Influence of motor load (treadmill) on blood gas partial pressure with clinically healthy and respiratory diseased horses with special consideration of hypoxic status

VII Summary

The aim of this thesis is the examination of horses with respiratory disease before and after motor stress. For this purpose, the horses were moderately exercised on a treadmill (1,7 m/s) for 20 min. In practice, n= 75 horses (age: 2,5 – 18 years) were examined. Of these 75 horses n=37 animals showed respiratory symptoms. These symptoms were classified using a score system that utilised clinical and laboratory symptoms. Symptomatic horses were assigned to three groups exhibiting low (n=11), middle (n=8) and high (n=8) levels of respiratory disease. An additional number of n= 3 healthy horses were exercised on a high speed treadmill (intervals between trot and gallop). The n= 35 healthy horses (without clinical signs of respiratory disease) were used for the determination of the reference range of the examined parameters.

Blood samples from A. carotis communis and V. jugularis externa were taken before, immediately after and 15 min after the end of the motor stress. Laboratory diagnosis includes the measurement of bloodgases, hemoxymetric findings as well as hematological and biochemical

values.

Findings in healthy horses

O_2 levels of arterial blood were paO_2 = 13,5 ±0,61 kPa (normoxaemia) and $paCO_2$ = 5,8 ± 0,43 kPa (normocapnia). After motor stress, the paO_2-value (7 of 10 clients) increased slightly in agreement with other authors. In contrast, the $paCO_2$-values decreased (8 of 10 clients).

Analysis of blood from the jugular vein of the same horses showed values of $pjvO_2$ = 5,7 ±0,76 kPa and $pjvCO_2$ = 6,2 ±0,26 kPa. After motor stress these results did not change markedly.

The alveolo-arterial O_2-pressure difference ($AaDO_2$) is also a useful parameter for the differential diagnosis of respiratory diseases. For healthy animals in rest the values of these parameters were $AaDO_2$ was = -0,6 ±0,53. After motor load these values decreased slightly ($AaDO_2$ = -0,9 ±0,54).

The O_2-saturation graph and thus the affinity between O_2 and haemoglobin in blood were detected with the help of the parameter of "half-saturated-pressure" (p50). The values for healthy horses in rest were pa50 = 3,7 ±0,18 kPa and pjv50 = 3,6 ±0,26 kPa.
Loading of haemoglobin (saO_2) with O_2 was 98,1 ±0,75 % and $sjvO_2$ = 77,3 ±4,38 % for healthy horses in rest.
In rest, the values of reduced artery hemoglobin (raHb) in healthy animals were not increased over 1,6 g/l (raHb = 0,9 ±0,30 g/l). After motor load these values were only minimaly different. The corresponding jugularvenous finding was rjvHb = 30,2 ±8,64 g/l.
The blood-lactate values of healthy horses in rest were 1,04 ±0,53 mmol/l and persisted after motor load (0,96 ±0,75 mmol/l) without significant differences. The lactate-pyruvate-quotient in blood was 9,9 ± 2,69; after locomotion it did not show significant change (9,0 ± 2,23).

Findings of respiratory diseased horses

Arterial partial pressure in rest was paO_2 = 13,6 ±1,00 kPa in the case of a low degree respiratory disease (normoxaemia), paO_2 = 12,7 ±1,50 kPa intermediate disease and paO_2 = 10,1 ±1,95 kPa high grade respiratory disease (hypoxaemia). After motor load, paO_2-values of diseased horses decreased on the average (low-grade 12,6 ±1,02 kPa, intermediate 11,8 ±1,27 kPa, high grade 10,2 ±1,25 kPa). However, the individuals showed different reactions with both increased and decreased values observed after motor load. Possibly, these non-uniform reactions are due to underlying differences in the aetiology of the respiratory ailments shown by the clients. The different aetiologies are probably associated with differences in the subclinical functions of outside respiration. The comparison between healthy and respiratory diseased horses showed that the motor load used in these series of experiments did not lead to a critical situation with regard to pulmonary function.
The corresponding arterial CO_2-partial pressure in rest differed slightly between the groups (low-grade diseased: $paCO_2$ = 5,9 ±0,38 kPa, intermediate diseased: $paCO_2$ = 5,7 ±0,33 kPa, high grade diseased: $paCO_2$ = 6,1 ±1,41 kPa). These results did not significantly change after locomotion. In jugularvenous blood we found results of $pjvO_2$ = 5,3 ±0,75 kPa (low-grade), $pjvO_2$ = 5,0 ±0,47 kPa (intermediate) and $pjvO_2$ = 4,9 ±0,49 kPa (high grade). The results are below the results of healthy horses ($pjvO_2$ = 5,7±0,76 kPa). After exercise, the $pjvO_2$-values of diseased animals increased in most cases (18 x increase, 9 x decrease after locomotion).
The differences between alveolar and arterial O_2-pressure ($AaDO_2$) are correlated to the degree of the observed respiratory disease (at rest: low-grade -0,3 ±0,93, intermediate 0,9 ±1,44 kPa and high grade 2,9 ±0,7 kPa; after exercise: low-grade 0,8 ±0,82 kPa, intermediate 1,9 ±1,12 kPa and high grade 2,8 ±1,00 kPa).

The raHb-values increased in correlation to the degree of disease (raHb = 1,0 ±0,57 g/l (low-grade), raHb = 2,1 ±1,68 g/l (intermediate) and raHb = 4,76 ±1,99 g/l (high grade) at rest. The horses with high grade respiratory disease never showed values of raHb greater than 2 g/l in rest. After locomotion, the raHb increased to 1,9 ±1,37 g/l (low-grade), raHb = 2,6 ±0,65 g/l (intermediate) and raHb = 5,4 ±1,00 g/l (high grade). None of the horses with high grade disease showed a raHb < 4 g/l after locomotion. Corresponding rjvHb-values did not show significant difference between the diseased client groups (rjvHb results before and after exercise: (low-grade) 31,0 ±8,67 and 32,4 ±16,75 g/l, (intermediate) 33,5 ±6,95 and 30,4 ±8,48 g/l and (high grade) 45,1 ±14,36 and 45,9 ±13,87 g/l). As these results demonstrate, an increase in the degree of respiratory disease is correlated with a rise in the portion of functional haemoglobin in arterial blood which is not loaded with oxygen (raHb). Presumably, there are enough working cardiovascular and/or erythrocytic mechanisms for compensation of functional disorders that guarantee an essential supply with oxygen in diseased horses. The relatively uniform increase of raHb-values corresponded to the severity of the disease, i.e. this parameter might be of diagnostic value. It is important to show the results of raHb as the absolute value (g/l), not as percentage (%).

Haemoglobin loading with oxygen (saO_2) showed values between saO_2 = 133,4 ±9,64 g/l (low-grade), saO_2 = 136,8 ±12,56 g/l (intermediate) and saO_2 = 139,6 ±15,55 g/l (high grade) at rest. The corresponding jugularvenous results were $sjvO_2$ = 101,8 ±1[a],69 (low-grade), $sjvO_2$ = 101,4 ±20,66 (intermediate) and svO_2 = 100,8 ±7,09 (high grade) before exercise. After locomotion, the arterial and jugularvenous sO_2-values decreased in all 3 groups.

The oxygen saturation graph showed a significant leftshift (pa50 = 3,4 ±0,21 kPa (low-grade), pa50 = 3,3 ±0,23 kPa (intermediate) and pa50 = 3,2 ±0,23 kPa (high grade) for the diseased groups compared with the healthy clients (pa50 = 3,7 ±0,18 kPa) in rest. Corresponding pjv50-values showed that not all of the respiratoryly diseased groups (low-grade 3,0 ±0,26 kPa, intermediate 3,0 ±0,15 kPa, high grade 3,3 ±0,36 kPa) decreased significantly below the results of the healthy group immediately after exercise. This left-shift of the O_2-saturation graph cannot be explained with the “Bohr-effect”.

Blood lactate of horses suffering from different degrees of the respiratory disease did not show significant differences (blood-[lactat]: 0,8 ±0,15 mmol/l (low-grade), 0,9 ±0,18 mmol/l (intermediate) and 1,1 ±0,38 mmol/l (high grade)) at rest. Results increased slightly immediately after locomotion (blood-[lactat]: 0,8 ±0,37 mmol/l (low-grade), 1,1 ±0,33 mmol/l (intermediate) and 2,1 ±1,87 mmol/l (high grade)). In summary our exercise model did not show any effects due to lack of oxygen in horses with respiratory partial- ($\Downarrow paO_2$, $\Rightarrow paCO_2$)

and global-insufficience ($\Downarrow paO_2$, $\Uparrow paCO_2$). There was no clear hyperlactatemia or lactacidemia after defined exercise.

Reduced pulmonary function with a reduction of blood gas pressure leads to compensatory mechanisms (cardiovascular, erythrocytic and respiratory) such as an increase in blood flow and the number of erythrocytes, as well as their affinity for O_2. Possibly the three relative independent functional mechanisms (cardiovascular, erythrocytic and respiratory) have a different importance for maintaining O_2 accommodation in the organism. According to our investigations and some sources of literature, we think that reduction in pulmonary function (hypoxemia, hypercapnie) has less consequences for the oxygen supply of the body periphery than a limitation in the function of cardiovascular determinants (ischaemia).