The use of the standard exercise test to establish the clinical significance of mild echocardiographic changes in a Thoroughbred poor performer

C Meyer[a*], R Gerber[b] and A J Guthrie[c]

ABSTRACT

A 4-year-old Thoroughbred gelding racehorse was referred to the Onderstepoort Veterinary Academic Hospital (OVAH) with a history of post-race distress and collapse. In the absence of any obvious abnormalities in the preceding diagnostic work-up, a standard exercise test was performed to determine an underlying cause for the post-race distress reported. In this particular case oxygen desaturation became evident at speeds as slow as 6 m/s, where PO_2 was measured at 82.3 mm Hg. Similarly at a blood pH of 7.28, PCO_2 had dropped to 30.0 mm Hg indicating a combined metabolic acidosis and respiratory alkalosis. The cause of the distress was attributed to a severe hypoxia, with an associated hypocapnoea, confirmed on blood gas analyses, where PO_2 levels obtained were as low as 56.6 mm Hg with a mean PCO_2 level of 25.4 mm Hg during strenuous exercise. Arterial oxygenation returned to normal immediately after cessation of exercise to 106.44 mm Hg, while the hypocapnoeic alkalosis, PCO_2 25.67 mm Hg, persisted until the animal's breathing normalized. The results obtained were indicative of a dynamic cardiac insufficiency present during exercise. The combination of an aortic stenosis and a mitral valve insufficiency may have resulted in a condition similar to that described as high-altitude pulmonary oedema, with respiratory changes and compensation as for acute altitude disease. The results obtained were indicative of a dynamic cardiac insufficiency present during exercise and substantiate the fact that an extensive diagnostic regime may be required to establish a cause for poor performance and that the standard exercise test remains an integral part of this work-up.

Key words: aortic stenosis, desaturation, mitral valve insufficiency, pulmonary oedema, racehorse, standard exercise test, Thoroughbred.

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INTRODUCTION

Poor performance related to metabolic disturbances, while often implicated, may be difficult to prove conclusively because of the lack of objective criteria on which to base a diagnosis. The use of a standard exercise test in conjunction with additional tests, such as endoscopy and telemetric echocardiography performed during exercise, can provide information relating to cardiopulmonary and subsequent metabolic function.

CASE HISTORY AND DIAGNOSTIC FINDINGS

A 4-year-old Thoroughbred gelding racehorse was referred to the Onderstepoort Veterinary Academic Hospital (OVAH), South Africa, with a history of poor performance and post-race distress, with collapse noted on 2 separate occasions. On admission the horse appeared in good body condition. A complete blood count with analysis of selected serum indices was performed with no significant abnormalities noted. Faecal and urine analyses were also normal. Similarly, no significant abnormalities were detected during a lameness evaluation at the walk or trot on different surfaces . The heart rate, respiratory rate and rectal temperature were all within normal limits.

A resting ECG revealed a physiological second-degree atrioventricular block. No further abnormalities in the rate, rhythm and regularity of the heart were noted at rest or, by means of a telemetric ECG, during exercise. A grade 1 murmur of both the mitral and tricuspid valves could be auscultated at rest. Echocardiography confirmed a mild mitral and tricuspid valve insufficiency. Heart size, wall thickness and contractility all appeared to be within normal limits. An increase in the velocity of flow (265 cm/sec; reference range 72–130 cm/sec[15]) was detected at the aortic valve during early systole, indicative of a mild stenosis.

Endoscopy of the upper airways was performed at rest and during maximum exercise. Mild asymmetry of the left laryngeal arytenoid cartilage that disappeared during exercise and mild, chronic pharyngeal lymphoid hyperplasia were also noted. No significant disturbances of either the anatomical or the dynamic positioning of the upper respiratory tract were noted during exercise. In the absence of any obvious pathological abnormalities on the initial diagnostic assessment, a standard exercise test was performed.

The horse was trained over approximately 1 week to run on the treadmill. A catheter was placed in the transverse facial artery to collect arterial blood for evaluation of gaseous exchange during exercise. Venous blood, while appropriate for measuring acid-base status, cannot be collected as an alternative for the determination of the oxygen status of an animal during exercise[7]. During the standard exercise test, oxygen desaturation became evident at speeds as slow as 6 m/s, where PO_2 was measured at 82.3 mm Hg. Similarly at a blood pH of 7.28, PCO_2 had dropped to 30.0 mm Hg (reference range >40.6 mm Hg[3]), indicating a mixed metabolic acidosis and respiratory alkalosis. At a speed of 11 m/s, PO_2 levels obtained were as low as 56.6 mm Hg (reference range >70 mm Hg during maximal exercise[9]) with a mean PCO_2 level of 25.4 mm Hg. At this stage the horse showed obvious signs of distress and marked hyperventilation. The cause of the distress was attributed to a severe hypoxia, with an associated hypocapnoea[3]. Arterial oxygenation returned to normal (PO_2 106.44 mm Hg) immediately after cessation of exercise, while the hypocapnoeic alkalosis (PCO_2 25.67 mm Hg) persisted until the animal's breathing became normal.

In addition to the blood gas analyses performed, further assessment of cardiovascular response to exercise was studied

[a]Equis Veterinary Practice, PO Box 2155, Hillcrest, 3650 South Africa.

[b]Department of Animal Production, Tshwane University of Technology, Private Bag X680, Pretoria, 0001 South Africa.

[c]Equine Research Centre, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

*Author for correspondence. E-mail: bonzi@mweb.co.za

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by means of pulmonary arterial pressure measurements taken during exercise. These measurements were obtained using an intravascular transducer placed in the left jugular vein and passed through the left atrium and left ventricle into the pulmonary artery. The position of the catheter was verified by the presence of typical pulmonary arterial pressure tracings on the chart recorder[9]. In this case the mean pulmonary artery pressure was recorded at 120 mm Hg during maximal exercise when the heart rate averaged approximately 200 beats/minute. Pulmonary arterial pressures have been measured in resting, as well as in submaximally and maximally exercising horses. Results obtained showed a mean pressure recording of 26.40 ± 3.83 mm Hg at rest with a 30 % increase during submaximal exercise, followed by a pressure plateau of approximately 80 mm Hg during maximal intensity exercise at a heart rate of approximately 200 beats/minute[7,5,12]. In this case, the significant increase in the mean pulmonary arterial pressure can potentially be attributed to a hypoxic pulmonary vasoconstriction[5,21] and/or a substantial increase in transmural pressure throughout the lungs due to a mitral valve insufficiency in combination with mild aortic stenosis. Mean pulmonary arterial pressures also returned to normal values immediately after cessation of exercise. The results obtained were indicative of a dynamic cardiac insufficiency during exercise. It was concluded that the horse would be unlikely to compete successfully and further cardiovascular decompensation could be expected with age and the animal was consequently euthanased.

At necropsy there was a mild increase in the amount of pericardial fluid as well as mild thickening of the right and left atrioventricular valves. No lesions were detected histopathologically in the myocardium. Histopathology performed on the lungs revealed a moderate, diffuse bronchitis and peribronchitis characterized by a mixed inflammatory infiltrate consisting of lymphocytes, plasma cells and eosinophils. No pathogens were detected in association with these lesions, which appeared to have an allergic origin and may have been indicative of early stages of chronic obstructive pulmonary disease. No other significant lesions were detected.

DISCUSSION

The majority of Thoroughbred horses in training with reported poor performance and time off from racing are primarily diagnosed with disorders of the musculoskeletal system[7]. Additional causes contributing to a decreased performance include upper and lower respiratory tract disorders[20]. By contrast, poor performance related to metabolic disturbances may be difficult to identify and in these instances the use of a standard exercise test in conjunction with additional tests, such as endoscopy and telemetric echocardiography performed during exercise, can provide a range of information relating to cardiopulmonary and metabolic function[1,7,10,20].

Once an animal is able to run confidently at the front of the belt at high speed, the standard exercise test can be completed in a single time period[7,20]. The standard operating procedure for the test consists of an initial warm-up period followed by incremental increases in velocity over 1-minute intervals[7,20]. The number of incremental steps completed gives an indication of the horse's ability to perform maximally and also provides an indication of whether an individual's performance is comparable to other horses under the same conditions[7,19,20]. The test is also performed with the treadmill at an incline to increase the intensity of the exercise without the need to attain excessive velocities. The test is terminated when the horse shows signs of exhaustion and is no longer able to maintain its position at the front of the treadmill without aid[19].

In normal, healthy horses at sea level, arterial oxygen and carbon dioxide blood gas tensions have a mean value of approximately 100 mm Hg and 45 mm Hg respectively, at rest[7]. Arterial oxygen blood gas tension levels fall (±70 mm Hg) while PCO_2 levels increase (>48 mm Hg) during maximal exercise intensities[3,7,11,21]. In Thoroughbred horses, and similarly in man, high-intensity or maximal exercise bouts are therefore characterized by hypoxia, hypercapnoea and a metabolic acidosis[11]. The metabolic acidosis is due to an efflux of lactate from working muscles into the circulation, the production and release of which is exponentially proportional to an increase in exercise intensity[6,8].

The reasons for the exercise-induced hypoxia, associated with an apparently paradoxical hypercapnoea in the absence of severe respiratory disease, are unclear. As exercise-induced arterial hypoxaemia and hypercapnoea are closely related to exertion level, several physiological mechanisms have been proposed for this phenomenon. These include speculations that increased arteriovenous shunts develop during maximal intensity exercise, with resultant ventilation perfusion mismatching[1,7,18]. This condition is further exacerbated by an increase in the blood-alveolar diffusion distance due to the development of a transient interstitial pulmonary oedema. Marked hypoventilation occurs and the simultaneous high cardiac output at exercise, may result in a pulmonary capillary transit time which is insufficient for complete equilibration of oxygen across the respiratory membrane, resulting in a combination of factors culminating in inadequate oxygen uptake and removal of CO_2 by the exercising animal[1,2,7,10,17,18,21]. It should be noted that in some studies in humans, supreme athletic individuals exercising at supramaximal intensities exhibit a hypocapnoea secondary to hyperventilation[11]. This is most likely due to the activation of the medullary respiratory centre by extreme hypoxia. There is, however, no respiratory response to hypoxia until PO_2 reaches 60 mm Hg or less with PCO_2 accumulation providing the stimulation for normal respiratory drive[3,21]. Although it is not clear at which capillary blood levels of PO_2 the central circulation becomes impaired, extreme hypoxia is incompatible with normal neural activity. In military aviation medicine studies, it has additionally been shown that hypoxia associated with hypocapnoea may increase the severity of temporary or permanent mental retardation when compared with hypoxia associated with eu- or hypercapnoea[4].

The clinical findings in this case are comparable to those seen with an acute ventilatory response to high altitude. Under these conditions the peripheral chemoreceptors, dominated by the carotid body, become stimulated by a fall in ambient and inspired PO_2[7,21]. The immediate ventilatory response, an acute increase in ventilation, is thus triggered by the decrease in arterial PO_2. While this hyperventilation has a beneficial effect on oxygenation, this effect is limited as a potentially adverse hypocapnoea and alkalosis rapidly develop. Initially the problem is resolved by a compromise where the increased ventilation does not adequately replace depleted oxygen, but the acid-base disturbance that results, is considerably less than might occur with a full ventilatory response[3,21]. A rare, far more life-threatening disorder, typically associated with acute altitude sickness, has also been recorded. This syndrome of high-altitude pulmonary oedema, has been described more commonly in males, although an underlying or predisposing factor has not yet been identified. This condition is characterized by an increase in pulmonary arterial pressure and pulmonary oedema. It has been suggested that high pulmonary arterial pressure can lead to changes in transmural pressures with leakage of fluid across membranes

and subsequent perivascular oedema formation and alveolar flooding[4].

The combination of the aortic stenosis and the mitral valve insufficiency in this case may have resulted in a condition similar to the high-altitude pulmonary oedema described, with respiratory changes and compensation as for acute altitude disease. The tricuspid valvular insufficiency, has been described as a concurrent abnormality in many horses suffering from mitral valve insufficiency[11,14,16], with the resultant pulmonary regurgitation during systole most likely exacerbating the clinical condition. While congestive heart failure is the most common clinical presentation in these cases, it is not inconceivable that a mild congenital, or acquired, cardiac anomaly may be overlooked initially, only to present at a later date as congestive heart failure. This may be particularly true of horses not required to perform maximally at an early age.

Although there was evidence of mild COPD on histopathological examination of the lungs, studies performed on horses suffering from this condition failed to show any significant decrease in PCO_2 at any stage of the gallop[13]. In addition to the less efficient alveolar ventilation during exercise, these animals also show decreased resting PO_2 levels when compared to normal subjects[13]. By contrast, this case showed no abnormalities in arterial O_2 or CO_2 content at rest and the pulmonary changes described at *post mortem* were likely secondary to inhibition of pulmonary clearance mechanisms during exercise, and not clinically significant at the time of investigation.

The aim of the standard exercise test is to evaluate metabolic parameters, which may predispose to exercise intolerance and poor performance. It may also assist in the evaluation of findings, such as the heart murmurs described in this case, where the initial clinical significance of these findings might be inconclusive.

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