References

Keenan, D. M. (1979). Changes of blood metabolites in horses after racing with particular reference to uric acid. *Aust. Vet. J.* **55**, 54–57.

Rose, R. J., Purdue, R. A. and Hensley, W. (1977). Plasma biochemistry alterations in horses during an endurance ride. *Equine Vet. J.* **9**, 122–126.

Snow, D. H. and Mackenzie, G. (1977). Effects of training on some metabolic changes associated with submaximal endurance exercise in the horse. *Equine Vet. J.* **9**, 226–230.

Preliminary Studies on the Relationship of Red Blood Cell Potassium Concentration and Performance

E. MUYLLE, C. VAN DEN HENDE, J. NUYTTEN, W. OYAERT and K. VLAMINCK
Department of Medicine, Large Animal Clinic, State University of Ghent, Belgium.

Summary

No correlation was found between plasma and red blood cell potassium concentration, when samples taken from about 200 horses were examined. In 43 horses, the relationship between red blood cell potassium concentration and performance was evaluated. Of 11 horses that were considered to have a low red blood cell potassium concentration, 9 were performing unsatisfactorily on the racecourse. It is suggested that a low red blood cell potassium concentration correlates to a low muscle potassium concentration.

Introduction

Several motives incited us to examine the potassium status in racehorses and to look for a possible relationship with fitness.

Normal individuals

1. According to Williamson (1974) the serum K^+ in winning horses varies between 3.7 and 4.0 mmol/l.
2. During muscular exercise the K^+-ions released from the muscle cells are among the most potent arteriolar vasodilators (Ganong 1977).
3. The work-induced K^+-release stimulates C fibres which mediate the increase in cardiorespiratory reflex activity (Schumacher *et al.*, 1979).
4. There exist close correlations between extracellular increases in potassium, the increase in muscle blood flow, the increase in oxygen consumption and the increase in muscle performance (Hirche *et al.*, 1980).

Potassium-depleted individuals

1. Bilbrey *et al.* (1973) demonstrated that in K^+- deficient dogs, a decreased potassium release from contracting muscles was associated not only with a marked impairment of exercise hyperaemia but also with necrosis of skeletal muscle.
2. The force–velocity relationships of K^+- depleted cat papillary muscles were sharply depressed and the reduced tension development was clearly correlated with a decrease in calcium (Ca) binding capacity of the sarcoplasmic reticulum (Sack *et al.*, 1974).

3. The muscle blood flow is insufficient, leading to hypoxia, anaerobic glycolysis and metabolic acidocis. This results in a damage of muscle membranes, inducing an excessive K^+ loss which cannot be restored by a deficient Na^+/K^+ pump (Oster *et al.*, 1978).
4. Dengler *et al.* (1979) found weaker contractions in rat skeletal muscles associated with reduced amplitudes of the action potential and slowing of the depolarization process.
5. In cows with parturient paresis the Ca content in the muscle cells is normal but there is a decrease in the muscle K^+ concentration (Kcwalczyk and Mayer, 1972).
6. In periodic hypokalaemic or hyperkalaemic paralysis, a reduced K^+ content was associated with an increased or decreased resting membrane potential, respectively (Buruma and Bots, 1978).

Although Stern *et al.* (1981) maintain that changes in plasma K^+ concentrations and total body K^+ content are linearly related, several statements indicate that one cannot equate the plasma K^+ concentrations with total body stores (Lindeman 1976) with any degree of confidence.

Different observations indicate that determination of the K^+ content of red blood cells might be a valuable approach in estimating total body K^+ stores.
1. ECG changes of K^+ deficit are always correlated with a low K^+ concentration in the red blood cells (RBC), irrespective of the kalaemia (Soloff *et al.*, 1960).
2. Potassium levels in human muscle biopsies and in RBCs are well correlated to some degree, whilst plasma K^+ levels are not (Ibsen 1974).
3. Digitalisation reduces the muscle K^+ and the RBC K^+ to the same extent (Ericsson *et al.*, 1981).
4. In horses with severe diarrhoea the RBC K^+ may decrease to about 75 mmol/l, whilst plasma K^+ is normal, reduced or even elevated. Assuming an equal depletion in muscle, the total loss of K^+ in a 500 kg horse is about 4500 mmol. This is the same as the figure quoted by Carlson (1979).

Material and Methods

In three training centres for trotters, blood samples were taken every three weeks from 43 horses. Sampling was performed by jugular puncture in the morning before feeding. The blood was collected in lithium heparinized vacuum tubes and immediately transported to the laboratory. Packed cell volume (PCV) was determined with the microhaematocrit centrifuge (five minutes at 10 000 rev./min.). Red blood cell potassium was determined according to the method described by Ibsen (1974). This technique is based upon the determination of whole blood potassium (K^+.WB) after haemolysis, plasma K^+ (K^+.P) and PCV.K^+RBC will then be: $\frac{100}{PCV}(K^+.WB - K^+.P) + K^+.P$. Potassium was determined by flame photometry.

At each of the training centres the horses were fed twice daily. Food consisted of concentrates (oats) and hay, and all horses received the same diet. There were no differences between the stables of the respective trotters.

An evaluation of the state of fitness of the respective racehorses was obtained by asking the opinions of the trainer and the jockey and by careful registration of the race performances. Horses were estimated to be fit when they could equal or better their record time over a certain distance or when they could go faster than their normal competitors.

For the evaluation of the possible correlation between plasma K^+ and RBC K^+, additional blood samples were taken from about 200 horses presented at the clinic for various disorders.

Results and Discussion

Correlation of plasma potassium and red blood cell potassium

The results of this study are given in Table 1.

TABLE 1. Correlation of plasma potassium and red blood cell potassium.

n	RBC K^+ mmol/l	Plasma K^+ mmol/l	Coefficient of correlation
512	93.9 ± 7.1	3.6 ± 0.5	+ 0.076

The absence of any correlation corresponds with the results of Lindeman (1976) and Ibsen (1974).

A comparison of plasma K^+ and red blood cell K^+ in individual horses showed clearly that severe hypokalaemia was in no way a reliable parameter for RBC K^+ depletion. Conversely, hyperkalaemia did not exclude a RBC K^+ depletion.

Correlation between RBC K^+ and performance

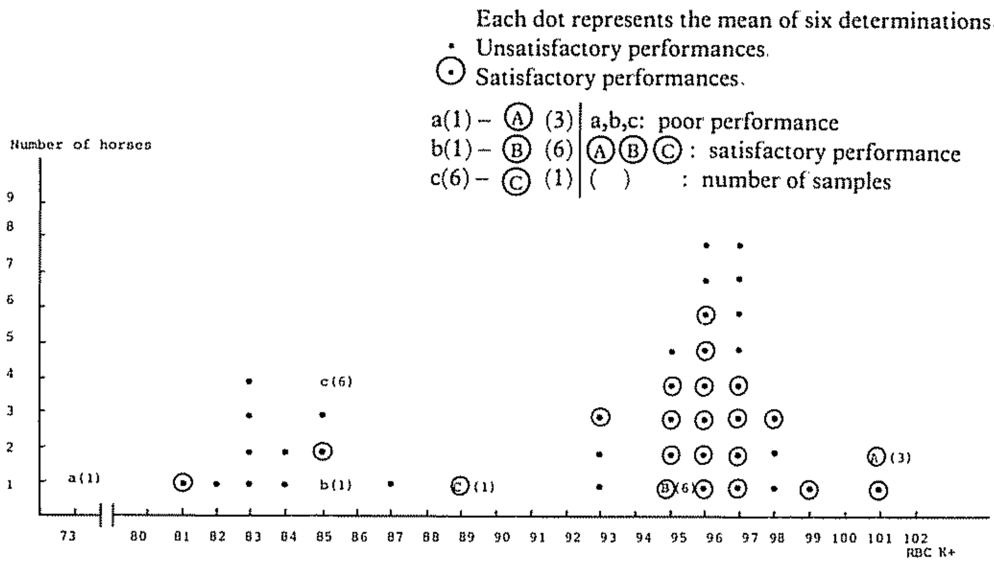
Only those results obtained during the last four months of the experimental period will be given because, initially, some technical problems might have given erroneous results.

It was observed that plasma K^+ levels varied irregularly in the respective horses in successive samples. On the other hand, the potassium content in the red blood cells was very stable in successive samples from most horses. As illustrated in Fig. 1, a constant high RBC K^+ was found in 26 out of the 43 trotters studied. The potassium content in this group varied between 95 and 101 mmol/l. The dots represent the mean potassium levels of all determinations (about six from the respective horses). The standard deviation never exceeded 4 mmol/l. Horses represented by single dots were performing below their capacities. Horses represented by encircled dots were competing very well, as they won regularly or improved their record time.

In 11 out of the 43 trotters, RBC K^+ varied constantly between 81 and 87 mmol/l. Only two horses of this group were competing to a satisfactory degree, although their performance was rather irregular, i.e. they won some races but raced very poorly in others.

Three horses identified as A, B and C initially fell into the low K^+ group and were performing poorly at that time. An increase in their RBC K^+ was accompanied by a gradual improvement in their performance. The increase in RBC K^+ of horses A and B occurred early in the experimental period, whereas horse C maintained a RBC K^+ of 85 mmol/l for three months. A spontaneous increase in RBC potassium concentration was accompanied by an obvious improvement in performance.

FIGURE 1. Mean RBC K^+ in 43 trotters in training.



Conclusions

The results indicate very clearly that the determination of plasma potassium is an unreliable parameter for assessing the red blood cell potassium content. Some preliminary experiments on muscle potassium determinations, which are being carried out at this time but which are too small in number to allow conclusive statements, point towards a correlation between RBC K^+ and muscle K^+.

As far as the correlation between performance and RBC K^+ is concerned, it is evident that the data presented here do not allow definite conclusions to be drawn, especially as two trotters with a constant low RBC K^+ were performing rather well. These horses will be followed during the coming months.

The possibility that low RBC K^+ could be related to some blood lines was ruled out because there were no family ties between the trotters with the low potassium concentrations.

It is possible that the magnesium status of the horses is of importance, since several authors found correlations between magnesium deficiencies and potassium depletion (Whang *et al.*, 1967; Schils 1969; Francesco *et al.*, 1981; Güllner *et al.*, 1981).

References

Bilbrey, G. L., Herbin, L., Carter, N. W. and Knochel, J. P. (1973). Skeletal muscle resting membrane potential in potassium deficiency. *J. Clin. Invest.* **52**, 3011–3018.

Buruma, O. J. and Bots, G. T. (1978). Myopathy in familial hypokalaemic periodic paralysis independent of paralytic attacks. *Acta Neurol. Scand.* **57**, 171–179.

Carlson, G. P. (1979). Fluid therapy in horses with acute diarrhea. *Vet. Clin. N. Am. Large Anim. Pract.* **1**, 313–329.

Dengler, R., Hofmann, W. and Rüdel, A. (1979). Effects of potassium depletion and insulin on resting and stimulated skeletal rat muscle. *J. Neurology* **42**, 818–826.

Ericsson, F., Carlmark, B., Jogestrand, T. and Sundqvist, K. (1981). Effect of digoxin upon intracellular potassium in man. *Scand. J. Clin. Lab. Invest.* **41**, 457–463.

Francesco, L. L., Saurin, L. L. and Dibana, G. F. (1981). Mechanism of negative potassium balance in the magnesium deficient rat. *Proc. Soc. Exp. Biol. and Med.* **168**, 382–388.

Ganong, W. F. (1977). *Review of medical physiology.* 8th edition. Lange Medical Publications. p. 471.

Güllner, H., Gill, J. R. and Bartler, F. C. (1981). Correction of hypokalemia by magnesium repletion in familial hypokalemic alkalosis with tubulopathy. *Am. J. Med.* **71**, 578–582.

Hirche, H., Schumacher, E. and Hageman, H. (1980). Extracellular K^+ concentration and K^+ balance of the gastrocnemius muscle of the dog during exercise. *Pflügers Arch.* **387**, 231–237.

Ibsen, H. (1974). Red blood cell potassium as a measure of body potassium in Thiazide-treated patients with essential hypertension. *Scand. J. Clin. Lab. Invest.* **34**, 161–165.

Kowalczyk, D. F. and Mayer, G. P. (1972). Cation concentration in skeletal muscle of paretic and non-paretic cows. *Am. J. Vet. Res.* **33**, 751–757.

Lindeman, R. D. (1976). Hypokalemia: Causes, consequences and correction. *Am. J. Med. Sci.* **272**, 5–17.

Muylle, E., Van Den Hende, C., Nuytten, J. and Oyaert, W. (1980). Evaluation of fitness in race horses. *Proc. Acad. Soc. Large Anim. Vet. Med.* **5**, 27–33.

Oster, J. R., Perez, G. O. and Vaamonde, C. A. (1978). Relationship between blood pH and potassium and phosphorus during acute metabolic acidosis. *Am. J. Physiol.* **235**, F345–F351.

Sack, D. W., Kim, N. D. and Harrison, C. E. (1974). Contractibility and subcellular calcium metabolism in chronic potassium deficiency. *Am. J. Physiol.* **226**, 756–763.

Schils, M. E. (1969). Experimental magnesium depletion. *Medicine* **48**, 61–85.

Schumacher, E., Hageman, H., Hirche, H. and Bovenkamp, U. (1979). Extracellular K^+ activity and K^+ balance of working skeletal muscle. *Pflügers Arch.*, Suppl. to Vol. **368**, R23.

Soloff, L., Kanasky, S., Boutwell, J. and Rowell, D. (1960). The relationship of the electrocardiographic pattern of potassium depletion to the concentration of potassium in red blood cells. *Am. J. Med. Sci.* **240**, 280–288.

Stern, R. H., Cox, M., Feig, P. and Singer, I. (1981). Internal potassium balance and the control of the plasma potassium concentration. *Medicine* **60**, 339–354.

Whang, R., Morasi, H. T., Rogers, D. (1967). The influence of sustained magnesium deficiency on muscle potassium repletion. *J. Lab. Clin. Med.* **70**, 895–902.

Williamson, H. M. (1974). Normal and abnormal electrolytic levels in the racing horses and their effect on performance. *J. S. Afr. Vet. Ass.* **45**, 335–340.