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No Ventilatory Response to CO_2 in Thoroughbreds Galloping at 14 m s^{-1}

G. L. LANDGREN,[1] J. R. GILLESPIE and D. E. LEITH

[1]*RR 2, Box 54, Sheyenne, ND 58374 and Department of Clinical Sciences, College of Veterinary Medicine, Kansas State University, Manhattan, KS 66506, USA*

ABSTRACT We wondered why horses hypoventilate during heavy exercise. We measured ventilatory responses to inspired CO_2 (0, 3 and 6%) concentration in 6 Thoroughbred horses (515 ± 50 kg) standing and galloping on a horizontal treadmill. At rest, minute ventilation ($\dot{V}_E$, BTPS) increased by 35 l min^{-1} $torr^{-1}$ ($PaCO_2$ 42–46 torr) from a mean of 68 l min^{-1} (air) to 213 l min^{-1} (6% CO_2). At 10 m sec^{-1}, $\dot{V}_E$ increased by 14 l min^{-1} $torr^{-1}$, from 1 300 l min^{-1} (air) to 1 460 l min^{-1} on 3% CO_2 ($PaCO_2$ 43–55 torr), with no further change on 6% CO_2 ($PaCO_2$ 55–66 torr). At 14 m s^{-1}, $\dot{V}_E$ remained at 1 800 l min^{-1}, though $PaCO_2$ rose (50 to 80 torr). Perhaps ventilatory drive sometimes does not increase with $PaCO_2$. Alternatively, perhaps ventilation is sometimes independent of changes in drive, influenced by the relation between power available for breathing and power required; both might be speed-dependent if strong mechanical links exist between breathing and locomotion. We cannot distinguish among these mechanisms.

Key words: Horses; hypercapnia; control of breathing; maximum ventilation.

INTRODUCTION

Many horses become hypercapnic during heavy exercise while most humans do not. Is this apparent hypoventilation a feature of their control of breathing, or does exercise hyperpnea approach mechanical limits to ventilation in horses? We reasoned that if ventilation were to increase in response to added CO_2 during exercise, then the baseline hypoventilation could be attributed to control and we could rule out mechanical limits. To see if that occurred, we measured ventilatory responses to added CO_2 in exercising horses.

METHODS

Animals. Six healthy Thoroughbred geldings weighing 515 ± 50 kg (mean ± SD; range 450–590 kg) were trained to run at speeds up to 14 m s^{-1} (85% $\dot{V}O_2$max, approximately) on a treadmill (Sato, Uppsala). Each had previously had its left carotid artery surgically relocated beneath the skin.

Ventilation. A mask was secured over the horse's nose and mouth and sealed by inflating an inner tube. Two centrifugal blowers in series pulled a bias flow of air through the mask via an open system (Fig. 1) made with 15 cm inner diameter ducts. Bias flow varied during the breathing cycle (i.e. the blowers were not a constant-flow source) so it was necessary to calculate flow at the airway opening ($\dot{V}ao$) as the instantaneous difference between flows measured upstream ($\dot{V}in$) and downstream ($\dot{V}out$) from the mask. $\dot{V}ao$ was integrated for V_T. Fleisch-type flow meters (Meriam Instrument Co., Cleveland, OH) were used located 5 m from the mask. A small pair of flow meters (15 cm dia) and low bias flows (< 20 l s^{-1}) were used for measurements at rest, and larger meters (25 cm dia) and higher bias flows (120–170 l s^{-1}) were used during exercise. The larger flow meters were mounted between conical

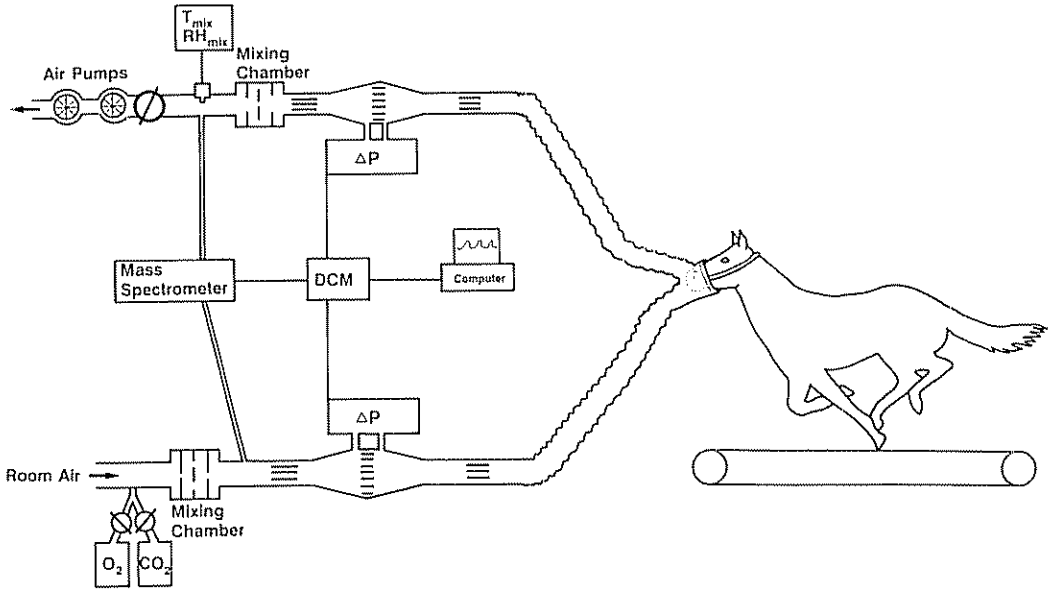

Fig 1 Schematic of the experimental setup. DCM = data collection module. Tmix, RHmix = temperature and relative humidity of mixed expired and bias flow gas. ΔP = pressure difference across pneumotachograph.

sections that tapered from 25 to 15 cm over a length of about 1 m.

The flow meters, mounted in their inlet and outlet sections, were individually calibrated out of the system by pulling a range of steady flows through them from the room via a 5 cm ASME standard nozzle, while pressure drop across the nozzle was measured with a micromanometer (34FB2 TM, Meriam Instrument Co, Cleveland, OH). From that pressure drop flow was calculated using standard equations provided with the nozzle. Pressure drops across the flow meters were measured with differential transducers (PX 163-005BD 5V, ±12 cmH_2O, Omega Engineering, Stamford, CT). The flowmeters' pressure-flow curves were linear within 2% over the range of use. These transducers' calibrations (output vs pressure) were checked daily with the micromanometer. The system's frequency responses were assumed to be adequate. Signals were stored in a data collection module (DCM, MRG Aspect, Freedom, NH) during experiments and later transferred to a host computer for analysis using commercial software (DADiSP Worksheet, DSP Development Corp, Cambridge, MA).

During experiments, mean $\dot{V}out$ often exceeded mean $\dot{V}in$ by about 5% of the bias flow, with resulting apparent excess of expired over inspired $\dot{V}ao$, V_T, and $\dot{V}_E$. We had to estimate the sources of those differences and then deal with them. Variability in the calibrations of the two flow meters presumably contributed small differences randomly distributed about zero. Temperature and composition of expired gas (mixed with bias flow gas) differed from those of inspired gas, which could make $\dot{V}out$ exceed $\dot{V}in$ by no more than 2%, because the flow of actual expired gas (minute ventilation) was not more than 20% of the total (bias) flow. Greater differences presumably indicated the presence and magnitude of inward leaks between the two flow meters, and introduced an error in the baseline (zero) value of $\dot{V}ao$ that had to be eliminated. This was done in the following way.

During computer analysis of experimental

data, a single overall mean value for $\dot{V}in$ and another for $\dot{V}out$ was established over the entire duration of each record section studied. When those means differed, $\dot{V}out$ was offset to make them equal. For establishing the 'zero' (reference) flow for $\dot{V}ao$, it made no difference which flow signal was offset, nor how much offset was necessary. The procedure had the further effect, however, of setting mean inspired and expired V_T equal, thus overestimating inspired V_T (ATPH where H is humidity) and underestimating expired V_T (BTPS, influenced by the respiratory exchange ratio, R) by half of the true difference between them, perhaps as much as 4% each. Mean V_T and respiratory frequency (fr) were calculated from samples usually of 5 (at rest) or 10 (during exercise) sequential breaths. We treated this as inspired V_T (ATPH) and corrected to BTPS using the same ambient (inspired) temperature and humidity values (24°C and 11 torr PH_2O) for all data. Thus V_T (BTPS) was presumably overestimated by the same fraction as inspired V_T, up to about 4%. Minute ventilation was calculated as V_T (BTPS) × fr; we use the symbol $\dot{V}_E$, acceptable because our approach sets inspired and expired ventilations equal.

Mask pressures varied with conditions; at the highest bias flows, mean values were about −8 cm H_2O and we estimate swings no greater than +6 and −4 cm H_2O around that mean during breathing. The mask and these substantial breathing loads probably influenced breathing, but not, it is thought, in ways to change our interpretations.

$\dot{V}O_2$ and $\dot{V}CO_2$ during air breathing The stream of bias flow and expired gases was mixed (in baffled 200 l barrels, one for measurements at rest and 6 in parallel during exercise) and sampled for measurement of 'mixed expired' O_2, CO_2, and N_2 fractions (F_EO_2, F_ECO_2, F_EN_2). These and inspired gas fractions (F_IO_2, F_ICO_2, F_IN_2) were measured with a mass spectrometer (Model 1100, Perkin Elmer, Pomona, CA). 'Mixed expired' gas temperature and humidity measured at the sampling site (Model 880 Thermohydrometer, General Eastern, Watertown, MA) and ambient pressure measured with a mercury barometer were recorded at the time of experiments. Mean $\dot{V}in$ (bias flow at the upstream flowmeter, not the animal's minute ventilation) was treated as V_E for calculation of $\dot{V}O_2$ and $\dot{V}CO_2$ (STPD) by standard equations;[12] $\dot{V}O_2$ and $\dot{V}CO_2$ might be underestimated by equal fractions, up to about 5%, as a result of leaks.

Arterial blood A 16 gauge teflon catheter (Delmed A-cath, Delmed, Inc., Canton, MA) was inserted in the left carotid artery. Blood collected through it in heparinized syringes was stored in ice water (≤1 h approx.) until analyzed for PaO_2, $PaCO_2$, and pHa (Model 213, Instrumentation Laboratories, Lexington, MA). Values were corrected to the temperature (Tpa) measured with a thermistor catheter (Columbus Instruments, Columbus, OH) passed into the pulmonary artery via the right jugular vein while the pressure measured through its lumen was monitored. Lactate concentration ([LA]a) in plasma from arterial blood was also measured (Mod 23L, Yellow Springs Instruments, Yellow Springs, OH).

CO_2 mixtures Inspired gas composition was regulated by adding O_2 and CO_2 to the bias inflow. The gases were mixed (3 baffled 200 l barrels in parallel) and sampled for measurement of gas fractions with the mass spectrometer. F_IO_2 was held at 20.95±0.1% and F_ICO_2 was set at 3, 6, or 9%.

Protocol Each horse was assigned 3 test days at least 3 days apart. On a test day, a horse breathed either room air or 3 or 6% CO_2, at rest and during exercise at 10 and 14 m s^{-1}. These speeds elicited O_2 consumptions approximating 55 and 85% of the maximum value for Thoroughbred horses in our laboratory (Table 1). Rest measurements were made after at least 2 min of breathing the desired gas, after which the horse warmed up on the treadmill for 2 min at 3 m s^{-1} while the inspired gas composition was adjusted at a higher bias flow rate. The treadmill was then brought up to the test speed and the first blood sample withdrawn

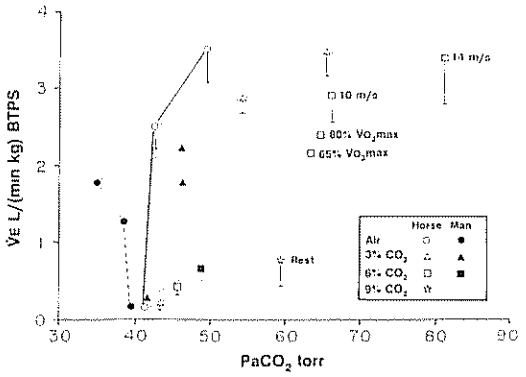

Fig. 2. Ventilatory responses to exercise (% $\dot{V}O_2$ max or running speed) and to inspired CO_2 in Thoroughbred horses ($n=6$) and human subjects.[6] Bars are ±1 SD.

over the first 15 s. Samples were then withdrawn for 15 s at 30 s intervals for 3 min, by which time $\dot{V}O_2$ and $\dot{V}CO_2$ appeared to have plateaued. Data collected at 2 and 2.5 min were combined for further analysis. During a 30 min rest period between runs the horse was walked about 500 m to a scale for weighing. Measurements were made on a separate day while horses breathed 9% CO_2 at rest.

Experimental design and analysis. A split plot design[11] was used. The whole plot (%CO_2) was in Latin square design with the subplot (speed) in a repeated measures design. Linear regression was used to fit ventilatory response curves to the data.

RESULTS

Air breathing. Mass-specific $\dot{V}O_2$ and $\dot{V}CO_2$, and [LA]a and pHa at the end of exercise, are compared in Table 1 with values for human athletes.[6] Ventilatory responses to exercise are compared in Fig. 2.

CO_2 breathing. Ventilatory responses to inspired CO_2 at rest and during exercise are compared with those of humans in Fig. 2. At rest, horses' $\dot{V}_E$ increased by 35 l min^{-1} torr^{-1} ($PaCO_2$ 42–46 torr) from a mean of 68 l min^{-1} on air to 213 l min^{-1} on 6% CO_2. At 10 m sec^{-1}, $\dot{V}_E$ increased by 14 l (min torr^{-1}), from 1 300 l min^{-1} on air to 1 460 ($p<0.05$) on 3% CO_2 ($PaCO_2$ 43–55 torr), with no further change on 6% CO_2, though $PaCO_2$ rose to 66 torr. At 14 m sec^{-1}, $\dot{V}_E$ did not change from 1 800 l min^{-1} on air when CO_2 was added, despite a rise in $PaCO_2$ from 50 to 80 torr.

DISCUSSION

While breathing air and galloping at 14 m s^{-1}, our Thoroughbred horses hypoventilated by two criteria: first, mean $PaCO_2$ at this speed was higher than the value at rest (50 vs 42 torr), and second, they failed to hyperventilate in response to a mild metabolic acidosis (Base Excess about −3 meq l^{-1}).[1,4,9] To explore the mechanisms responsible for that hypoventilation, we turn first to the findings at 10 m s^{-1}.

Table 1. *Comparison during air breathing of 6 horses with human subjects*[6]

Mb = body mass. $\dot{V}O_2$max in these Thoroughbreds is about 150 ml min^{-1} kg^{-1} and that of the human subjects was about 62 ml min^{-1} kg^{-1}

	Horse	Human	Horse	Human
Speed, m s^{-1}	10	2.1	14	2.7
Grade, %	0	10	0	10
$\dot{V}CO_2$/Mb, ml min^{-1} kg^{-1}	70±9	40	131±24	51
$\dot{V}O_2$/Mb, ml min^{-1} kg^{-1}	79±8	42	128±20	49
% $\dot{V}O_2$max	~55	67±7	~85	79±8
[LA]a, mmol l^{-1}	2±1	5±1	13±5	9±3
pHa	7.47±0.02	7.36±0.02	7.31±0.06	7.31±0.03

We may assume that ventilation at 10 m s^{-1} was not maximal, even when inspiring CO_2, because greater ventilation was observed at 14 m s^{-1}. If that assumption is true, then for explanations of the small or absent response to increased $PaCO_2$ at 10 m s^{-1} we have to look to the behavior of the ventilatory controller, influenced of course by mechanical, chemical, and neural factors.[8,14]

But perhaps that assumption is false. Mechanical links between ventilation and locomotion in galloping horses have been postulated but remain to be demonstrated,[3,5] and if they exist, their nature and strength are unknown. Nevertheless, it is conceivable that tight mechanical links operate to set *speed-dependent* mechanical limits to ventilation in horses, so that perhaps maximal $\dot{V}_E$ is around 1 500 l min^{-1} at 10 m s^{-1} and about 1 800 l min^{-1} at 14 m s^{-1} in our horses. Such speed-dependent limits could arise if, at any given speed, fr were linked to fs and V_T to stride length in such a way that increasing either one markedly increased the power required for breathing (or some other relevant variable, e.g., mean muscle force of breathing), or decreased the power available, or both.

Adding 3% CO_2 to the inspired gas at 10 m s^{-1} caused a slight increase in $\dot{V}_E$, so there was an increase in ventilatory drive, and the horse's breathing apparatus was capable of responding to it; ventilation was not mechanically limited in horses breathing air at 10 m s^{-1}. But adding 6% CO_2 caused no further increase in $\dot{V}_E$. If we suppose that increases in $\dot{V}_E$ were still possible (because they were observed at 14 m s^{-1}), then either there was no increase in drive when $PaCO_2$ increased from about 55 to 66 torr, or drive increased but was offset by other factors, for example decreased functional capacities (e.g. fatigue) of the muscles used for breathing. The latter suggestion seems untenable under our protocol at 10 m s^{-1}, and because at 14 m s^{-1} greater ventilation is sustained for similar times under more severe conditions (higher $PaCO_2$ and lower pHa, e.g., on 6% CO_2). But the former suggestion (no change in drive), though we cannot rule it out, seems equally doubtful; again we have to entertain the possibility of speed-dependent mechanical limits to exercise ventilation in horses.

What can we say of the mechanisms responsible for hypoventilation in air-breathing horses at 14 m s^{-1}? Only that a higher $PaCO_2$ failed to elicit greater ventilation. We cannot say whether ventilatory drive failed to increase, or whether mechanical limits to ventilation, speed-dependent or otherwise (e.g. fatigue) now came into play. If, at this speed, these horses achieved higher ventilations with a different chemical stimulus (e.g. hypoxia), then neither ventilatory muscle fatigue nor a speed-dependent limit to ventilation could explain the hypoventilation. Demonstration of higher ventilation at still higher speed would leave open the possibility of a speed-dependent limit.

While breathing 6% CO_2 at 10 and 14 m s^{-1}, but never while breathing air, one of our horses (Snip) halved fr and doubled V_T.[7] Thus, while tight coordination persisted between breathing and galloping, the change in the pattern of coordination shows that the links between fr and fs, and between V_T and stride length, are not absolute. The same conclusion is reached by other experimental approaches.[2] We think this observation tells us two other things. First, it argues against very strong mechanical links between fr and fs and between V_T and stride length, because the animal continued to run at the same speed. Second, it shows that under these conditions, inhalation of 6% CO_2 did in fact influence the control of breathing.

These arguments do not rule out the possibility that ventilation was mechanically limited at 14 m s^{-1}. Mechanical limits to hyperpnea appear to operate in humans during maximum voluntary ventilation maneuvers, both brief and sustained. The responsible mechanisms set limits to both inspiratory and expiratory flow rates[10] so that, in effect, V_T becomes a dependent function of inspiratory and expiratory times. A consequence is that the maximum ventilation is relatively

insensitive to changes in fr; V_T automatically varies inversely with fr, leaving $\dot{V}_E$ essentially constant over a wide range of fr. Thus, we cannot rule out the possibility that Snip was ventilating maximally at both of the observed combinations of fr and V_T. We have no evidence, however, that both inspiratory and expiratory flows were maximal, and we see that V_T can vary at a given stride length; so evidence from these experiments does not establish the existence of speed-dependent limits to ventilation in galloping horses.

Finally, we comment on our methods. We think that leaks existed in the system despite our efforts to eliminate them, and that most of the excess of mean $\dot{V}$out over mean $\dot{V}$in that we often saw, varying from experiment to experiment, is best explained by inward leaks between the flow meters. We think the method used to offset them allowed us to calculate instantaneous $\dot{V}$ao correctly, with the reservation that it set aside real differences due to changes in respired gas temperature and composition and thus introduced errors in our calculated V_T and $\dot{V}_E$, reliably estimated at about +4%, independent of the magnitude of the leak and the associated offset of mean $\dot{V}$out. We are less confident about the magnitude of possible errors in $\dot{V}O_2$ and $\dot{V}CO_2$. If we had measured mean flow at the site where mixed expired gas was sampled, inward leaks would not have caused errors in calculations of gas exchange during air breathing. But because we used mean $\dot{V}$in, inward leaks downstream from that point presumably diluted the mixed expired gas, with resulting underestimation of calculated $\dot{V}O_2$ and $\dot{V}CO_2$ by about 5% if $\dot{V}$out reported most of the leaks. The errors would be greater if there were leaks into the system between the $\dot{V}$out flowmeter and the gas sampling point. The variability of our $\dot{V}O_2$ and $\dot{V}CO_2$ measurements should be high if variable leaks (and associated errors) were present.

In subsequent studies we have minimized leaks and both located and quantified them by measuring the progressive dilution of a tracer gas as a function of distance along the system, and measured gas exchange with more confidence using a third flow meter where mixed expired gas is sampled. In a study, a subset of 4 horses (also studied here) breathing air and galloping at 14 m s^{-1}, $\dot{V}O_2$ of 134±9 ml kg^{-1} min^{-1}) (mean and SD) was recorded.[13]

In summary, Thoroughbred horses breathing air hypoventilate while galloping at 14 m s^{-1} on a level treadmill. At 10 m s^{-1}, they increase ventilation slightly when F_ICO_2 is increased to 3%; we conclude that no mechanical limit to ventilation existed. No further increase in $\dot{V}_E$ is seen when F_ICO_2 is increased to 6%; this may reflect the behavior of the ventilatory controller and not ventilatory limitation, as greater ventilations are achieved at 14 m s^{-1}; but we cannot rule out the possibility of speed-dependent limits to $\dot{V}_E$ at both 10 and 14 m s^{-1}. At 14 m s^{-1}, there is no ventilatory response to added CO_2; at this speed we cannot distinguish among the several possible mechanisms for the hypoventilation observed when breathing air, or rule out a mechanical limit to $\dot{V}_E$.

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1 : 2 Ratio of Breathing to Stride Frequencies in a Galloping Horse Breathing 6 % CO_2

J. R. GILLESPIE, G. L. LANDGREN and D. E. LEITH

College of Veterinary Medicine, Kansas State University, Manhattan, KS 66506, USA

ABSTRACT. Galloping horses take one breath per stride; the reasons are unknown. To study them, we sought to change the relation of breathing and locomotion. We measured tidal volume (V_T) respiratory frequency (fr), and other variables, and noted fr : fs (stride frequency), in 6 Thoroughbreds at rest and galloping with inspired CO_2 fraction (F_ICO_2) = 0, 3, and 6%. At 14 ms^{-1} and constant fs, one horse halved fr and doubled V_T on 6% CO_2. Its [LA]a and [H^+]a were higher than those of others (20 vs mean 10 mM l^{-1} and 74 vs mean 62 nmol l^{-1} respectively), and it defended $PaCO_2$ better (increment 26 vs 33 torr). At fr : fs = 1 : 2, perhaps dead space ventilation was reduced. V_T was high (0.06 l kg^{-1}bw) so elastic breathing loads were likely great. Inertial mechanisms driving ventilation, if they exist, were overridden. This horse could change fr : fs and V_T : stride length, but fr and fs remained tightly linked; the mechanisms remain obscure.

Key words. Respiration; ventilation; control of breathing; exercise; hypercapnia; horses.

INTRODUCTION

Respiratory frequency (fr) of galloping horses is ordinarily entrained 1 : 1 with stride frequency (fs).[1-4] The mechanisms are unknown, though mechanical interactions between breathing and locomotion are likely to play a role.[2] It seems possible that such interactions could influence or limit ventilation and/or locomotion, perhaps contributing to the hypoventilation observed in galloping horses. To study the underlying mechanisms, we sought ways to change the relation of fr and fs in galloping horses. We thought that high inspired fractions of CO_2 (F_ICO_2) might increase the chemical drive to breathing and cause an increase in ventilation at the same speed. That would require an increase in fr and/or tidal volume (V_T) and necessitate a change in fr : fs and/or V_T : stride length; i.e. it would show the possibility of 'uncoupling' fr from fs and/or V_T from stride length and further show that baseline V_E was not maximal.

MATERIALS AND METHODS

Animals and methods are described elsewhere.[4] We studied 6 healthy Thoroughbred horses (515 ± 50 kg mean ± SD, range 450–590), at rest and galloping at 10 and 14 m s^{-1} (approximately 55 and 85% of $\dot{V}O_2$-max for Thoroughbreds in our laboratory) on a level treadmill (Säto, Uppsala) with F_ICO_2 = 0, 3, 6%.

We added O_2 and CO_2 to the inspired gas to vary F_IO_2 while holding F_ICO_2 constant at 0.21, and measured the composition of inspired and mixed expired gases with a mass spectrometer (Model 1100, Perkin Elmer, Pomona, CA). Barometric pressure and the temperature and humidity of the room and mixed expired gases were measured.

Centrifugal blowers drew a bias flow of the desired gas mixture (about 20, 120, and 170 l s^{-1} at rest and at 10 and 14 m s^{-1}, respectively) through a mask sealed over the horse's nose and mouth. Mask pressures varied with conditions; at 14 m s^{-1}, swings of