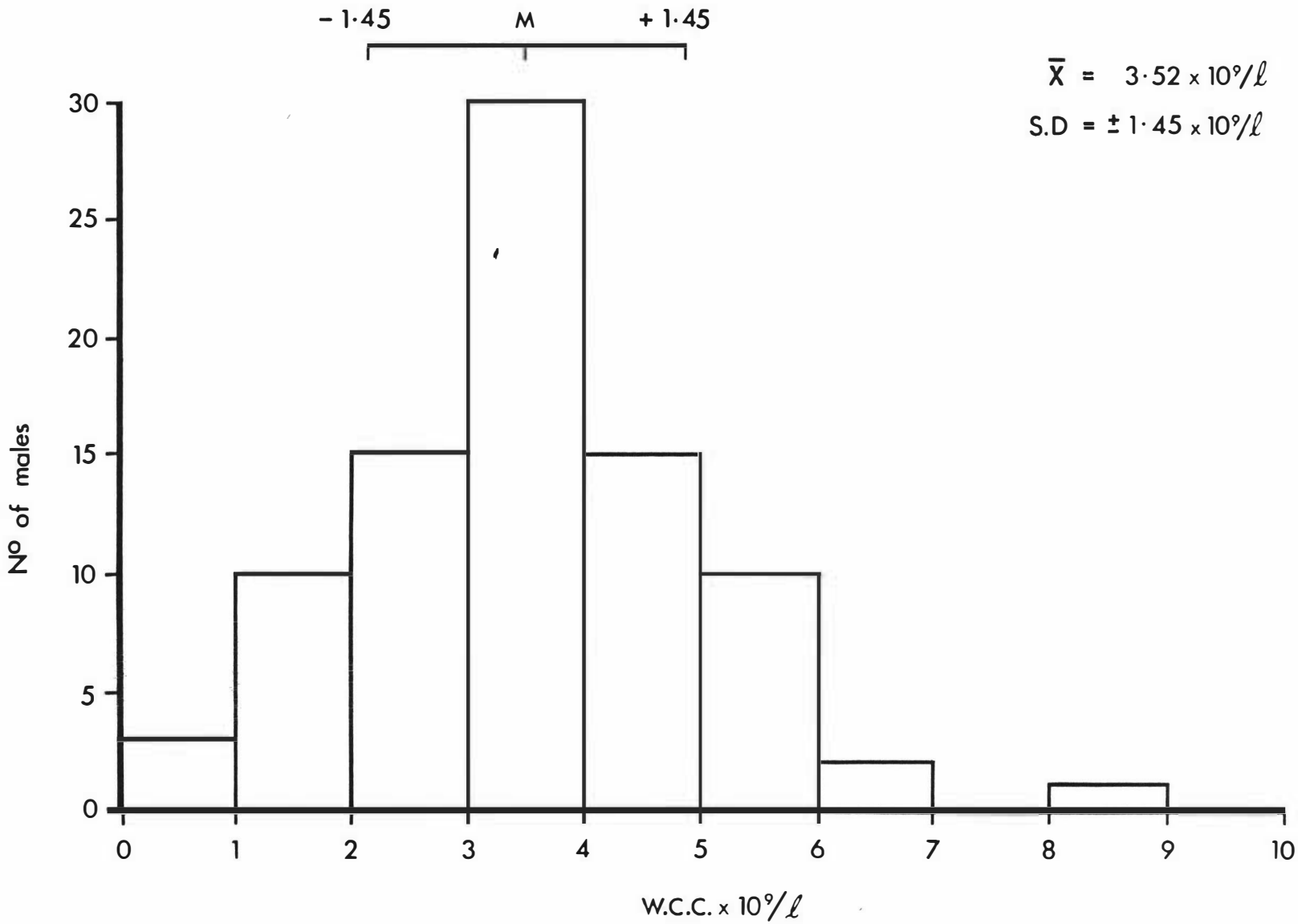


Fig 8 : Distribution of W.C.C. in 86 adult males



Biochemical

The values of the biochemical parameters for normal female and male deer, along with an indication of significance as tested by the analysis of variance between [illegible]

P.36 line 4 correction

With the exception of the pH, albumin, blood urea, calcium and magnesium values,

were present between the different groups for all parameters measured. The most marked differences were found for potassium, sodium, glucose and SGOT values (Table V).

The electrophoretograms for serum proteins were similar for all deer sampled. This was irrespective of age or sex. A representative sample of the pattern obtained is shown in fig. 9.

THE EFFECT OF DRUGS ON HAEMATOLOGICAL AND BIOCHEMICAL PARAMETERS.

The results for the various haematological and biochemical parameters in blood, collected from deer tranquillized with either xylazine hydrochloride* or fentanyl-azaperone** mixture are shown in table VI. The effect of the tranquillizers is to lower the level of many of the parameters. Statistical comparison of data (Table VII) from deer affected with the most commonly used tranquillizer, fentanyl-azaperone, with normal values shows that the differences, except for blood urea, glucose and calcium, were significant ($P < 0.05$).

Blood gas analyses were unable to be measured in the blood samples collected from fentanyl-azaperone tranquillized animals. It was noted, however, that the blood appeared a very dark red in colour suggesting little oxyhaemoglobin was present.

* "Rompun" (Bayer)
*** "Fentaz" (Ethnor)

TABLE V

ormal values for the biochemical parameters in the two female and one male groups

ORMALS	9 m.o.F					14 m.o.F					18 m.o.M				
'arameter	N	$\bar{X}$	S.D.	S.E.	C.V.%		$\bar{X}$	S.D.	S.E.	C.V.%		$\bar{X}$	S.D.	S.E.	C.V.%
›H						33	7,255 a	.097	.017	1.34	25	7,289 a	.057	.011	0.78
?co_2						31	61,38 a	13.62	2.45	22.2	25	50,65 b	7.01	1.4	13.84
Bicarbonate						34	26.03 a	2.44	.42	9.37	25	23.43 b	3.71	.74	15.96
Lactate (mmol/l)						27	1.94	1.043	.2	53.76					
Total serum Proteins g/l	10	58.6 a	6.45	2.04	11.0	19	66.19 b	1.38	.316	2.08	89	66.08 b	5.96	.63	9.02
Albumin	10	34.32 a	6.12	1.93	17.83	19	33,34 a	3.64	.835	10.92	28	33.67 a	3.59	0.68	10.66
Globulin	10	24.28 a	5.63	1.78	23.19	19	32.19 b	4.77	1.094	14.82	28	28.72 a	3.74	0.71	13.02
ratio A/G		1.4/1					1.04/1					1.17/1			
SGOT (S.F.U.)	10	44.37 a	8.76	2.77	19.74	19	56.93 a	16.77	3.85	29.46	90	110.94 b	48.27	5.09	43.5
SGPT (S.F.U.)	10	12.7 b	1.98	.63	15.6	19	19.11 a	4.32	.99	22.6	40	19.7 a	3.54	.56	17.97
Blood urea (mmol/l)	11	11.24 a	1.24	.37	11.03	18	9.93 a	1.04	.25	10.47	90	11.06 a	2.05	.22	18.54
Phosphate (mmol/l)	11	2.45 a	.25	.08	10.2	17	2.43 a	.4	.097	16.46	89	2.08 b	.55	.06	26.4
Glucose (mmol/l	11	9.44 a	2.64	.79	27.97	18	6.99 b	1.12	.26	16.02	88	10.09 a	3.39	.36	33.6
Sodium mmol/l	12	153.25 a	17.36	5.01	11.33	18	130.67 b	8.8	2.07	6.73	90	154.7 a	15.88	1.67	10.26
Potassium mmol/l	11	4.74 a	1.2	.36	25.32	18	3.42 a	.36	.085	10.53	89	9.81 b	5.51	.58	56.17
Calcium (mmol/l)	11	2.22 a	.18	.05	8.11	17	2.21 a	.58	.14	26.24	90	2.22 a	.14	.015	6.3
Magnesium (mmol/l)	11	.75 a	.08	.02	10.67	17	.72 a	.04	.01	5.56	90	.71 a	.08	.01	11.27

N.B. Parameters with the same letter in the same row do not differ significantly with the analysis of variance, parameters with different letters differ significantly ($P < 0.05$)

$\bar{X}$ = Mean S.D. = Standard deviation S.E. = Standard error C.V. = Co-efficient of variation

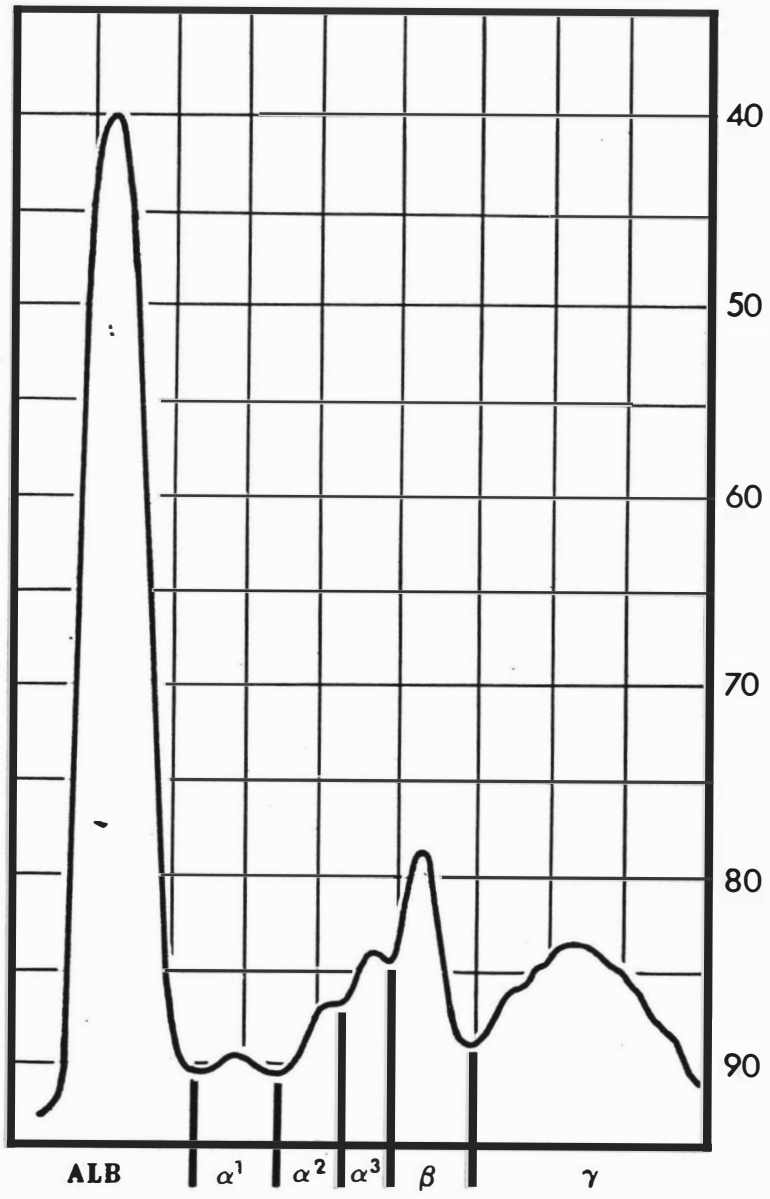


Fig 9 : A representative normal Electrophoretogram

(818)
Total Protein 57.3g/l,
Albumin 35.0g/l. α_1, 1.1g/l;
α_2, 2.3g/l; α_3, 2.3g/l:
β, 5/7g/l; γ, 10.9g/l

TABLE VI

Haematological and biochemical values in blood collected from Red Deer tranquillized with Rompun and Fentaz.

	ROMPUN					FENTAZ				
Parameter	Sex	N	$\bar{X}$	S.D.	S.E.	C.V.%	Sex	N	$\bar{X}$	S.D.	S.E.	C.V.%
Hb	M	8	12.35	1.4	.49	11.3	M	9	12.73	1.04	.347	8.17
P.C.V.		8	.337	.04	1.42	11.87		9	.374	.028	.009	7.49
M.C.H.C.		8	36.71	.697	.25	1.9		9	34.0	1.225	.408	3.6
W.C.C. x 10^9/1		8	5.375	1.99	.7	37.02		9	7.0	1.799	.60	25.7
Neutr "		8	4.290	1.796	.63	41.86		9	4.935	1.6	.53	32.42
Eosin "		8	0.051	0.058	.02	113.72		9	0.258	0.22	.073	85.27
Lymph "		8	0.872	.339	.12	38.88		9	2.085	.872	.291	41.8
Mono "		8	.145	.145	.05	100.0		9	.0845	.064	.021	75.74
pH		8	7.425	.092	.033	1.24						
Pco_2		8	45.25	7.854	2.776	17.35						
Bicarbonate		8	28.98	4.58	1.62	15.8						
T.P.		7	67.3	2.92	1.1	4.34		7	63.73	1.71	0.698	2.68
Albumin		7	33.2	1.69	0.639	5.09		7	31.95	0.82	0.334	2.56
Globulin		7	34.1	2.33	0.88	6.83		6	31.95	1.56	.64	4.88
SGOT		8	100.75	23.18	8.2	23.01		9	77.66	14.27	4.756	18.37
SGPT		8	21.59	4.86	1.72	22.5						
Blood urea		8	10.96	1.2	.42	10.97		9	10.98	1.64	.546	14.94
Phos.		7	2.17	.73	.276	33.6		9	1.655	.37	.123	22.35
Glucose		6	12.93	5.23	2.135	40.44		9	8.092	1.519	.506	18.77
Na^+		8	151.5	13.59	4.8	8.97		7	134.37	6.06	2.29	4.51
K^+		8	2.8	.65	.23	23.21		7	5.568	1.397	.528	25.09
Ca^{++}								7	2.198	.187	.07	8.51

$\bar{X}$ = Mean
S.D. = Standard deviation
S.E. = Standard error
C.V. = Co-efficient of variation

TABLE VII

The statistical comparison of blood parameters between normal males and "Fentaz" affected males using Students "t" test.

Parameter	S^2	"t"	d.f.	Significance 5% level
Hb	2.2325	8.4437	93	S
P.C.V.	0.0016	8.0562	93	S
W.C.C.	2.2	6.696	93	S
SGOT	2154.623	2.05079	97	S
Blood urea	4.0777	0.1133	97	N.S.
Phosphate	0.2887	2.26	96	S
Glucose	10.7186	1.7438	95	N.S.
Sodium	238.567	3.3544	95	S
Potassium	28.5466	2.0227	94	S
Calcium	0.0206	0.3906	95	N.S.

CLINICAL, HAEMATOLOGICAL, BIOCHEMICAL AND PATHOLOGICAL CHANGES IN CAPTURED DEER.

The samples collected from captured deer were divided into two groups; those in which the animals lived (captured data) and those which died (myopathic data).

Affected animals studied varied in age and in size. The myopathic syndrome was found to be the same, irrespective of whether the animals were captured with the use of drugs or not.

Clinical signs

Data for rectal temperatures, heart rate and respiratory rate from the two groups are presented in Table VIII.

The respiratory rates varied from 36 to 160 per minute within thirty minutes of capture for both the captured and the myopathic groups respectively. As shown in Table VIII these returned to normal levels within 24 hours.

The rectal temperatures of both groups showed a rise and in one case in the myopathic group it reached the maximum limit of the thermometer ($43^{o}C$). This animal was very distressed and warm to the touch suggesting a condition similar to malignant hyperthermia. She died within thirty minutes of capture. However, in both groups values rapidly returned to normal (Table VIII).

The heart rate was higher in the myopathic group than in the captured. There appears to be a downward trend but insufficient data does not allow deductions as to when a normal heart rate was reached.

TABLE VIII

Comparison of clinical parameters between captured and myopathic groups of red deer.

Group	Time after Capture	No. of animals	Mean Temperature °C	Mean Respiratory rate/min	Mean Heart rate/min
Captured	½ hr	3	40.8	36	87.33
	1 hr	5	36.3	44.4	48.8
	1½ hr	2	39.7	30	100
	>24 hr	2	39.8	18	66
Myopathic	½ hr	1	43+	160	380
	1 hr	3	38.9	25.5	92
	2½ hr	1	40.5	42	91
	3½ hr	1	39.5	41	80
	>24 hr	5	38.04	22.4	47.6

Although some of the animals were bright in appearance after capture many were very exhausted. A 'wry' neck often developed within 45-60 minutes after capture (fig.10). Some animals showed either knuckling over on the fore or hind legs, dragging a leg or reluctance to move within hours. A partial 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 the acute cases the animals became recumbent after capture and died within a short time.

Urination often occurred without warning soon after capture. The urine was a very dark, almost black, coffee colour (fig.11). Examination revealed the presence of red blood cells, haemoglobin and other debris but the presence of myoglobin could not be proven.

The blood from captured and myopathic animals was frequently found to be very dark. After separation the serum was pink.

Haematological and biochemical

A summary of the haematological and biochemical results for both the captured and myopathic groups is presented in Table IX.

Although very few haematological readings were taken, the myopathic animals had high haemoglobin and PCV levels; this was still apparent at 24 hours post capture.

In addition the total white blood cell count was lowered in the myopathic group.

The main biochemical changes from normal were:-

(a) An initial lowering of blood pH in the myopathic group, whereas the captured group tended to be variable (fig.12). Changes from the normal range were not great, but both groups were higher than normal at 24 to 36 hours after capture.

(b) Lactic acid levels markedly increased within 30 minutes of capture. The levels at this time were approximately four times normal in the captured group and seven times normal in the myopathic group (fig.13). Levels were still 1.5 to 2 times the normal values at 24 and 36 hours after capture.

(c) Pco_2 changes were at a higher level within the first half hour for the myopathic group than for the captured group. The peak for the captured group occurred one hour after capture and was more prolonged than in the myopathic group.

The bicarbonate levels tended to be higher in the myopathic group.

(d) The SGOT levels rose to a very high value in myopathic group (Table IX) compared with the rise in the captured group and the levels in this latter group returned to near normal values after 36 hours.

(e) Potassium levels rose and returned to normal in the captured animals but in the myopathic animals they rose to a higher level and had a slower rate of fall.

(f) The blood urea levels dropped initially in both groups and then rose to high levels in a shorter time in the myopathic group than in the captured

Fig.10. Captured animals showing the 'wry' neck which developed soon after capture.

Fig. 11 Urine from a myopathic animal showing the very dark discolouration.

group.

(g) Glucose levels were shown to drop below normal in both groups over the first few hours after capture but in the captured animals they rose again towards the normal levels at the 36 hour period.

(h) Total protein, phosphate, sodium, calcium and magnesium levels did not vary greatly from the established normal values (Table V and IX).

Examples of the electrophoretograms for serums taken from captured and myopathic animals are presented in figs. 14 and 15 respectively. The "captured" animals showed marked increases in the α_2 and α_3 regions.

Similar changes occurred in the myopathic case at the half hour period. The electrophoretogram of the "myopathy" at one hour shows a great increase in the α_2, α_3 region and the 24 hour electrophoretogram continues this change and shows in addition a high peak between β area and the γ globulins.

Gross Pathology

After reflection of the skin, pale areas were observed in the skeletal musculature on both sides of the body (fig.17). Extensive areas of severe haemorrhage occurred between the skin and fascia. The site of haemorrhage varied from animal to animal and was single or multiple. Haemorrhages as well as pale areas were evident in the skeletal muscles. The muscles affected varied from animal to animal as did the severity of the lesions. Those most frequently affected were Muscularis trapezius. and M. triceps brachii in the

TABLE IX

Summary of parameters for captured and myopathic animals

Time post capture	Captured					Myopathic		
	½ hr. $\bar{X}$	1 hr. $\bar{X}$	1½ hr. $\bar{X}$	3 hr $\bar{X}$	36 hr. $\bar{X}$	½hr $\bar{X}$	1 hr. $\bar{X}$	24 hr. $\bar{X}$
No. of animals	3	3	2	1	2	4	2	5
Haemoglobin					14.5		19.05	19.39
Packed Cell Vol.					42		49	54.2
M.C.H.C.					34.5		39.75	35.89
W.C.C. x 10^9/l					13.1 (1)		1.9	3.935
pH	7.293	7.187	7.275	7.15	7.505	7.17	7.325	7.372
Pco_2	30.03	71.33	45.66	70.38	38.6	81.4	58.25	51.07
B. units								
Bicarbonate		25.43	20.32	24.0	27.75		28.52	34.48
Total protein	65.23	64.3	70.0	71.4	60.95	62.42	72.0	71.48
Albumin								
Globulin								
SGOT (S.F.U.)	233.17	253.5	526.5	729.5	83.35	1424.25	2852.5	7352.4
SGPT (S.F.U.)					12.3			7100.0
Blood urea mmol/l	6.24	6.97	9.35	8.68	12.65	5.58	7.965	15.26
Phosphate "	.84	.51	1.53		1.285	.795	1.585	2.814
Glucose "	8.57	4.12	3.93	4.03	6.3	7.77	5.91	4.728
Sodium "	179.67	186.5	175.2	199.0	152	178	182.8	162.5
Potassium "	9.57	14.43	11.5	8.2	4.14	19.125	20.5	12.06
Calcium "	2.16	2.84	2.04	2.43	1.98	2.365	2.05	1.81
Magnesium "	.96	1.17	1.15	1.6	.745	1.07	1.33	.998
Lactate "	7.86	7.02	4.65	6.03	4.51	13.55	9.5	3.83

(1) Very high as one animal had infected severe wound.

Fig 12 : Graph of pH for captured and myopathic groups

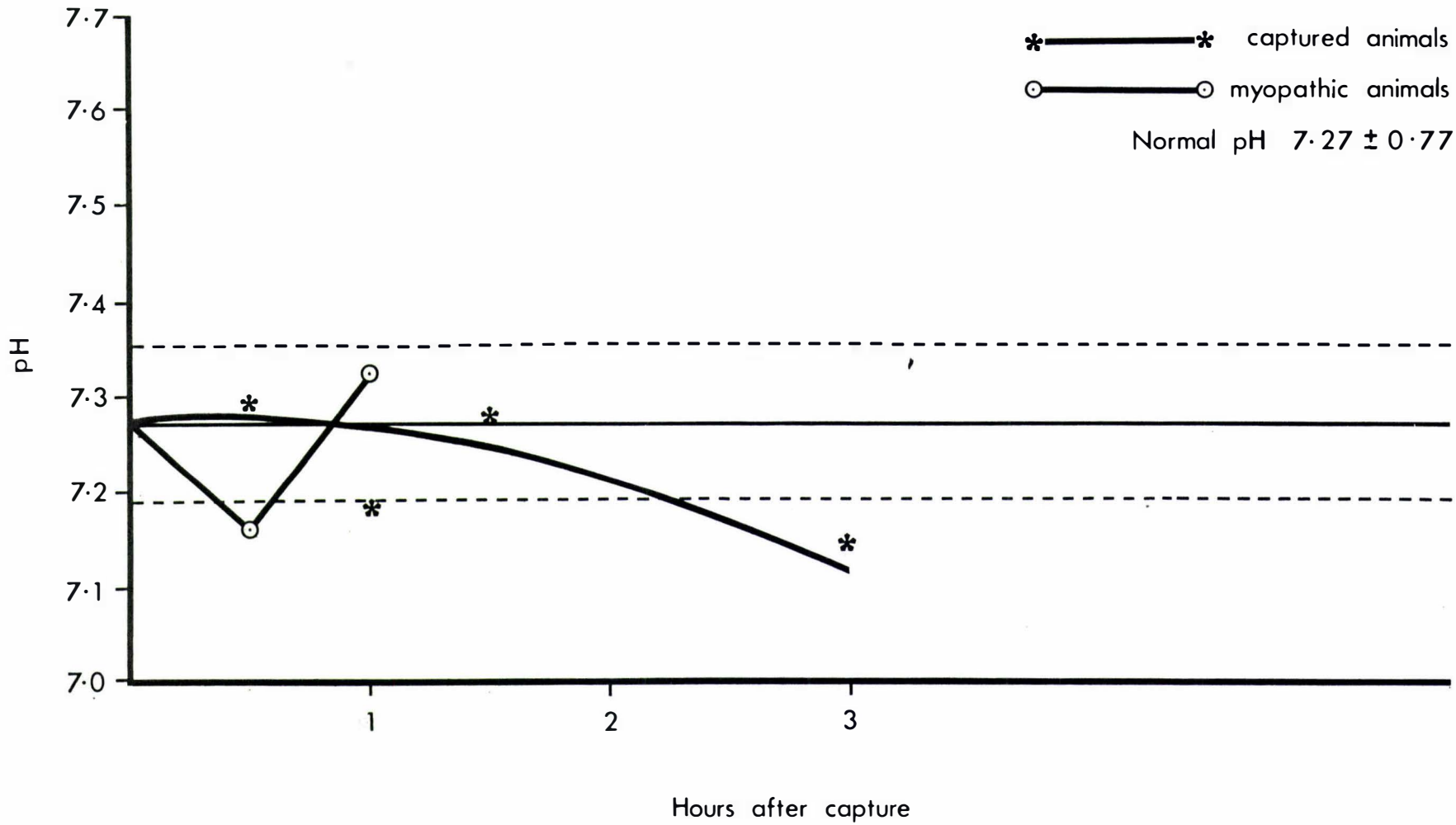
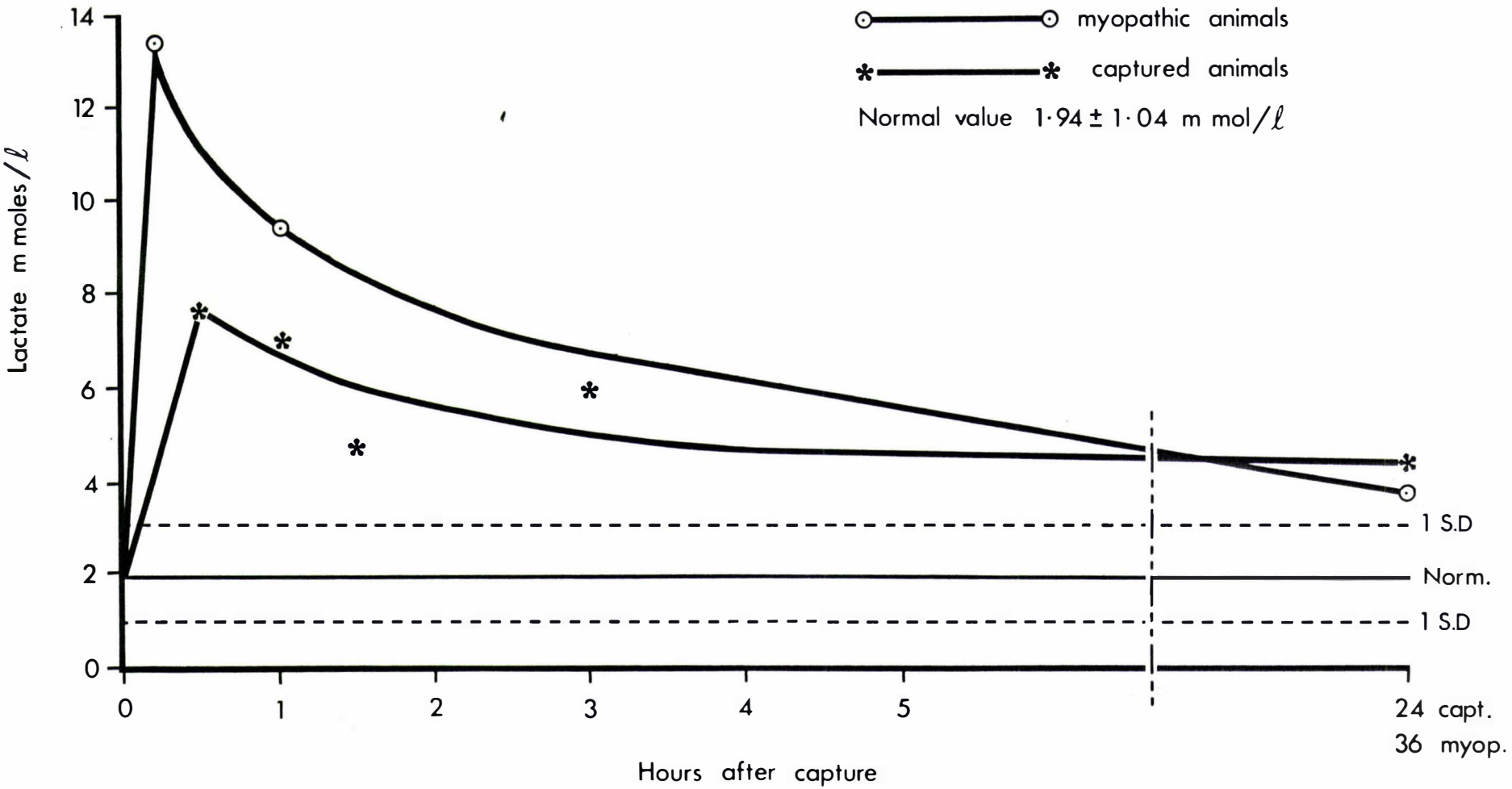


Fig 13 : Graph of Lactate levels (m moles / ℓ) for captured and myopathic groups



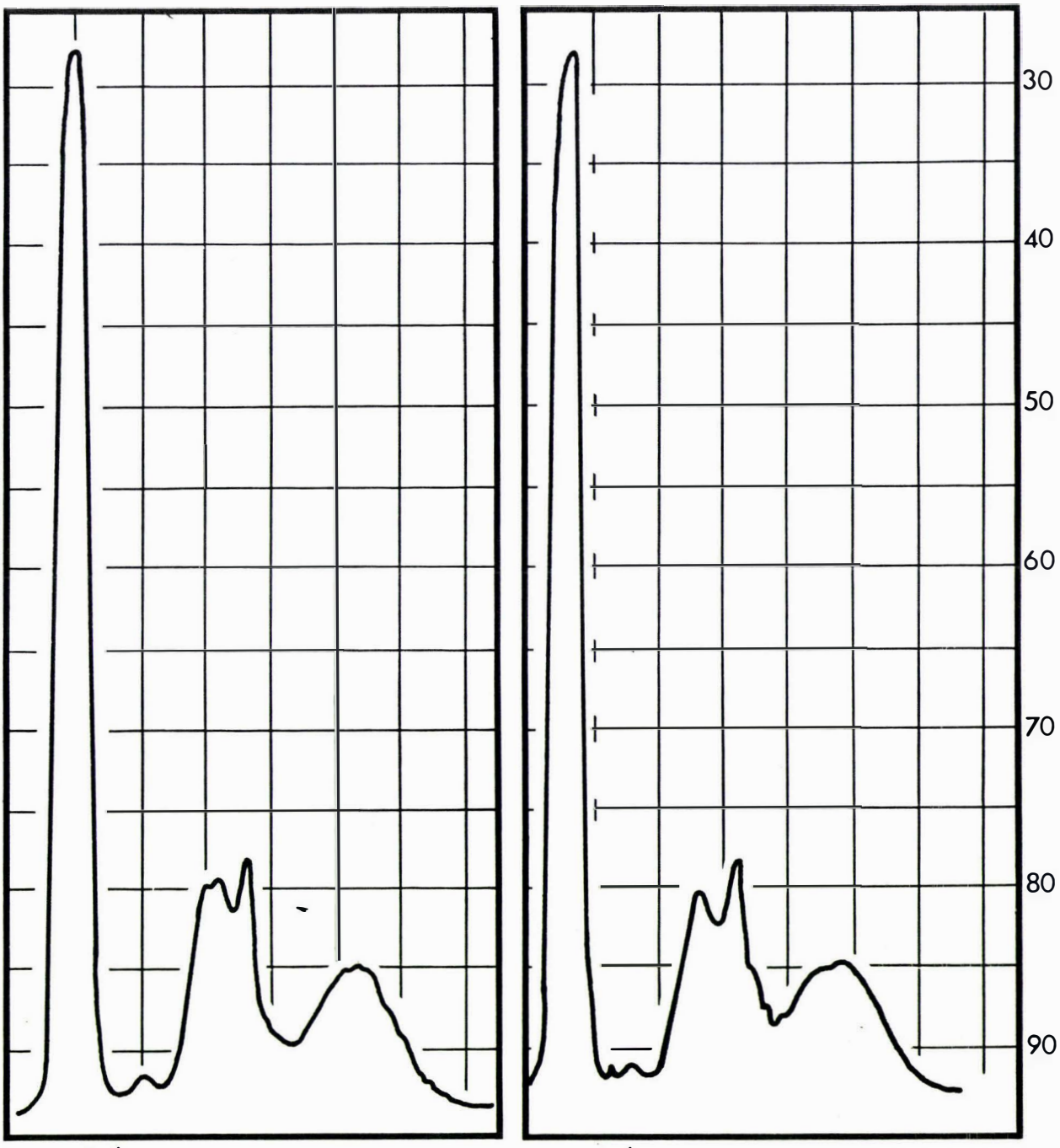
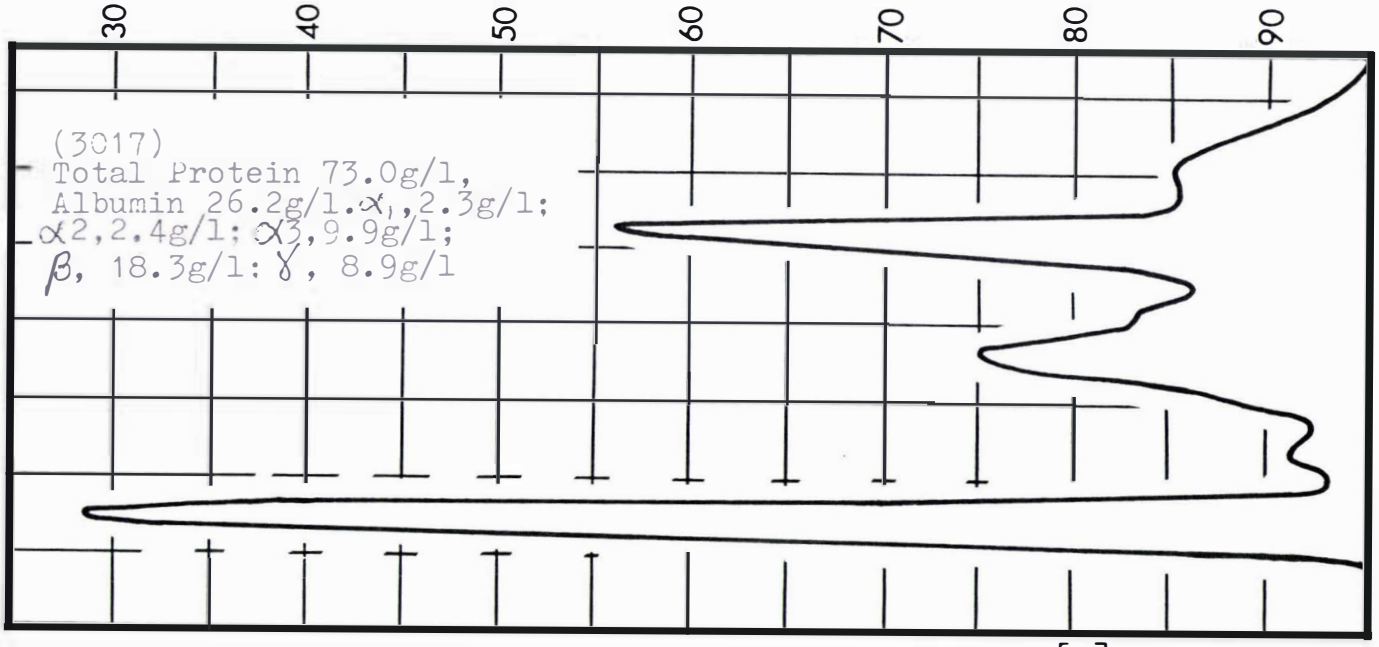


Fig 14 : Examples of Electrophoretogram from "captured" animals

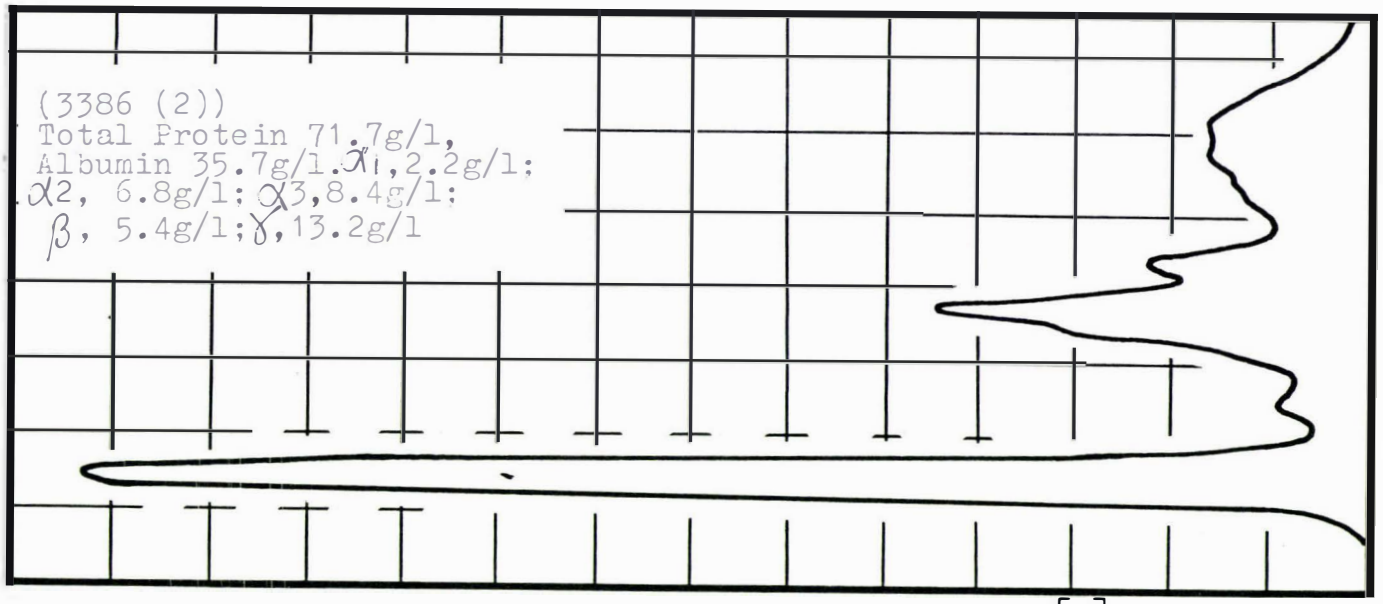
(3388 (3))
Total Protein 63.1g/l,
Albumin 31.9g/l. α_1, 1.1g/l;
α_2, 9.3g/l: β, 8.5g/l;
γ, 12.2 g/l

(3388 (2))
Total Protein 64.0g/l
Albumin 33.5g/l. α_1, 1.1g/l:
α_2, 8.7g/l: β, 7.6g/l:
γ, 13.1g/l.

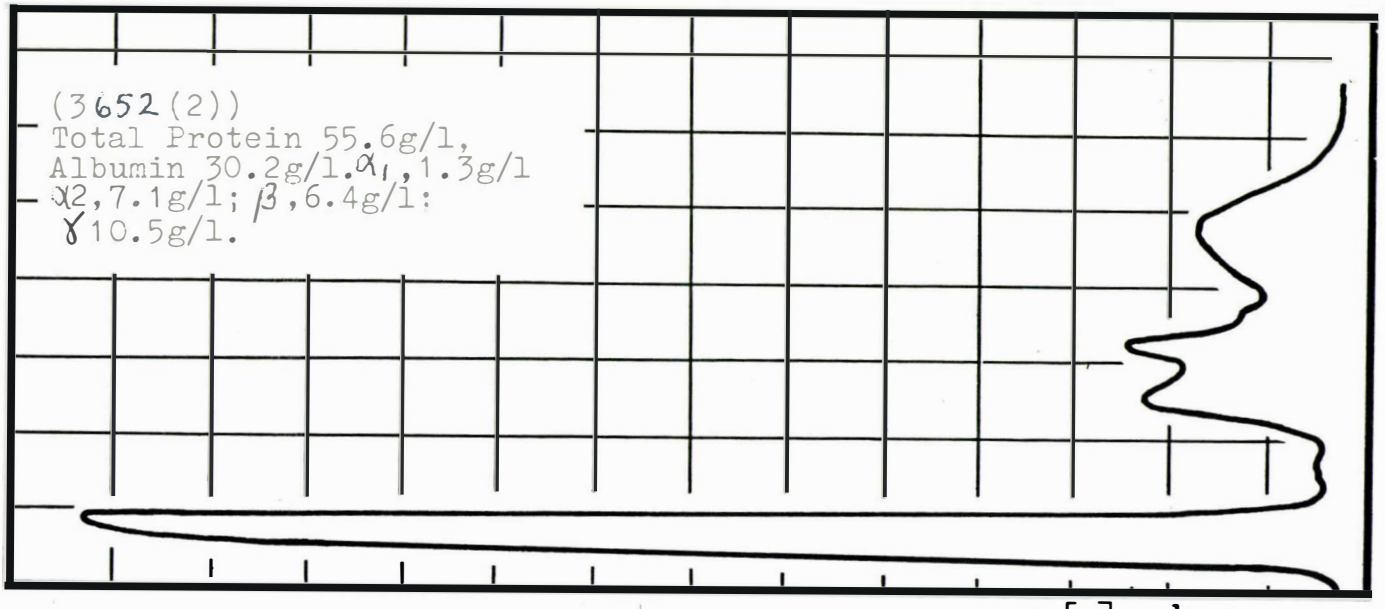
Fig 15 · Examples of Electrophoretograms from "myopathic" animals



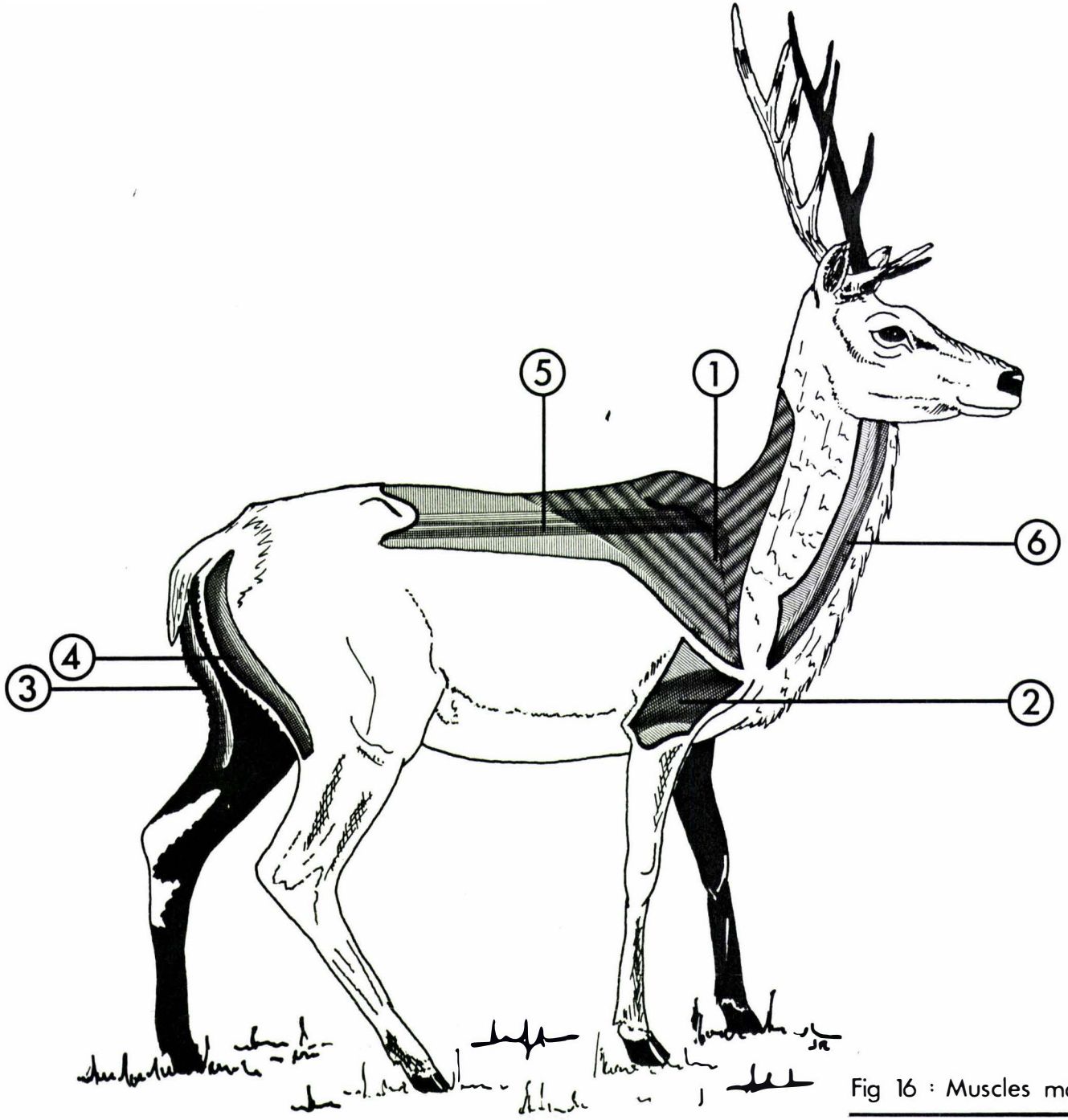
3017 [3] 24 hours



3386 [2] 1 hour



3652 [2] ½ hour



1. Muscularis trapezius
2. M. triceps brachii
3. M. semitendenosis
4. M. semi - membranosis
5. M. longissimus dorsi
6. M. cleidobrachial

Fig 16 : Muscles most frequently affected with gross myonecrosis

fore leg: M. semitendenosis and M. semi-membranosis in the hind leg (fig.16). Often the M. longissimus dorsi was affected but it could only be seen after incising the muscle.

Petechiae were present over the serosa of the intestines on the mensentery and in the ventral cardiac groove.

The lungs appeared oedematous and showed evidence of interstitial emphysema. Large amounts of straw coloured fluid were present within the thorax and often in the pericardium.

It was most noticeable that a very tenacious brown pigmented material covered the necropsy equipment. This had the appearance of haematin.

Histopathology

Representative samples from each myopathic affected animal were examined:

Lung - The 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 eosinophilic 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 they 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, which varied in severity and extent, were commonly observed.

The glomeruli were congested. The juxta-glomerular 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. On staining with Perl's Prussian Blue stain for iron there was a light blue staining reaction in contrast to the more intense blue which may be expected to be haemosiderin (fig.18).

In the severe cases where death was prolonged there was often a severe nephrosis. The proximal tubules were always involved and there was often a fine vacuolation and/or a granular degeneration present 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

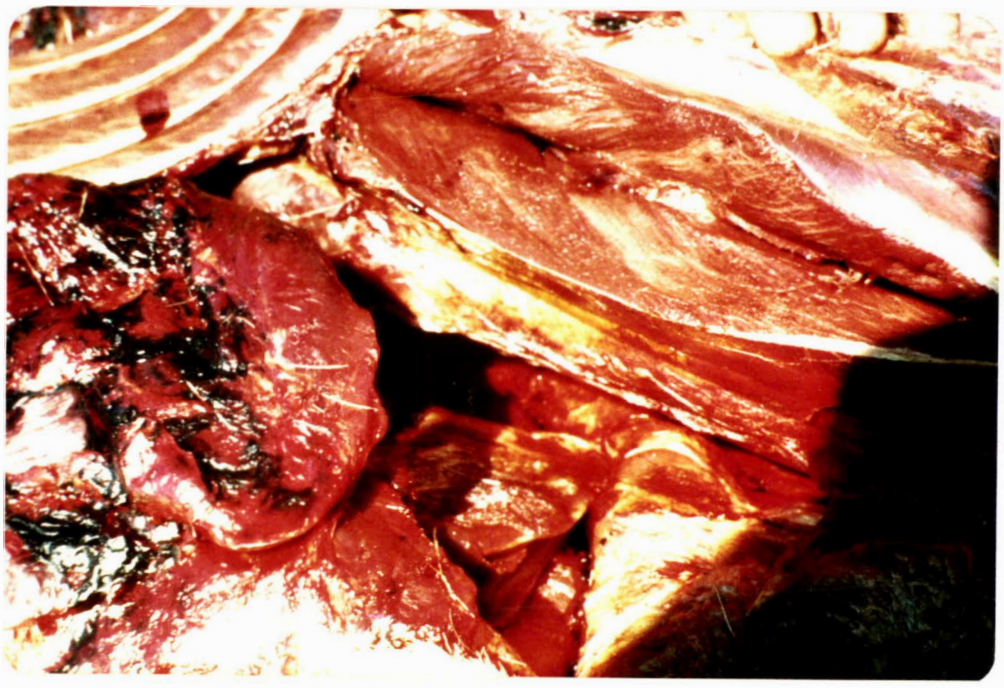


Fig. 17 Pale streaks in muscles at necropsy

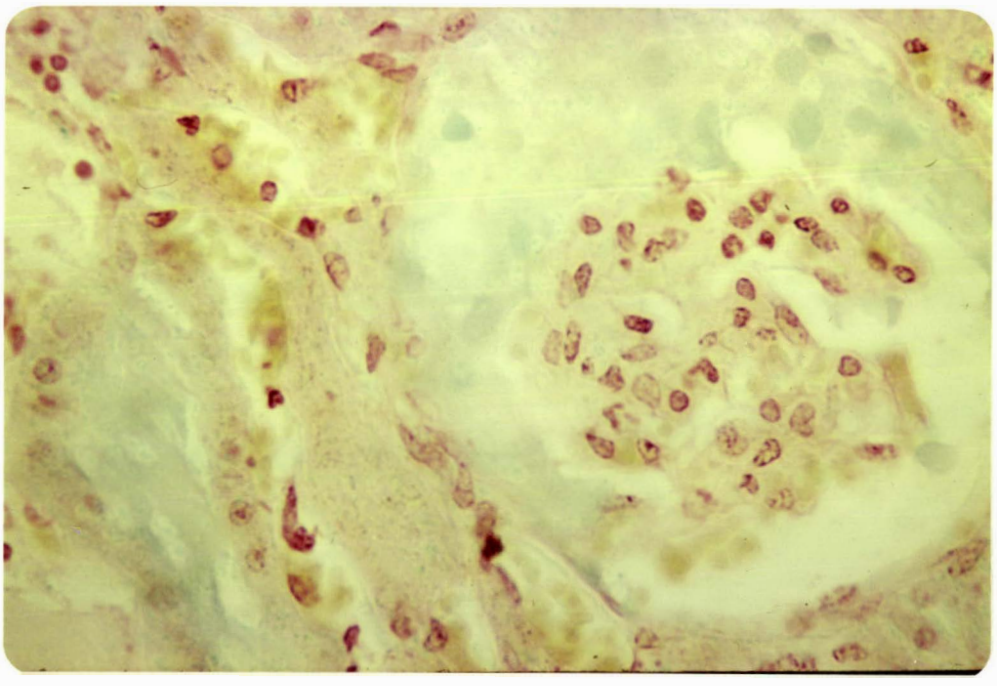


Fig. 18 Kidney Tubules showing pale blue staining globules (Perl's Prussian blue stain, X420)

of having recovered from thyroid hyperplasia; the epithelial cells being cuboidol or columnar and with involution of the walls still present. However, the colloid was well stained and homogeneous. Exhaustion, in a few, was suggested by the presence of macrophages in poorly stained, ragged colloid.

Gastro-intestinal 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 and macrophages containing large numbers of brown-greenish 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 oesinophils.

Spleen - In the three examined all were heavily congested. The germinal centres were small and with a few lymphocytes present.

Brain and cervical cord - No changes were present in the three brains or cervical cords examined. A small number of 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 four 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 outer walls of the ventricles showed similar lesions.

It was noticed in more than one heart of animals with the acute syndrome that the Purkinje fibres appeared ragged at the edges and the cytoplasm depleted and granular.

Skeletal muscle - Bilaterally paired samples were examined from the Muscularis cleidobrachialis, the long and short heads of M. triceps brachii, M. longissimus lumborum, M. semimembranosus (see fig. 16). Sections were taken from other muscles when pale areas were observed.

The lesions could be described as either acute or chronic.

Acute: The fibres appeared distorted, swollen, disrupted, clumped with a loss of striations (fig. 19). The nuclei were swollen and there was separation of the myofibrils. Macrophages were occasionally present. Many of the less affected fibres had a granular degeneration of the cytoplasm. Large numbers of red blood cells were present between the fibres.

Chronic: The sarcolemmal cells were well rounded up and tended to form lines. In places many more macrophages were present. Both regeneration and fibrous tissue were present together (fig.10).

On an anatomical basis it was found that muscles on one side of the neck were often more affected than the other. This was most pronounced in animals with 'wry' neck (fig.10), where muscles on the concave side were less affected than those on the convex side.

The back muscles were less affected than the neck,

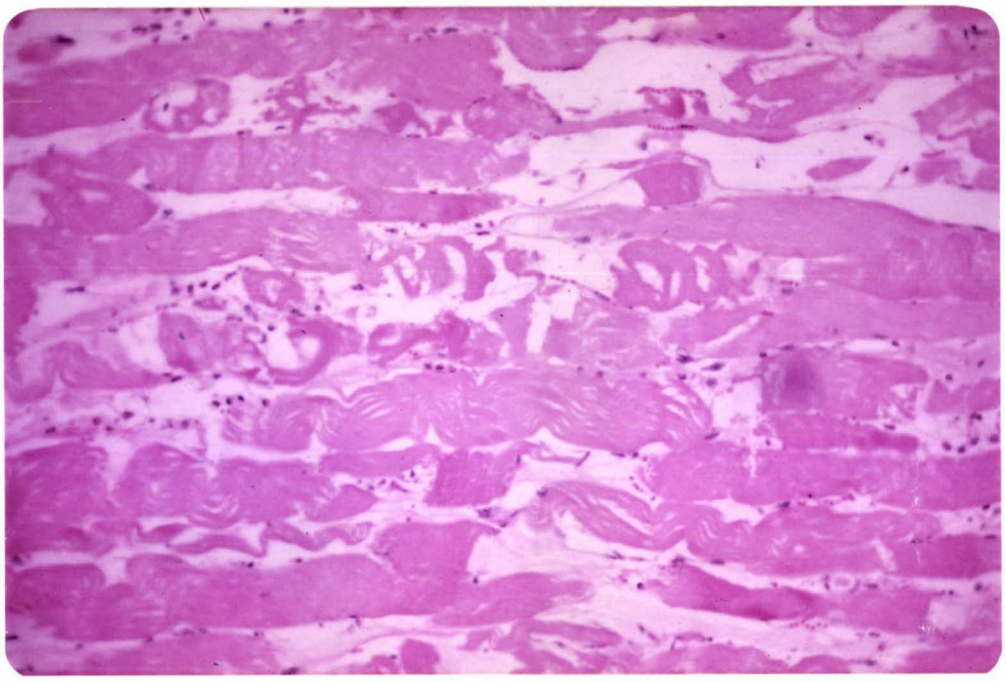


Fig. 19 Acute condition showing disarrayed ragged degenerate fibres and loss of striation.
Haematoxalin Eosin stain, X420

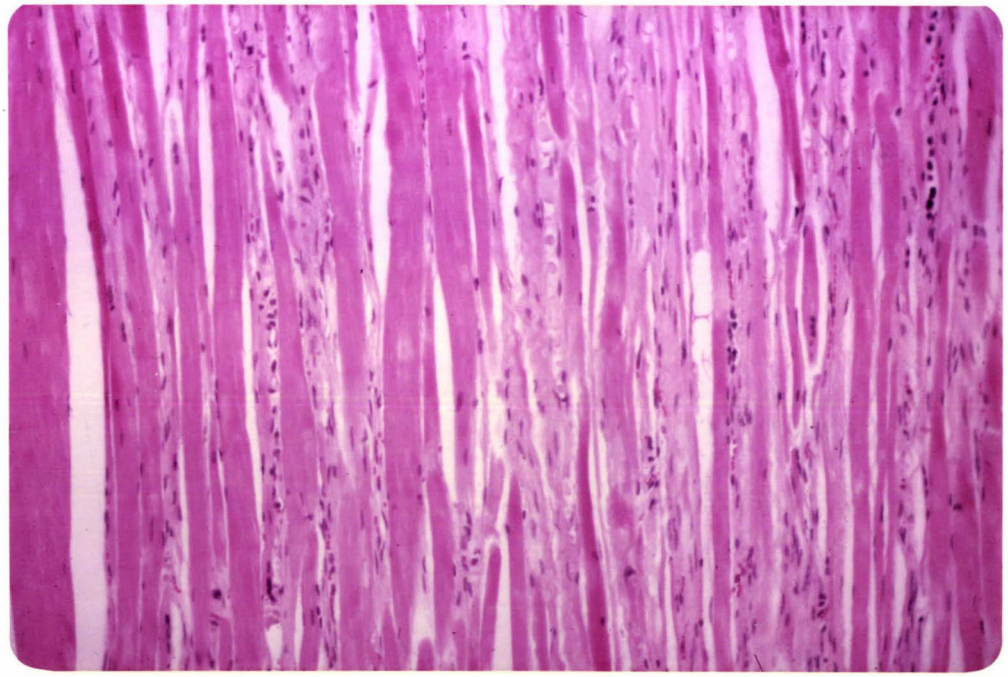


Fig. 20 Chronic condition showing regeneration enlargement of sarcolemmal cells and fibrosis
Haematoxalin Eosin stain, X420

shoulder or leg muscles, severity generally following in that order.

EPIDEMIOLOGY

Study Areas

Animals suffering from the post-capture myopathic syndrome originated, for the purposes of this study, from the areas indicated on the location map (fig.1):

(a) the mountainous country, south west of Mesopotamia Station, Rangitata Valley (August 1975).

(b) the high hill country between the McKenzie Basin and the Hakataramea Valley situated on Haldon Station (September, 1976).

(c) the high hill country between St. Bathans in the north west area of the Maniototo Plain and Omarama at the head of the Waitaki Valley (October, 1976) (fig.21).

The last two areas were used for studying the methods of capture.

Both the areas studied are steep tussock-covered hill country with occasional small pockets of remnant bush. In winter the areas are covered with deep snow. It rapidly disappears in the valleys but remains on the hill tops all winter.

Conditions of capture

The helicopters used were (1) a three seater Hellyer and (2) a two seater Hughes. It was considered by the experienced catching team that the best results were obtained when:

1. temperatures were cool. This minimised the chance of heat exhaustion.
2. there was no wind so that interference with the darting of the animals was minimal.

3. the animals were disturbed as little as possible thereby reducing the problem of fear
4. there were numerous groups of animals
5. the catching team was experienced.

The first three conditions were fulfilled by capturing in the early morning just after dawn or in the late evening. The fourth condition depended on the country and the number of deer present.

The catching teams in this study were experienced having worked together individually for several seasons.

The catching season begins in approximately March and ends in October.

Results

These are summarised in tables X and XI.

Recommendations

The data obtained was insufficient for a full epidemiological evaluation. However from the observations the following recommendations were made:

1. that there be a reduction in the volume of Nalorphine used to 0.25-0.5 ml.
2. that the catching season should be early in the year when animals are in good nutritional state.
3. that the younger, smaller muscled females and males should be preferred.
4. that animals carried beneath the helicopters should be in loose bags totally covering the animals. This not only allows the animal to breathe freely, but also the darkness assists in keeping them quiet.

The difficulty in breathing when travelling at 70 knots or more beneath the helicopter was considered to be one of the most important contributing factors.

Fig. 21 Deer catching country St Bathans. Vehicle in foreground is typical of many of the transporters used for deer.

Fig. 22 Captured deer confined within carrying bags.

5. if darkened transportation trailers cannot be used, the animals should be blindfolded.
6. all captured animals should be placed in a darkened enclosure, provided with food and water, and kept there for two or more days. Releasing them at night, singly, maintains the quiet behaviour.

Any treatment in the form of drenching or ear-tagging should be carried out with the minimum of fuss at this time.

The recommendations 1, 3 and 4 were tested on ten animals, of which nine survived (Table XI). This, however, was too small a number to be statistically significant.

TABLE X

Epidemiological data

	Mesopotamia	Haldon	St. Bathans
No. caught:	77	14	6
No. lame/wry neck: at least	7	5	3
No. dead:	4F 2M	4F 2M	2F
No. caught with drugs:	77	13	6
No. caught without:	-	1	-

Drugs used: capture - All with Fentanyl/Azaperone

strength and vol. used 10 mg/ml/80 mg/ml 2-2½ mls

antidote - Nalorphine

strength and vol. used 10 mg/ml 2-2½ mls.

Site of injection: usually back or hind quarters

Delay before recumbency: 10-15 minutes

Delay before antidote given: approx. 5 minutes after recumbency.

Distance run (21 animals):

short 18 < 2 km These are subjective

long 3 > 2 km estimates by the pilots.

Age: approx. 6 young

15 mature

Nutritional State Mesopotamia All good

Haldon moderately good

St. Bathans moderately good

Transportation from capture site: All in bag beneath helicopter (fig.22).

Transportation to farm: Mesopotamia Helicopter

Haldon darkened trailer

St. Bathans blindfolded in open loosebox

Dark house only used at St. Bathans.

Period in dark house - 2 days

Time of release from darkhouse - singly at night.

TABLE XI

Post Capture losses

Farm	Year	No. caught	No. Dead	%
Mesopotamia Stn.	1975	77	4	5
Papamoa	1975	9	3	30
St. Bathans Stn.	1975	8	2	25
Mt. Peel Stn.	1976	14	7	50
Haldon Stn.	1976	66	(15 (myopathy	22.7
			(5 (other causes	7.5
After changes to catching techniques				
Haldon Stn.	1976	10	1	10

DISCUSSION

Anticoagulant, sampling, container and storage effects.

Anticoagulant

The study of normal haematological data required an anticoagulant which did not distort the leucocytes. The choice lay among fluoride/oxalate, ethylamine-diamine-tetra acetate (EDTA), sodium citrate and heparin.

Sodium citrate (3.8% aqueous solution) is normally used in the proportion of 1 part to 9 parts of blood. The 10% dilution effect was considered undesirable.

The anticoagulant heparin has been found not to alter erythrocyte volume (Pennock and Jones, 1966) but Schmidt *et al* (1953), found it distorted cell morphology and was therefore unsuitable for differential counts.

Anderson (1969) developed a mixture of amonium oxalate, potassium oxalate and ammonium fluoride, which was compared with EDTA by Manston *et al* (1974). They found that the fluoride/oxalate mixture gave consistently lower levels for PCV and although EDTA allowed a slight amount of glycolysis to take place, it was preferable for haemotology.

Disodium, dipotassium and dilithium EDTA anticoagulants were studied by Lewis and Stoddart (1971) who found that concentrations of disodium EDTA and dipotassium EDTA greater than 2 mg/ml blood caused significant increases in PCV and a fall in mean corpuscular haemoglobin concentration.

Below 2 mg/ml blood the change in red blood cell size was of little significance for clinical purposes. The dipotassium salt having the greater solubility of the three at room temperature was therefore preferable.

Container effects

Initiators, catalysts, plasticizers and stabilizers used in the manufacture of plastics may leach out and thereby cause haemolysis, affect complement activity, blood group antibodies and protein activation in coagulation studies (Clark *et al* 1970). Lewis and Stoddart (1971) compared siliconized glass, non siliconized glass and plastic containers and found that the polystyrene (the container commonly used for blood) did not differ from glass for routine blood counts. Both materials had a marked effect on platelet counts and a slight effect on red blood cell counts by comparison with siliconized glass. Other plastics had a notable variance on platelet counts.

Effects of Storage and Time on samples

Blood was collected into EDTA and stored at $4^{o}C$ and room temperature by Gregory and Reid (1976). It was tested at intervals up to 48 hours. Using narrow limits of acceptance, accurate results were obtained up to 24 hours after collection with storage at either $4^{o}C$ or room temperature. Haemoglobin and PCV continued to be tolerably accurate i.e. $\pm$ 2-3% of the original value up to 48 hours. However, the leucocyte count with its accepted limit of 8% up to 24 hours started falling outside the zero time value at 8 hours with the trend widening up to 48 hours.

Manston *et al* (1974) found that tests for glucose in blood and plasma, haematocrit, mean corpuscular volume, erythrocyte count and haemoglobin at 96 hours after zero time gave consistent values when samples were stored at $4^{o}C$. At higher temperatures some haemolysis and glycolysis occurred.

In the present study dipotassium EDTA was the anti-coagulant in blood used for the haemoglobin PCV and differential smear examinations. Heparinized blood was used for pH determination and the plasma for biochemical tests. Coagulated blood serum was used for biochemical autoanalyser tests as the plasma tended to "silt up" the tubing.

Silicone glass and polystyrene containers were used. Platelet counts were not attempted.

All serum samples when separated from the red cells were divided and kept in a chilled Dewar flask. At the laboratory the samples were frozen. Batches of tests were processed on thawed samples. The divided frozen samples prevented continual thawing and freezing, thus minimizing the possible effects of time and storage on the results.

NORMAL PARAMETERS

Haematology and clinical biochemistry

The haematological results obtained in this study are, with some small variations, in agreement with the data obtained by other authors for red deer (Table XII). Part of this variation may be due to the small number of samples used in some studies.

An example of the variation found is the haemoglobin values, which are higher in this study than those obtained by Upcott and Hebert (1965) and Blaxter *et al* (1974) Blaxter *et al* used eleven penned animals bled for about 160 observations.

Other points to note from table XII are the high PCV values obtained, and the total white cell counts

TABLE XII

Comparison of normal haematological parameters of this study and other authors.

	No. of Animals	Hbg/dl	PCV	MCHC g/dl	WCC X109/l	
This study (red deer)	111	17.12 ±1.51	0.477 ±0.043	36.11 ±3.88	4.03 ±1.81	
Upcott & Hebert (1965)	16 <3 y.o.	14.5 ±0.69	0.465 ±0.0046	31.9 ±0.65	2.95	3.44 <3 y.o.
	14 >3 y.o.					2.93 >3 y.o.
Blaxter et al (1974)	11*	16.2	0.449	36	4.4	

*Ca 160 observations

(WCC) which are very low when compared with the normal ranges of other domesticated species (Schalm et al 1975). While the WCC values obtained by Upcott and Hebert (1965) were lower than the value obtained in this study, this may have been due to the 14 months old female group having significantly ($P < .05$) higher counts than the other two groups (Table XIII). These animals were sampled in February and were neither pregnant nor lactating. It is possible that there may have been a steroid influence on the WCC due to the hormonal cycling near the period of mating (April-May).

In addition to a possible effect of age on the total WCC it was noted that the neutrophil: lymphocyte ratio reversed as animals became older. This was noted previously by Upcott and Hebert (1965) and they suggested that it occurred at about 3 years of age.

Another effect of sex noted in this study was the significantly ($P < 0.05$) higher value of the PCV in males (Tables XIV). It is likely that this is due to haemoconcentration as a result of being yarded overnight without water and/or for a tendency of the animals to fight. The roaring season was approaching and this may have been the cause of the friction between the males. This may also be the reason for the higher Pco_2, bicarbonate, GOT and potassium levels in the male animals.

The apparent discrepancy of the analysis of variance with the students 't' test for PCV lies in the sample treatments.

The mean square of each group 0.00031, 0.00087

and 0.00165 for the 9 m.o. females, 14 m.o. females and the males respectively, indicates the variability of treatment in them, and any real difference which may exist between the groups could well be accounted for by random variation. The analysis of variance is accurate for large numbers of samples and the two female groups are composed of small numbers, hence the test is unreliable in detecting the difference between the female groups which are swamped by the large number of samples in the male group.

Serum biochemical parameters for red deer have not been published. Comparisons with data from the white-tailed deer (White and Cook 1974) show that levels of sodium are similar whereas potassium levels in adult female red deer are lower, and in the male deer higher.

Because potassium levels are high in red cells, it is possible that this variation could have occurred with different collecting handling and storage techniques. However phosphate levels which are also at a high concentration in red cells did not show such variation. Significant ($P < 0.05$) differences in sodium and glucose levels between young and adult females may have been the result of nervousness in the young animals with consequent adrenal effect on these two parameters. Total protein differences are normal between young growing animals and adults (Schalm et al 1975). However, the red deer in this study had much higher albumin:globulin ratios than the white-tailed deer in the study of White and Cook (1974). This may be due to the apparently healthy disease status of the farmed red deer with a consequent

low level of circulating globulins.

Effect of tranquillizing drugs

Morgan and Upton (1975) in testing the effect of sampling techniques on acid-base balance in sheep found that the use of "Rompun" led to significantly ($P < 0.05$) higher pH, plasma carbon dioxide and base deficit values and lower haematocrit and haemoglobin values.

In this study both the haemoglobin and haematocrit in normal animals were significantly ($P < 0.05$) depressed by both "Rompun" and "Fentaz". The WCCs were also increased significantly ($P < 0.05$) but the Pco_2 in animals affected with "Rompun" was slightly depressed. Unfortunately for logistic reasons the blood gases were not able to be measured for the "Fentaz" affected animals.

The SGOT in both the drug treated groups remained within the normal range (111 ± 48 S.F.U.) even though some muscle damage was thought to have occurred in the yarding of the animals. No significant difference was found between the two groups for glucose levels.

Although there is no significant difference ($P < .05$) for sodium and potassium between the "Fentaz" affected animals and the normal males, it was considered that the levels of both groups were elevated due to the stress in yarding overnight.

Morgan and Upton (1975) recorded a difference in the calcium and magnesium levels between the veni-puncture and canula sampling. In this study veni-puncture was used and so there was no opportunity to verify this observation.

The method of injecting the stags with the tranquillizer and waiting for the animal to become recumbent before sampling could have caused slight differences in values between animals.

POST CAPTURE MYOPATHY SYNDROME

The clinical and histological evidence presented in this study closely resembles that reported by authors describing the condition in other species.

In studies on the response of animals to exercise it may be expected that there is an increase in heart and respiration rates, the magnitude of which would depend on the work intensity. In horses Lindholm and Saltin (1974) found a linear increase in heart rate related to work intensity with a maximum heart rate of 240/minute. Similarly Harthoorn and Van der Walt (1974) found that Blesbok had heart rates of 250/minute when run over short distances (2 km).

Most of the animals in this study were considered to have run over short distances and they had heart rates generally between 80-90 beats/minute. These were not usually taken directly at the end of the chase. It is, however, interesting to note that the female with an heart rate of approximately 380 beats/min. ran for a very long distance and was in an excitable state from the time she was first seen. Her temperature was also the highest recorded and this was parallel to the findings of the Blesbok. Those that ran over the furthest distances had the highest temperature (Harthoorn and Van der Walt 1974).

The changes in the biochemical parameters studied have obviously been the result of the stress before capture. Some of the variations observed were probably

influenced by the variation in the speed and duration of the chase.

The rise in lactic acid levels indicated that a high level of anaerobic glycolysis occurred and was still persistent at 36 hours after capture. Although the number of animals studied was small, it was observed that the myopathic animals reached a higher level which peaked earlier than the level in captured animals. High levels of lactate after exertion have been recorded in man (Osnes and Hermansen 1972), in pigs (Jørgensen and Hyldgaard-Jensen 1975) and in horses (Lindholm and Saltin 1974). The time for lactate levels to return to normal was thought to relate to the fitness of the animal. The continuing high levels (at 36 hours in this study) may mean that the deer were unfit or that there was a slow release of lactate from the muscles.

Glucose levels were initially similar to those of the "Fentaz" affected animals with a decline well below normal. This indicated that it was being utilized over the first few hours with the levels being inversely related to those of lactic acid. These two findings together signify that anaerobic glycolysis was occurring.

Lactate influences the hydrogen ion concentrations of the blood, (Henry et al) so that the pH levels normally fall when acid 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.

The alkalosis could have been due to fear instilled by the sound of the helicopter causing an increased adrenaline secretion and giving rise to a respiratory alkalosis.

The Pco_2 levels were low soon after capture in captured animals, slowly rising to the normal range. However, the myopathic animals had an higher initial Pco_2 becoming stabilized in the normal range within the hour. There does not appear to be any correlation between the rise in lactic acid levels and the changes in Pco_2 as found by Milne (1974) in horses. However, this may have been due in part to the temperature of the animals body, particularly after a run.

The serum glutamic oxalo acetic - transaminase (SGOT) released from damaged muscle rose significantly. The myopathic group had much higher levels than the captured group;- these levels were still rising at 24 hours, which would suggest that an ongoing necrosis was present.

Potassium rose to levels 2½ times that of the "Fentaz" affected animals one hour after capture in the 'captured' group and then fell to within the normal range within 36 hours after capture. The peak is before the SGOT peak and this may be due to it being a smaller more mobile ion and therefore more easily released from damaged cells. Also GOT has a mitochondrial fraction which would not be released until cell necrosis had occurred.

In the myopathic group a potassium level nearly

four times that of the normal range was reached. These high levels, as for example the individual levels of 28 mmol/l reached in the first half hour after capture, probably contributed to the sudden deaths. This would be due to the extracellular fluid potassium rising and water passively entering the cells moving into the mitochondria and endoplasmic reticulum causing cell swelling and degeneration. Other probable causes of increased potassium were:-

(1) an efflux of K^+ ions from intracellular stores in exchange for H^+ ions during a metabolic acidosis.

(2) a diffusion of K^+ ions from the intracellular space when muscle and liver glycogen is reduced.

(3) release from red cells as a result of intravascular haemolysis (Bergström et al 1971. Gilligan et al 1943 Kjellmer 1965).

At 24 hours the levels were decreasing. This was probably due to the effect of aldosterone control. This probable demand for aldosterone may be the reason for the high number of adrenal cortical cells showing eosinophilic degeneration or necrosis.

Blood urea levels in both groups were initially below those of the normal animals and of the "Fentaz" affected animals. However, the increased levels in both captured and myopathic groups indicated, particularly in the myopathic group, that tubular damage was occurring. 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 lead to kidney failure, uraemia and eventual death.

The calcium levels of both groups rose slightly. 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). However, it was slight and transient.

The serum inorganic phosphate levels were initially depressed; more than can be attributed to the effect of "Fentaz". However, they recovered within 1-1½ hours. Adrenaline and muscular work could have caused a lowering of the inorganic phosphate (Codazza et al 1974).

PATHOGENESIS

A schematic outline on the possible pathogenesis of the post capture myopathy syndrome is given in fig. 23.

The histological lesions in muscle taken from myopathic animals were the response of the tissue to one or more insults. They closely resembled the lesions found in the selenium-vitamin E deficiency condition of sheep and cattle (Jubb and Kennedy 1970); the "tying-up" syndrome of draught horses (Azoturia) (Carlström 1931, 1932) and the porcine stress syndrome of Landrace and Pietrain pigs (Ludvigsen 1953, Briskey et al 1959 (a).

Although it is possible that the triggering mechanism in each case may differ, the similarity of the lesions suggests that early on there is a common pathway. This is probably derangement of mitochondrial function (Lindholm et al 1974).

Carlstrom (1931, 1932) postulated that a carbohydrate-rich diet, in conjunction with a few days of rest, resulted in an increased glycogen storage, leading to the production of an abnormally large amount of lactic acid as a result

of subsequent exercise which in turn resulted in the swelling of the affected muscle fibres (Lindholm et al 1974).

It has been found that the muscle fibres involved in the acute stage of the 'tying-up' syndrome of horses were richer in glycogen than in the later stages suggesting that there had been an increased glycogen storage when compared with normal healthy horses (Lindholm et al 1974). Animals with lesser muscle mass may reduce the resultant lactic acidosis (Osnes and Hermansen 1972).

One of the major effects of adrenaline is to activate glycogen breakdown to form glucose (Lewis 1965) elevating the levels in both blood stream and muscle. The glucose is then used for energy. Pyruvic acid is the normal product but if insufficient oxygen is available, anaerobic glycolysis occurs leading to high levels of lactic acid. The reduced nicotinamide-adenine-dinucleotide (NADH) uses pyruvate to convert back to NAD and forms lactic acid. Consequently high levels of lactic acid accumulate in the blood and muscle tissue. The levels in the blood do not necessarily reflect the levels in the muscles these being higher.

As the lactic acid level rises the bicarbonate (the principle buffering system) level reduces the pH changes. This gives rise to the metabolic acidosis which is partly compensated for by increased speed and depth of respiration to reduce the partial pressure of carbon dioxide. The observations of myopathic deer would support that this has occurred.

Two possible contributing factors to the syndrome are:-

(1) the reduction in depth and rate of respiration by 'Fentaz' drug and its antidote nalorphine.

(2) the continued stimulation and secretion of adrenaline producing further lactic acid from glucose.

The first contributing factor can be partially corrected by reducing the dose of the various drugs and ensuring that a good oxygen supply is present.

The second factor can be reduced by eliminating or reducing the stimuli either by drugs or utilizing the quietening effect of darkness.

It has been found that the levels of adrenaline in the porcine stress syndrome continue at high levels (Topel et al 1974), pointing to either a continued high secretion or an inability to inactivate the adrenaline. The levels of adrenaline in the red deer have not been determined.

Muscle metabolism has been extensively studied in relation to physical activity and fibre composition in laboratory animals (Blanchauer and Van Wijhe 1962; Romanul 1964 a b; Karpati and Eugel 1967), cats (Romanul and Van der Meulen 1966; Dubowitz and Newman 1967), birds (Wilson, Cahn and Kaplan 1963) horses (Lindholm and Piehl 1974), pigs (Topel et al 1968; Jørgensen and Hyldgaard-Jensen 1975) and cattle (Keller and Stanbridge 1973, Keller 1974).

Three different types of muscle fibre have been recognised by the various authors. The first are the fast twitch white fibres with a high activity of myosin A.T. Pase, a well developed glycolytic enzyme system and a low mitochondrial content and oxidative activity;

these fibres fatigue readily (Close, 1972; Lindholm and Piehl 1974). They correspond to the W fibres of Ashmore and Doerr (1971) and the group I fibres of Romanul (1964b). The second are the fast twitch red fibres with a high activity of myosin A.T. Pase, a moderately developed glycolytic system, a high total oxidative enzyme activity and many mitochondria; these fibres show less fatigue than the fast twitch white fibres (Close 1972). They correspond to the R fibres of Ashmore and Doerr (1971) and the group II fibres of Romanul (1964b). The third are the slow-twitch intermediate fibres with a slow activity of myosin A.T. Pase and glycolytic enzymes, a high mitochondrial content and oxidative enzyme activities: these fibres show little or no fatigue (Close 1972) and correspond to the R fibres of Ashmore and Doerr (1971) and the group III fibres of Romanul (1964b).

Some myopathies are known to affect primarily one fibre type and produce characteristic histological patterns. The selective involvement of one fibre type provides a clue as to which metabolic pathway may be involved. This has been used by Hamilton et al (1974) in the study of inherited muscular dystrophy in mink, by Ruth and Van Vleet (1974), in the study of selenium/vitamin E deficiency in swine and by Lindholm et al (1974) in the study of acute rhabdomyolysis in standard-bred horses. Lindholm et al (1974) considered the damaged fibres were of the fast twitch variety suggesting that they become susceptible to damage after the aerobic respiration changes to anaerobic respiration.

Ruth and Van Vleet (1974) found that the fast twitch fibres (their type I) were selectively destroyed and that

there was a lack of phosphorylase activity in the fast twitch white fibres (their type II).

Determination of the various muscle types in the red deer and which type is destroyed in post-capture myopathy have as yet to be done and may help elucidate the metabolic pathway.

Fig. 23 POST CAPTURE MYOPATHY PATHOGENESIS

APPENDICES

TABLE XIII

Normal haematological values

Parameter	Sex	Age	No. of Animals	$\bar{X}$	S.D.	S.E.	C.V.%
Haemoglobin g/100 mls	F	9 m.o.	6	16.39	1.56	0.64	9.52
	F	14 m.o.	19	17.23	1.39	0.32	8.07
	F	9 m.o. + 14 m.o.	25	17.028	1.449	0.29	8.51
	M	< 18 m.o.	86	17.15	1.53	0.16	8.92
	All		111	17.12	1.51	0.1433	8.82
Packed cell volume	F	9 m.o.	6	.42	.017	.007	4.05
	F	14 m.o.	18	.45	.0295	.007	6.55
	F	9 m.o. + 14 m.o.	24	.44	.0298	.006	6.77
	M	< 18 m.o.	86	.487	.041	.004	8.42
	All		110	.477	.043	.004	9.01
Mean Corpuscular Haemoglobin concentration	F	9 m.o.	6	38.77	2.71	1.11	6.99
	F	14 m.o.	18	38.56	1.34	.32	3.47
	F	9 m.o. + 14 m.o.	24	38.61	1.687	.34	4.37
	M	< 18 m.o.	86	35.43	4.06	.44	11.46
	All	110	110	36.11	3.88	.37	10.74
White cell count $X10^9/l$	F	9 m.o.	8	3.5	.89	.31	25.43
	F	14 m.o.	19	6.5	1.5	.34	23.08
	F	9 m.o. + 14 m.o.	27	5.62	1.94	.37	34.5
	M	< 18 m.o.	86	3.52	1.45	.16	41.2
	All		113	4.03	1.81	.17	44.91
Neutrophils $X10^9/l$ (absolute) (Nos.)	F	9 m.o.	8	1.469	.627	.22	42.9
	F	14 m.o.	19	2.723	1.26	.29	46.27
	F	9 m.o. + 14 m.o.	27	2.35	1.245	.24	52.98
	M	< 18 m.o.	85	2.50	1.12	.12	44.8

$\bar{X}$ = Mean S.E. = Standard error
S.D. = Standard Deviation C.V. = Co-efficient of variation

TABLE XIII (cont.)

Parameter	Sex	Age	No. of Animals	$\bar{X}$	S.D.	S.E.	C.V.%
Eosinophils $X10^9/l$	F	9 m.o.	8	0.361	0.755	.27	209
	F	14 m.o.	19	0.714	0.417	.096	58.4
	M	18 m.o.	85	0.326	0.051	.006	15.6
Lymphocytes $X10^9/l$	F	9 m.o.	8	1.821	0.505	.18	27.7
	F	14 m.o.	19	2.623	0.628	.14	23.9
	F	9 m.o. + 14 m.o.	27	2.386	0.694	.13	29.1
	M	18 m.o.	85	0.878	0.583	.06	66.4
Monocytes	F	9 m.o.	8	0.096	0.07	.02	72.9
	F	14 m.o.	19	0.202	0.11	.03	54.5
	M	18 m.o.	85	0.083	0.067	.01	80.7

$\bar{X}$ = Mean
S.D. = Standard deviation
S.E. = Standard error
C.V. = Co-efficient of variation

TABLE XIV

The statistical comparison of haematological parameters using Students "t" test. (Spiegel 1961)

A. Between 9 months old and 14 months old normal females

Parameter	S^2	"t"	d.f.	Significance 5% level
Hb.	2.0411	1.2555	23	N.S.
P.C.V.	0.00078	2.3424	22	S
W.C.C.	1.8418	5.2449	25	S

B. Between 14 months old normal females and adult normal males.

Hb	2.2694	0.2095	103	N.S.
P.C.V.	0.001545	3.6317	102	S
W.C.C.	2.1283	8.0507	103	S

TABLE XV

CAPTURED ANIMALS

Analyses of blood taken within first half hour of capture.

Animal No.	3652 (2)	4332 (2)	4332 (1)	$\bar{X}$ (3)
Temperature ^{o}C	39.6	41.5	41.3	40.8
Respiration rate/min	36	28	44	36
Heart rate/min	82	84	96	87.33
Haemoglobin g/100 mls.	12.9			
Packed cell Vol.X10^{3}	.38			
MCHC	34			
WCC x 10^{9}/l	4.3			
pH	7.18	7.2	7.5	7.293
Pco_2	38.0	35.45	16.64	30.03
Base excess	-14.0	-13.5		
Bicarbonate	13.5	13.3	5.1	
Total protein g/l	55.6	70.9	69.2	65.23
SGOT (S.F.U.)	191.5	323.5	184.5	233.17
SGPT (S.F.U.)	-	-	-	-
Blood urea mmol/l	2.5	9.3	6.92	6.24
Phosphate mmol/l	0.87	0.86	0.79	0.84
Glucose mmol/l	8.76	6.2	10.76	8.57
Sodium mmol/l	174.3	179.6	185.1	179.67
Potassium mmol/l	9.2	12.0	7.5	9.57
Calcium mmol/l	2.1	2.18	2.2	2.16
Magnesium mmol/l	0.82	1.02	1.04	0.96
Lactate mmol/l	9.21	2.95	11.43	7.86

$\bar{X}$ = Mean

TABLE XVI

CAPTURED ANIMALS

Analyses of blood taken within one hour of capture

Animal No.	3388(1)	3388(2)	3388(3)	3388(4)	3388(5)	4332(2)	$\bar{X}$
Temperature	36.4	36.1	36.2	36.4	36.2	40	36.88
Respiration rate	30	32	22	78	60	24	41
Heart rate	24	36	34	84	66	54	49.67
pH	7.2	7.15	7.2			7.2	7.187
Pco_2	68.5	80	65.5				71.33
Base excess							
Bicarbonate	24.4	26.4	25.5				25.43
Total protein g/l	65.8	64.0	63.1				64.3
SGOT (S.F.U.)	278.5	215.0	267.0				253.5
SGPT (S.F.U.)							
Blood urea mmol/l	6.8	7.05	7.05				6.97
Phosphate mmol/l	.52	.51	.49				0.51
Glucose mmol/l	4.99	4.33	3.03				4.12
Sodium mmol/l	191.1	186.5	181.9				186.5
Potassium mmol/l	12.3	15.4	15.6				14.43
Calcium mmol/l	2.66	2.52	3.35				2.84
Magnesium mmol/l	1.12	1.0	1.39				1.17
Lactate mmol/l	7.16	7.33	6.58				7.02

TABLE XVII

CAPTURED ANIMALS

Analyses of blood taken within 1½ hours of capture

Animal No.	4332(2)	4332(1)	$\bar{X}$
Temperature ^{o}C	39.8	40.5	40.15
Respiration rate/min	30	42	36
Heart rate/min	48	91	69.5
pH	7.3	7.25	7.275
Pco_2	53.06	38.26	45.66
Base excess		-10	
Bicarbonate	25.5	15.15	20.32
Total protein g/l	70.0		70.0
SGOT (S.F.U)	526.5		526.5
SGPT (S.F.U)			
Blood urea mmol/l	9.35		9.35
Phosphate mmol/l	1.53		1.53
Glucose mmol/l	3.93		3.93
Sodium mmol/l	175.2		175.2
Potassium mmol/l	11.5		11.5
Calcium mmol/l	2.04		2.04
Magnesium mmol/l	1.15		1.15
Lactate mmol/l	2.45	6.85	4.65

$\bar{X}$ = Mean

TABLE XVIII

CAPTURED ANIMALS

Analyses of blood taken within 3 hours of capture

Animal No.	4332(1)	$\bar{X}$
Temperature ^{o}C	39.5	39.5
Respiration rate/min	41	41
Heart rate/min	80	80
pH	7.15	7.15
Pco_2	70.38	70.38
Bicarbonate	24.0	24
Total protein g/l	71.4	71.4
SGOT (S.F.U.)	729.5	729.5
SGPT (S.F.U.)	-	-
Blood urea mmol/l	8.68	8.68
Phosphate mmol/l	-	-
Glucose mmol/l	4.03	4.03
Sodium mmol/l	199.0	199.0
Potassium mmol/l	8.2	8.2
Calcium mmol/l	2.43	2.43
Magnesium mmol/l	1.6	1.6
Lactate mmol/l	6.03	6.03

$\bar{X}$ = Mean

TABLE XIX

CAPTURED ANIMALS

Analyses of blood taken after 36 hours after capture

Animal No.	3656 (406)	3656 (413)	$\bar{X}$
Temperature ^{o}C	39.9	39.7	39.8
Respiration rate/min	18	18	18
Heart rate/min	68	64	66
Haemoglobin	14.3	14.7	14.5
Packed cell Vol.	42	42	42
MCHC	34	35	34.5
WCC x 10^{9}/l	9.7	16.5(infected)	13.1
pH	7.41	7.6	7.505
Pco_2	51.6	25.6	38.6
Base excess		+ 5.5	
Bicarbonate	31.9	23.6	27.75
Total protein g/l	67.8	54.1	60.95
SGOT (S.F.U.)	85.6	81.1	83.35
SGPT (S.F.U.)	12.6	12.0	12.3
Blood urea mmol/l	13.8	11.5	12.65
Phosphate mmol/l	1.55	1.02	1.285
Glucose mmol/l	7.9	4.7	6.3
Sodium mmol/l	151	153	152
Potassium mmol/l	3.45	4.83	4.14
Calcium mmol/l	2.29	1.67	1.98
Magnesium mmol/l	.72	.77	.745
Lactate mmol/l	4.0	5.02	4.51

$\bar{X}$ = Mean

TABLE XX

MYOPATHIC ANIMALS

Analyses of blood taken within half hour of capture

Animal No.	3385	3652	4332(3)	4332(4)	$\bar{X}$
Temperature °C	41	>43.0	39.8	39.5	40.825
Respiration rate/min	78	160	36	24	74.5
Heart rate/min	84	ca.380	168	84	179
Haemoglobin g/100 ml	-	15	-	-	
Packed cell Vol.	-	46.5	-	-	
MCHC	-	32.2	-	-	
WCC x 10^9/l	-	.5	-	-	
pH	7.25	7.05	7.22	7.15	7.17
Pco_2	78.4	93.2	72	82	81.4
Base excess	-	-	-	-	
Bicarbonate	-	-	28.2	-	
Total protein	68.4	53.3	60.8	67.2	62.42
SGOT (S.F.U.)	378	249	1520	3550	1424.25
SGPT (S.F.U.)					
Blood urea mmol/l	9.26	4.33	3.1	5.63	5.58
Phosphate mmol/l	.76	1.48	.48	.46	.795
Glucose mmol/l	13.1	5.77	7.59	4.63	7.77
Sodium mmol/l	184.6	180.5	152.5	194.4	178
Potassium mmol/l	28.2	13.8	6.4	28.1	19.125
Calcium mmol/l	2.39	2.25	2.31	2.51	2.365
Magnesium mmol/l	1.2	0.89	.95	1.25	1.07
Lactate mmol/l	12.3	21.89	8.87	11.16	13.55
Urine pH	8.0	-			
blood	Small	-			
glucose	-	-			
S.G.	1.038	-			

$\bar{X}$ = Mean

TABLE XXI

MYOPATHIC ANIMALS

Analyses of blood taken within one hour of capture

Animal No.	3386(1)	3386(2)	$\bar{X}$
Temperature ^{o}C	38.3	40	39.15
Respiration rate/min	24	18	21
Heart rate/min	60	56	58
Haemoglobin g/100 ml	20.1	18.0	19.05
Packed cell Vol.	53	45	49
MCHC	39.5	40	39.75
WCC x 10^{9}/l	2.4	1.4	1.9
pH	7.4	7.25	7.325
Pco_2	43.5	73	58.25
Base excess			
Bicarbonate	26.25	30.8	28.52
Total protein g/l	72.3	71.7	72
SGOT (S.F.U.)	2495	3210	2852.5
SGPT (S.F.U.)			
Blood urea mmol/l	7.21	8.72	7.965
Phosphate mmol/l	.77	2.4	1.585
Glucose mmol/l	3.93	7.89	5.91
Sodium mmol/l	177.8	187.8	182.8
Potassium mmol/l	20.8	20.2	20.5
Calcium	2.33	1.77	2.05
Magnesium mmol/l	1.32	1.34	1.33
Lactate mmol/l	8.74	10.26	9.5

$\bar{X}$ = Mean

TABLE XXII

MYOPATHIC ANIMALS

Analyses of blood taken more than 24 hours after capture.

Animal No.	3017(1)	3017(2)	3387	4073(1)	4073(2)	$\bar{X}$
Temperature	35.4	38.4	40	39.8	36.6	38.04
Respiration rate	12	19	56	13	12	22.4
Heart rate	38	40	64	48	48	47.6
Haemoglobin	18.85	18.4	20	19.8	19.9	19.39
Packed cell Vol.	52	53.0	58	54	54	54.2
MCHC	36.25	34.7	34.5	37	37	35.89
WCC x 10^9/l	4.125	6.15	2.2	3.65	3.55	3.935
pH	7.4	7.4	7.25	7.39	7.42	7.372
Pco_2	49.75	60.0	75.8	36.1	33.7	51.07
Base excess						
Bicarbonate	46.78	50.9	31.9	21.4	21.4	34.48
Total protein g/l	73.6	73.00	68.4	70.7	71.7	71.48
SGOT (S.F.U)	7345	7650	8855	937	11,975	7354.4
SGPT (S.F.U)				7100		7100.0
Blood urea mmol/l	11.6	12.0	10.2	20.5	22.0	15.26
Phosphate mmol/l	2.71	2.33	3.1	3.02	2.91	2.814
Glucose mmol/l	7.59	8.82	1.17	3.93	2.13	4.728
Sodium mmol/l	125.9	201.3	166.2	192.1	127.2	162.5
Potassium mmol/l	7.3	10.8	25.0	12.2	5.0	12.06
Calcium mmol/l	1.79	1.91	2.14	1.66	1.56	1.81
Magnesium mmol/l	.67	.76	1.14	1.2	1.22	.998
Lactate mmol/l	3.68	3.72	4.17	3.61	3.97	3.83
Urine pH	6.4			7.00		
blood	large			large		
glucose	dark			-		
S.G.	1.037			1.028		

$\bar{X}$ = Mean

STATISTICAL ANALYSES

The means, standard deviations, standard errors and co-efficients of variation were calculated by the standard formulae.

Data comparisons between the 9 m.o. females, the 14 m.o. females and the males were made using the Students 't' test. This has been supplemented by computing the analyses of variance for the parameters using the Teddybear programme devised by J.B. Wilson. All data was punched onto computer cards, the control cards added and processed on the University of Otago's computer.

Mean of population $\bar{X} = \frac{\sum_{j=1}^{N} X_j}{N}$

Standard deviation (σ) s (dispension about mean) $= \sqrt{\frac{\Sigma f(x-\bar{x})^2}{N}}$

$= \sqrt{(x-\bar{x})^2}$

Standard error $s_{y.x.}$ (measure of reliability of mean of sample representing the mean of whole population) $= \sqrt{\frac{\Sigma (Y-Yc)^2}{N}}$

$= \frac{\sigma}{\sqrt{N}}$

where Y = actual amount present Yc = estimated amount present

N = no. of animals.

Co-efficient of Variation V = $\frac{\text{Standard deviation}}{\text{Mean}} \times \frac{100}{1}$

(measures the variation or dispension from standard deviation) = $\frac{S}{\bar{X}}$

Variance population variance σ^2

sample variance from this population S^2

't' test for comparing population means

$$'t' = \frac{m_1 - m_2}{\sqrt{S^2\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}} \quad \text{for } (n_1 + n_2 - 2) \text{ degrees of freedom}$$

$$S^2 = \frac{(n_1 - 1)S_1^2 + (n_2 - 1)S_2^2}{n_1 + n_2 - 2}$$

S = standard deviation = $S.E.\sqrt{N}$

n = sample number

m = sample mean

S.E. = standard error = $\frac{S}{\sqrt{N}}$

ANALYSIS OF VARIANCE

All data was punched onto computer cards. The instruction cards for the Teddy Bear programme of analysis of variance were added in the appropriate places, and the whole analysed by computer.

This analyses the variation within groups and between groups and tests if all the samples are or could be from the same population. The probability of the significance was taken at the 5% level ($P < .05$).

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