Post-anaesthetic Forelimb Lameness in Horses

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FORELIMB LAMENESS following general anaesthesia is known to occur in horses but there are relatively few references to the condition in the literature. Pearson and Gibbs (1970) recognised "post-operative radial paralysis" as a hazard following laparotomy in lateral recumbency and it was suggested that the incidence could be reduced by placing a straw filled sack under the sixth cervical vertebra (Irwin, 1970).

Although the condition is often described as "post-operative radial (nerve) paralysis" the pathogenesis is far from clear. Marolt, Bego, Vukelic, Sankovic and Zeskov (1962) and Marolt (1968) attributed the condition to a temporary reduction of blood flow in the leg caused by compression of the brachial artery.

These authors produced symptoms experimentally by compressing the axillary artery or by severing the radial nerve proximal to its branch to the triceps muscle. They considered that a true radial nerve paralysis arose very rarely under "post-operative" conditions.

Gerber (1968) referred briefly to "an azoturia-like syndrome" sometimes observed after long lasting surgical interventions and suggested that the measurement of serum levels of muscle enzymes might be of value in establishing a prognosis.

Serum transaminase levels during various acute myopathies of horses were examined by Hansen (1970) and among the horses with elevated levels he lists two as cases of "post-operative myoglobinuria". No further clinical details are given (except the subsequent death of one of the animals) and the author does not record when blood samples were taken.

The studies reported here were prompted by the development of post-anaesthetic lameness in several horses and represent an attempt to establish whether the condition observed was a result of radial nerve paralysis or was primarily muscular in origin. Observations made on horses subsequently making an uneventful recovery are included for comparison.

MATERIALS AND METHODS

Horses

Observations were made on thirteen horses and four ponies varying in age from 1 to 25 years and from 206 to 690 kg in body weight. Eleven were clinical cases and the rest experimental. Some horses were anaesthetised several times giving a total of 24 anaesthetics. Details are shown in Table I.

Anaesthesia

The following anaesthetic procedures were carried out:

Premedication: Acepromazine 0.05 mg/Kg or Xylazine 2.2 mg/Kg.

Induction of anaesthesia: Thiopentone sodium 11 mg/Kg or Methohexitone sodium 5.5 mg/Kg with Suxamethonium chloride 0.1 mg/Kg.

Anaesthesia: Halothane/oxygen.

Two horses (Nos. 3 and 4) were premedicated with acepromazine and induced with 10 per cent chloral...
Hydrate at 0.1 mg/Kg; anaesthesia was maintained with chloral hydrate while breathing air. A thick straw bed was used for horse number eight, but all the others lay on a two inch thick rubber mat, with or without an additional padded rug.

**Blood samples**

All estimations were carried out on blood taken from the jugular veins. Samples from most animals were taken immediately before induction, at the end of anaesthesia, and thereafter at approximately 20 minute intervals throughout anaesthesia.

At the end of the administration of the anaesthetic both horses were turned over and samples taken after a five minute interval. Further samples were obtained before the animals stood up and then every two to three hours for the first 48 hours, subsequent samples were taken at eight hour intervals for two days and then daily for a further 3 days.

**Degree of lameness**

The degree of any lameness observed was classified from very slight (+) to severe (++++)

### TABLE I

**DETAILS OF HORSES AND PROCEDURES**

<table>
<thead>
<tr>
<th>Horse/ anaesthesia No.</th>
<th>Type</th>
<th>Age years</th>
<th>Sex</th>
<th>Weight Kg.</th>
<th>Procedure</th>
<th>Duration of anaesthesia hours</th>
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<tbody>
<tr>
<td>1</td>
<td>Hunter</td>
<td>7</td>
<td>G</td>
<td>550</td>
<td>Tooth extraction</td>
<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>2</td>
<td>Polo pony</td>
<td>-</td>
<td>G</td>
<td>476</td>
<td>Lens extraction</td>
<td>1\frac{1}{2}</td>
</tr>
<tr>
<td>G</td>
<td>Welsh mountain pony</td>
<td>2</td>
<td>G</td>
<td>255</td>
<td>Experimental</td>
<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>R</td>
<td>Welsh mountain pony</td>
<td>2</td>
<td>G</td>
<td>247</td>
<td>Experimental</td>
<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>O</td>
<td>Show-jumper</td>
<td>12</td>
<td>G</td>
<td>653</td>
<td>Exploration nasal sinuses</td>
<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>U</td>
<td>Thoroughbred</td>
<td>25</td>
<td>M</td>
<td>544</td>
<td>Experimental</td>
<td>3\frac{1}{2}</td>
</tr>
<tr>
<td>P</td>
<td>Thoroughbred</td>
<td>25</td>
<td>M</td>
<td>544</td>
<td>Experimental</td>
<td>3\frac{1}{2}</td>
</tr>
<tr>
<td>F</td>
<td>Thoroughbred</td>
<td>25</td>
<td>M</td>
<td>544</td>
<td>Experimental</td>
<td>3\frac{1}{2}</td>
</tr>
<tr>
<td>A</td>
<td>Anglo-arab</td>
<td>7</td>
<td>M</td>
<td>423</td>
<td>Experimental</td>
<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>8</td>
<td>Thoroughbred</td>
<td>8</td>
<td>M</td>
<td>649</td>
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<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>9a</td>
<td>Thoroughbred</td>
<td>5</td>
<td>G</td>
<td>477</td>
<td>Exploration nasal sinuses</td>
<td>1\frac{1}{2}</td>
</tr>
<tr>
<td>R</td>
<td>Anglo-arab</td>
<td>7</td>
<td>M</td>
<td>423</td>
<td>Experimental</td>
<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>O</td>
<td>Thoroughbred</td>
<td>6</td>
<td>G</td>
<td>598</td>
<td>Tumour excision</td>
<td>1\frac{1}{2}</td>
</tr>
<tr>
<td>U</td>
<td>Thoroughbred</td>
<td>8</td>
<td>M</td>
<td>518</td>
<td>Arthrotomy</td>
<td>1\frac{1}{2}</td>
</tr>
<tr>
<td>P</td>
<td>Thoroughbred</td>
<td>1</td>
<td>E</td>
<td>360</td>
<td>Radiography</td>
<td>35 mins.</td>
</tr>
<tr>
<td>B</td>
<td>Thoroughbred</td>
<td>11</td>
<td>M</td>
<td>504</td>
<td>Tumour excision</td>
<td>1\frac{1}{2}</td>
</tr>
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<td>G</td>
<td>Thoroughbred</td>
<td>7</td>
<td>M</td>
<td>423</td>
<td>Experimental</td>
<td>2\frac{1}{2}</td>
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<td>R</td>
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<td>M</td>
<td>423</td>
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<tr>
<td>O</td>
<td>Thoroughbred</td>
<td>5</td>
<td>G</td>
<td>477</td>
<td>Radiography</td>
<td>1\frac{1}{2}</td>
</tr>
<tr>
<td>U</td>
<td>Thoroughbred</td>
<td>4</td>
<td>E</td>
<td>522</td>
<td>Arthrotomy</td>
<td>1\frac{1}{2}</td>
</tr>
<tr>
<td>P</td>
<td>Welsh mountain pony</td>
<td>2</td>
<td>G</td>
<td>206</td>
<td>Experimental</td>
<td>3\frac{1}{2}</td>
</tr>
<tr>
<td>16</td>
<td>Welsh mountain pony</td>
<td>2</td>
<td>G</td>
<td>206</td>
<td>Experimental</td>
<td>2\frac{1}{2}</td>
</tr>
<tr>
<td>17</td>
<td>Show-jumper</td>
<td>-</td>
<td>G</td>
<td>690</td>
<td>Exploration corono-pedal joint</td>
<td>1\frac{1}{2}</td>
</tr>
</tbody>
</table>

**Biochemistry**

(a) **Lactate and pyruvate**: Lactate and pyruvate were determined enzymatically according to the method described by Fleischer (1970) using blood taken from the jugular vein and mixed immediately with an equal volume of 0.6M perchloric acid (B.D.H. Analar). Values are recorded as mg/100 ml whole blood.

(b) **Serum enzymes**: "Activated" serum creatine phosphokinase (CPK), serum glutamic oxaloacetic transaminase (GOT) and serum sorbitol dehydrogenase (SDH) were measured using the Boehringer Mannheim test combinations. All values were recorded as milli-international units/ml serum (mIU/ml).

(c) **Potassium**: Serum potassium was estimated by flame photometry and values recorded as milli-equivalents per litre of serum (mEq/L).

**RESULTS**

Nine horses (Nos. 1 to 9, Table I) developed post-anaesthetic lameness; horse No. 6 on three separate occasions. The intervals between anaesthetics 6a and 6b, and 6b and 6c were 13 months and three weeks respectively.

**Clinical observations**

The lameness always developed in the foreleg on which the horse lay during anaesthesia. On nine occasions the lameness was immediately obvious when the horses...
stood but in two (Nos. 5 and 7) it was not apparent
til 15 to 30 minutes later and took several hours to
develop fully.
The degree of lameness and its duration for each
animal are recorded in Table II. The horses most
severely affected (++++++) were unwilling to place
any weight on the leg and remained recumbent for the
first 24 or 48 hours; in those less affected (+++ and
++) the leg had a tendency to knuckle at the fetlock
and to give way when bearing weight. The characteristic
posture of the horse when affected is shown in fig. 1
(horse No. 8).

The lameness in each case was accompanied by a
marked hardening in the muscles of the shoulder region,
in particular the anterior superficial pectoral muscle,
the caudal end of the brachiocephalicus and the deltoideus
and triceps muscles. There was some variation between
animals in the degree to which individual muscles were
affected. In five horses (Nos. 1, 2, 3, 4, 5 and triceps muscles. There was some variation between
the caudal end of the brachiocephalicus and the deltoideus
muscles were noticeably swollen and in addition horses
Nos. 3, 8 and 13 developed large oedematous plaques
over the ribs on the same side. The masseter muscle
of horse No. 6 was very enlarged after the first anaesthetic
(a).

The duration of the lameness varied between one and
seven days (Table II) but the oedema persisted for longer.

Biochemical observations
(a) Lactate and pyruvate: The mean pre-anaesthesia
blood lactate level for all the horses was $8.9 \pm 4.2$ mg/
100ml whole blood, high levels (up to $21.7$ mg/100ml
whole blood) being seen in nervous horses. There were
no appreciable differences between the pre-anaesthetic
levels recorded in horses subsequently becoming lame
(Table II) and the two groups represented in Table III.
The blood lactate levels of horses which became lame,
where measured, are shown in Table II. At the termina-
tion of administering the anaesthetic the horse was
turned over and blood samples taken five minutes later.

<table>
<thead>
<tr>
<th>Horse No.</th>
<th>Degree of lameness</th>
<th>Duration of lameness, days</th>
<th>Maximum recorded enzyme value mIU/ml</th>
<th>Lactate mg% at end of anaesthesia</th>
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<tr>
<td>1</td>
<td>++++</td>
<td>7</td>
<td>14,000</td>
<td>3,000</td>
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<td>2</td>
<td>++</td>
<td>3</td>
<td>142</td>
<td>201</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>5</td>
<td>3,620</td>
<td>360</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>1</td>
<td>1,340</td>
<td>390</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td>overnight</td>
<td>378</td>
<td>330</td>
</tr>
<tr>
<td>6a</td>
<td>++++</td>
<td>7</td>
<td>710</td>
<td></td>
</tr>
<tr>
<td>6b</td>
<td>++++</td>
<td>4</td>
<td>3,260</td>
<td>1,170</td>
</tr>
<tr>
<td>6c</td>
<td>++</td>
<td>3</td>
<td>1,180</td>
<td>1,080</td>
</tr>
<tr>
<td>7a</td>
<td>++</td>
<td>2</td>
<td>217</td>
<td>1,340</td>
</tr>
<tr>
<td>8</td>
<td>++++</td>
<td>6</td>
<td>4,250</td>
<td>1,350</td>
</tr>
<tr>
<td>9a</td>
<td>+</td>
<td>2</td>
<td>81</td>
<td></td>
</tr>
</tbody>
</table>

* This animal was not moved before the blood sample was taken.
then fell to 4.7 within 15 minutes and then rose to 14.7 as the animal rolled onto its brisket. A level of 48.2 was recorded in the next sample taken as the animal struggled to stand.

The mean pyruvate levels, standard deviations from the mean and ranges measured in the three groups of horses (A, B and C) were:

1. Pre-anaesthesia
   - Group A: 0.8±0.3 (0.5--1.2), 1.2±0.7 (0.5--2.8)
   - Group B: 1.0±0.4 (0.3--1.8), 1.5±0.6 (0.7--2.7)
   - Group C: 0.9±0.2 (0.7--1.3) mg/100ml.

2. At the end of anaesthesia
   - Group A: 1.1±0.5 (0.5--2.3), 0.8±0.4 (0.3--1.2)
   - Group B: 1.1±0.4 (0.6--1.9) mg/100ml.
   - Group C: 1.2±0.5 (0.7--1.3) mg/100ml.

(b) Serum enzymes: The pre-anaesthesia levels of "activated" CPK are shown in Tables II and III; most results fell within the range 10-30 mU/ml serum and there are no obvious differences between the groups. The post-anaesthetic serum CPK levels of the lame horses rose dramatically (Table II), while the 24 hour levels of serum CPK in the horses recovering normally in general show only a slight increase (Table III). GOT levels followed the same pattern.

The changes in serum CPK and GOT levels of horse No. 8 during and after anaesthesia are shown in fig. 3. These graphs illustrate the typical pattern observed with the lame horses, an immediate post-anaesthetic rise of serum CPK and a less marked, slower increase of GOT.

(c) Serum potassium: There were no differences in serum potassium levels between groups, either before anaesthesia, at the end of anaesthesia or 24 hours later. However, in 17 of the 20 anaesthetics when values were recorded the serum potassium levels tended to rise during anaesthesia. The mean and standard deviation for all the horses of the samples taken at the end of administering the anaesthetic was 4.7±0.9 mEq/L compared with 4.0±0.5 mEq/L for the pre-anaesthetic samples, however, this increase is of a low order of significance (p<0.01 but >0.05). The 24 hour samples tended to show a return to pre-anaesthesia levels. The mean for the 24 hour samples was 4.0±1.1 mEq/L. The decrease was significant p<0.02.

DISCUSSION

Although post-anaesthetic forelimb lameness is often referred to as radial paralysis the present observations are more consistent with a muscular aetiology. In the cases reported the chief muscles involved were those of the shoulder region, which were hard rather than flaccid on palpation.

The increase of serum CPK occurring immediately after anaesthesia is highly significant in that elevated CPK is generally accepted to be a specific and sensitive indicator of muscle damage (Henson and Rao, 1966; Gerber, 1968) for whereas increases in serum enzyme levels occur in muscular dystrophies they are not found in cases of neurogenic origin in humans (Schmidt and Schmid, 1967) or horses (Gerber, 1968) nor consistently in diseases of the central nervous system (Langton et al., 1967; Smith and Healy, 1968).

It would appear that the lameness resulted from a mechanical impairment of function which was in some cases exacerbated by pain. Marolf et al., (1962) stated that, in their experience, the lameness did not last longer than one day but it can be seen from Table II that in

<table>
<thead>
<tr>
<th>Group</th>
<th>Horse/anaesthetic No.</th>
<th>Serum CPK levels mU/ml</th>
<th>Lactate at end of anaesthesia mg/100 ml whole blood</th>
<th>Mean and S.D.</th>
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<tr>
<td></td>
<td></td>
<td>Pre-anaesthetic</td>
<td>24 hours</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>7b</td>
<td>—</td>
<td>—</td>
<td>12.7</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>24</td>
<td>150</td>
<td>13.1</td>
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<td>11</td>
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<td>79</td>
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<td></td>
<td>12*</td>
<td>120</td>
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<td>15.3</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>14</td>
<td>510</td>
<td>13.7</td>
</tr>
<tr>
<td></td>
<td>17*</td>
<td>24</td>
<td>134</td>
<td>12.9</td>
</tr>
<tr>
<td>C</td>
<td>7c</td>
<td>18</td>
<td>60</td>
<td>13.0</td>
</tr>
<tr>
<td></td>
<td>7d</td>
<td>—</td>
<td>60</td>
<td>7.4</td>
</tr>
<tr>
<td></td>
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<tr>
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<td>15a</td>
<td>29</td>
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</tr>
<tr>
<td></td>
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<td>—</td>
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<tr>
<td></td>
<td>17*</td>
<td>24</td>
<td>134</td>
<td>9.7</td>
</tr>
</tbody>
</table>

* 12 and 17 same anaesthetic.
nine of the eleven cases listed it lasted for longer than this, on average four days, and in two cases (Nos. 1 and 6a) lameness persisted for a week.

While the results from horses recovering normally do not show the significant rise in lactate/pyruvate ratio during anaesthesia as reported by De Moor (1968), significant changes in blood lactate at the end of anaesthesia were demonstrated in the animals which subsequently became lame, provided they had been turned over. This suggests an inadequate blood flow in the affected limbs during anaesthesia.

In four of the cases from which samples were taken the values were significantly higher (p<0.01) than the control values from either groups B or C. The difference between groups B and C, although less significant, and the increase seen on turning horses Nos. 12 and 17 suggests that the rise in circulating lactate occurred after tissue perfusion was restored to the side underneath, when accumulated lactate entered the general circulation.

Although this seemed to occur normally to some extent, hence the difference between groups B and C, the animals becoming lame had accumulated a larger amount and showed a correspondingly greater increase.

The results from horses Nos. 8 (fig. 2) and 6c, in which levels were followed closely throughout anaesthesia, support this view since no marked increase in lactate in the general circulation was seen until the horses were turned over.

This rise in blood lactate, although comparable with the increases in lactate occurring normally after exercise, is highly significant since it would appear to be derived from a relatively small proportion of the body muscle mass. Thus the concentration in the affected muscles may be many times that found during exercise.

If a reduction in tissue perfusion occurs several factors probably contribute:

1. A reduction in blood flow through the brachial artery which was demonstrated using radiographical techniques by Marolf et al. (1962). This may be the result of external compression of the artery together with the fall in blood pressure induced by halothane (Vasko, 1962).

2. Local compression of smaller blood vessels by body weight and this is suggested by variations in the degree to which different muscles were affected in different animals and the occurrence of subsidiary swellings on the head, chest or flank in some cases.

Skinner, Spector and Yap (1969) have shown that ischaemic brain damage in rats leads to a persistent deficiency of tricarboxylic acid cycle intermediates within the brain cells. These authors suggest that one consequence of this could be a failure of energy dependent ionic pumping mechanisms which would result in sodium (and water) inflow into the cells.

Intracellular oedema in muscle after temporary ischaemia has been demonstrated by many workers (see review by Allbrook, Baker and Kirkaldy-Willis, 1966). The lesions would be, to some extent, self-potentiating if the increase in pressure due to the oedema should lead to a collapse of the venous return and subsequent inadequate blood circulation in the affected muscle.

While the degree of damage may be related to the degree of anoxia there is no evidence from the results presented that the duration of the anaesthesia or the weight of the horse is important. Exercise immediately following may make the condition worse by increasing the oxygen demand of the muscles.

The condition appears to share at least some features with equine paralytic myoglobinuria. In both the muscles involved are hard, swollen and painful and if the idea of an excessive production of lactate in the latter (Carlstrom, 1931), is correct then the local accumulation of lactate could be of prime importance in the pathogenesis of both conditions by causing direct damage to the cells, or, alternatively, the lactate accumulation could be a manifestation of a temporary insufficiency of the oxygen supply to the muscle.

A "vicious cycle" has been suggested for paralytic myoglobinuria (Smith and Jones, 1966) in which the accumulated lactate, by causing spasmotic contraction of the muscle reduces the free flow of blood and causes a still greater shortage of oxygen; the cramped or spasmotically contracted muscle continues to work thereby increasing the oxygen deficit in lactate accumulation. The only essential difference between the two conditions may be the point of entry into the vicious cycle.

The serum levels of the enzymes released from the damaged muscle (fig. 3) follow a course strikingly similar to the sequence observed in equine paralytic myoglobinuria (Cardinet, Littrell and Freedland, 1967), i.e. CPK rises sharply and declines quickly while serum GOT shows a more gradual but sustained increase. This is presumably due to the difference in their rates of disappearance from the plasma.

Elevation of serum CPK is considered to be a reliable indicator of muscle damage (Gerber, 1968). The
immediate post-anaesthetic rise of CPK in our experiments is noteworthy. If lysosomal enzymes are released at the same time these will no doubt contribute to the kind of self-potentiating lesion previously mentioned, especially if secondary membrane permeability contributes to the oedema.

Thus clinical observations supported by laboratory findings suggest that the forelimb lameness following general anaesthesia in lateral recumbency is a result of muscular ischaemia.

The criteria for the genesis of this condition are not clear since the same horse may be anaesthetised under apparently similar conditions for the same length of time and develop lameness on one occasion and not another (e.g. horses Nos. 7 and 9). Thus it is not certain whether measures taken to prevent its occurrence have any beneficial effect.

SUMMARY

Clinical and biochemical observations on eleven cases of post-anaesthetic forelimb lameness were compared with similar studies on horses anaesthetised in lateral recumbency making an uneventful recovery. It is suggested that the condition resulted from muscular ischaemia during the recumbent phase.

RÉSUMÉ

Les observations cliniques et biochimiques sur 11 cas de boiteries post anesthésiques sont comparées à celles de même nature faites sur des chevaux ayant été anesthésiés sans suite aucune.

Il semblerait que les boiteries soient, dans ces cas mentionnés, le résultat d'une ischémie musculaire durant la phase de decubitus.

ZUSAMMENFASSUNG


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REFERENCES


