Flowmetric comparison of respiratory inductance plethysmography and pneumotachography in horses

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These more sensitive diagnostic tests have facilitated earlier interventions, which theoretically should reduce or prevent the decline in lung function that can be seen in older horses. Unfortunately, even these newer tests, which are more sensitive, have not been adapted for field application and require significant energy supplies. Evaluation of lung function in the animal's natural setting would empower the veterinarian to detect these early changes in phase angle (i.e., thoracoabdominal asynchrony) derived from RIP could be employed as a measure of airway obstructions in humans (1, 10, 29). 

Previously, our laboratory considered the use of respiratory inductance plethysmography (RIP) for field application (23), because previous work suggested that relative changes in phase angle (i.e., thoracoabdominal asynchrony) derived from RIP could be employed as a measure of airway obstructions in humans (1, 10, 29). In contrast to these earlier studies, our laboratory found in horses that, despite remarkable changes in

INFLAMMATORY AIRWAY DISEASES and more severe recurrent airway obstructions are common clinical problems encountered in veterinary medicine (27, 32). Clinical examination is limited in sensitivity (28); therefore, more objective tests have been sought to improve early detection of these exercise-limiting problems. In contrast to human medical practice, in which lung function tests are employed routinely, the complexity, invasiveness, and limited sensitivity of conventional lung function tests performed during tidal breathing have hampered more widespread use of these in animals. Recent progress in the development of lung mechanical function tests, however, has been made on two fronts: 1) the application of a forced expiratory maneuver (6 or 2) forced oscillatory mechanics, which provide information concerning frequency dependence of resistance (22, 23). Both methods have been employed during histamine challenge, providing further information on airway reactivity and improving sensitivity to detect airway obstruction at a very early stage (6, 12, 13).

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breathing pattern and phase shifts in rib vs. abdominal displacement, the severity of both phase-shift and airway obstruction did not correlate sufficiently for diagnostic use (23). This was also evident in earlier studies in humans with chronic obstructive pulmonary disease (29). Furthermore, RIP, being a plethysmographic measurement, is prone to errors in the measurement of volume or flow, because of gas compression or rarefaction during obstructions (15, 17, 21). That volume displacement at the airway opening and thorax differ during obstruction served as a basis for unique indexes of airway obstruction in animals measured from double-chamber plethysmography (8, 18, 26, 31). This concept was also applied to the development of barometric whole body (single-chamber) plethysmography (3–5, 9, 11).

In this paper, we explore an analogous technology for use in large animals. For practical reasons, we chose boxless plethysmography for large animals (RIP) and compared the flow-derived indexes from RIP (volume, flow) with flow-derived measurements at the airway opening, which were measured using pneumotachography. Although there are clear differences between RIP and body plethysmography, we hypothesized that compression and rarefaction of gas due to airway obstruction would create similar phase and magnitude shifts that could be quantified.

A comparison between RIP and nasal flow was made in three settings: 1) histamine bronchoprovocation, 2) severe lower airway obstruction before and after bronchodilation, and 3) during experimentally induced hyperpnea in nonobstructed horses. Settings 1 and 2 were used to test the hypothesis that this new method of measurement gives similar results to conventional mechanics, and setting 3 was employed to examine the effects of increased respiratory frequency or flows in nonobstructed horses, as would occur during stress, excitement, and exercise (i.e., situations that might confound the measurements of airway obstruction).

**MATERIALS AND METHODS**

All procedures described were approved by the Institutional Animal Care and Use Committee at Tufts University.

**Details of the bench-top models of the test device.** The frequency response of the flow derived from RIP ($V_{dim}$) and that of flow derived from pneumotachograph ($V_{pn}$) signals were compared using a step test and an oscillatory model. For the step test, one inflated balloon (20-liter internal volume) was attached to one side of the pneumotachograph; the other side was closed with a rubber seal. The 11th intercostal space (Rib) and 18th rib (abdominal (Abd)) sensors were secured around the balloon. To perform the test, the rubber seal was pierced, and the time delay between $V_{dim}$ and $V_{pn}$ to reach a flow of 40 l/s (i.e., higher than the maximal flow encountered in our study) was measured for three runs. The average time delay was 12 ms. A second system was employed to examine potential phase differences between $V_{pn}$ and $V_{dim}$ during oscillation. This system employed a two-element (resistance and elastance) series model of the horse’s respiratory system. The resistance element was a 0.4-m noncompliant length of polyvinyl chloride (7.2-cm ID) attached to an elastic element, a large animal anesthesia bag (30 liters). The pneumotachograph was positioned at the proximal end of the tubing, and the inductance bands were wrapped in parallel around the spherical anesthesia bag, located at the distal end of the model. The model was oscillated (0.25–4.0 Hz) using constant end-expiratory pressure (2–5 cm H₂O), with either a mechanical ventilator (Bear 1, model BV-512, Bourns Medical Systems, Riverside, CA), or forced oscillatory mechanics delivery system (On the Nose, Scientific Solutions, Eden Mills, Ontario).

**Calibration, signal acquisition, and signal processing.** The pneumotachograph (Fleisch no. 5, OEM Medical, Lenoir, NC) was calibrated using a precision syringe (3-liter volume syringe, Hans Rudolph, Kansas City, MO). The pneumotachograph was connected via tubing to a differential pressure transducer (DP45–14, Validyne Engineering) and carrier demodulator amplifier. An esophageal balloon catheter was placed to the level of the mid thorax and connected to a differential pressure transducer (DP45–28, Validyne Engineering) and amplified. The opposite pole of the pressure transducer was connected to a side port in the gas-collection mask to obtain transpulmonary pressure measurements.

For calibration of RIP, an oscillator (large-animal oscillator, Ambulatory Monitoring, Sawmll, NY) was used, the signal from which was demodulated downstream using standard diagnostic hardware (Respitrace Interface, Ambulatory Monitoring, Saw Mill, NY). The sensitivity of the two RIP sensors (Rib and Abd bands) was made equal by adjusting their analog gain settings while stretching them dynamically (0.25–0.5 Hz) off the horse to identical lengths. This required a system of hangers, and their equivalence was later confirmed by placing them as close as possible to each other on a horse.

The Rib and Abd volume signals were summed to obtain a third analog volume signal, Sum (RIP interface, Ambulatory Monitoring). The three analog signals that were derived from these sensors (Sum, Rib, and Abd) were digitized (30 Hz; ADAPC, Buxco Electronics, Sharon, CT), displayed, and recorded on a personal computer by using data-acquisition software (XA BioSystem, Buxco Electronics). We differentiated the Sum signal to obtain Sum flow ($V_{dim}$) and applied smoothing (50 ms) to $V_{dim}$ and $V_{pn}$ equally.

The waveforms ($V_{dim}$ and $V_{pn}$) acquired in horses in each of parts I–III were analyzed post hoc using commercial software (AcqKnowledge, BIOPAC Systems). The RIP volume signal ($V_{dim}$) was calibrated to the inspired volume recorded from the pneumotachograph by adjusting the gain setting of $V_{dim}$ (volume) signal post hoc to correct for the difference in their amplitudes by using a multiplicative constant. The rationale to calibrate the Sum signal to $V_{pn}$ during inspiration was to normalize the signal to correct tidal volume ($V_T$) while tracking the dynamic events that represent compression or rarefaction. This permitted within- and between-subject comparisons. After calibration of the Sum signal, this signal was differentiated to obtain $V_{dim}$. Next, the two flow waveforms (i.e., $V_{dim} − V_{pn}$ flow) were digitally subtracted to obtain a third waveform that represented the dynamic differences between these flows, which was subsequently analyzed. The following variables were derived from the final subtracted waveform (see Fig. 2): 1) peak of the subtracted waveform during exhalation (SFE$_{max}$) and during inhalation (SFI$_{max}$); and 2) the integral of the subtracted waveform during the first 25% of exhaled volume (SFE$_{1_{25}}$) and first 25% of inspired volume (SFI$_{1_{25}}$). For calculation of these indexes, the beginning of inspiration and expiration was defined by the upward and downward directed zero crossings of the $V_{dim}$ signals, respectively.
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RESULTS

Bench-top models. In the step test, the time delay between \( V_{d\text{Sum}} \) and \( V_{pn} \) to reach a flow of 40 l/s in three successive runs was 11, 12, and 14 ms. In the oscillatory model, there was a slight phase delay between the \( V_{pn} \) and \( V_{d\text{Sum}} \) that varied nonlinearly with frequency (Fig. 1). There was a decay in peak amplitude of the \( V_{d\text{Sum}} \) signal with frequency, with the greatest changes appearing at frequencies of 3 and 4 Hz.

Part I in vivo: Effects of histamine-induced bronchoconstriction of horses. None of the horses reacted adversely to placement of the inductance bands, either before or after sedation with xylazine. Histamine aerosols altered the waveforms of \( V_{d\text{Sum}} \) in relation to \( V_{pn} \), in that the peak \( V_{d\text{Sum}} \) increased in relation to \( V_{pn} \) during the early portion of expiration, resulting in large increases in the subtracted \( V_{d\text{Sum}} - V_{pn} \) waveform (Fig. 2). As a result, the test variables (\( SF_{\text{Emax}}, SF_{\text{Fmax}}, SF_{\text{Ein}}, \) and \( SF_{\text{Iin}} \)) were altered in relation to histamine dose. Seven out of eight horses given histamine aerosol responded with a decrease in Cdyn and increases in RL and \( \delta P_{\text{tpmax}} \). In horse 8, there was minimal response to histamine other than tachypnea and coughing; therefore, this horse was not included in the comparison between test and conventional variables. In all of the seven remaining horses, \( SF_{\text{Emax}} \) increased with increased histamine dose (Fig. 3). In six of seven horses, there was an increase in \( SF_{\text{Ein}} \), but, in one horse with a tachypneic response, \( SF_{\text{Ein}} \) decreased at the highest histamine dose, after initially increasing as in the other horses. There were highly inconsistent changes observed for \( SF_{\text{Fmax}} \) and \( SF_{\text{Iin}} \) with increases and decreases as a result of histamine exposure. There was a significant correlation \( (r_s = 0.929, P < 0.001) \).
between the log dose of histamine that decreased Cdyn by 35% and the log dose of histamine that increased SFEmax by 35% (Fig. 4).

Part II in vivo: Bronchodilation of horses with heaves. All horses in this category presented with R L, Cdyn, and dPtpmax values compatible with severe, recurrent airway obstruction (22) (Fig. 5). The administration of albuterol aerosol caused significant decreases in RL and dPtpmax and an increase in Cdyn (Fig. 5) within 5 min. Before bronchodilation, horses with heaves showed marked differences between VdSum and Vpn, particularly in the early portion of expiration. The subtracted waveform was characterized by large, positive expiratory spikes and smaller inspiratory spikes in the negative direction (Fig. 6). Bronchodilation reversed these qualitative changes, accompanied by significant (P < 0.005) decreases in SFEmax and SFEmax. There were highly significant (P < 0.005) correlations between the test variables and RL or dPtpmax when pre- and postbronchodilator values were pooled (Fig. 7). However, there was only a trend for the correlation between Cdyn and SFEmax (r = -0.49, P = 0.054) and no significant correlation between Cdyn with SFEmax (r = -0.38, P = 0.14). Furthermore, there were no significant correlations between the test variables and VT (vs. SFEmax: r = -0.3, P = 0.25; vs. SFEmax: r = 0.1, P = 0.71) or frequency (vs. SFEmax: r = 0.26, P = 0.32; vs. SFEmax: r = 0.02, P = 0.95). Bronchodilation reversed these qualitative changes, accompanied by significant (P < 0.005) decreases in SFEmax and SFEmax. There were highly significant (P < 0.005) correlations between the test variables and RL or dPtpmax when pre- and postbronchodilator values were pooled (Fig. 7). However, there was only a trend for the correlation between Cdyn and SFEmax (r = -0.49, P = 0.054) and no significant correlation between Cdyn with SFEmax (r = -0.38, P = 0.14). Furthermore, there were no significant correlations between the test variables and VT (vs. SFEmax: r = -0.3, P = 0.25; vs. SFEmax: r = 0.1, P = 0.71) or frequency (vs. SFEmax: r = 0.26, P = 0.32; vs. SFEmax: r = 0.02, P = 0.95). Bronchodilation reversed these qualitative changes, accompanied by significant (P < 0.005) decreases in SFEmax and SFEmax. There were highly significant (P < 0.005) correlations between the test variables and RL or dPtpmax when pre- and postbronchodilator values were pooled (Fig. 7). However, there was only a trend for the correlation between Cdyn and SFEmax (r = -0.49, P = 0.054) and no significant correlation between Cdyn with SFEmax (r = -0.38, P = 0.14). Furthermore, there were no significant correlations between the test variables and VT (vs. SFEmax: r = -0.3, P = 0.25; vs. SFEmax: r = 0.1, P = 0.71) or frequency (vs. SFEmax: r = 0.26, P = 0.32; vs. SFEmax: r = 0.02, P = 0.95).
Variables SFEmax and SFImax were found to increase by a period of tachypnea in all horses (Fig. 8). The Lobeline infusion caused marked hyperpnea followed waveforms and/or slow the frequency response. To consequently attenuate the peaks and nadirs in the potential sampling and analysis errors, which may smoothing or filtering (16). These processes introduce confounding issue was the necessity for electronic
discussion
cally in the peak values (SFEmax, SFImax).

Chlodilation did not significantly alter the inspiratory test variables SFImax or SFIint. None of the inspiratory test variables correlated with any of the conventional variables, with the exception that SFImax showed a trend toward correlation with δPtPmax (r = 0.47, P = 0.067) and Rt (r = 0.46, P = 0.074).

Part III in vivo: Effect of lobeline-induced hyperpnea. Lobeline infusion caused marked hyperpnea followed by a period of tachypnea in all horses (Fig. 8). The variables SFEmax and SFImax were found to increase significantly with hyperpnea but not tachypnea (P < 0.001) (Fig. 9). The VdSum, Vpn, and subtracted (VdSum − Vpn, δPtP, δPtPmax, and SFIint) waveforms showed a distinct pattern during hyperpnea. During expiration, for instance, there was a very early positive spike, followed rapidly by a negative deflection during much of expiration (Fig. 9). This had the effect of increasing the absolute value of SFEmax but decreasing the absolute value of SFIint or causing SFIint to be a negative quantity. As a group, the area measurements SFEint and SFIint were not significantly altered by hyperpnea or tachypnea. The variables SFImax and SFIint were altered by hyperpnea in the opposite direction but to the same extent as SFEmax and SFEint. During tachypnea, when Vt returned to baseline, but f remained, on average, double that at baseline, there was no significant change in any test or conventional variable. Hence, hyperpnea, not tachypnea, produced changes in the test variables, specifically in the peak values (SFEmax, SFImax).

Discussion

Critique of the materials and methods. One potential confounding issue was the necessity for electronic smoothing or filtering (16). These processes introduce potential sampling and analysis errors, which may consequently attenuate the peaks and nadirs in the waveforms and/or slow the frequency response. To avoid these pitfalls, we smoothed the pneumotacho-

graphic and RIP signals in an identical fashion before our post hoc analyses, but potential errors may remain. Therefore, one should view our measurements as approximations of the difference between VdSum and Vpn. There would be a clear advantage to employ a band system that achieved measurements of external flow without the need for differentiation or smoothing, such as the piezoelectric system (25).

Frequency effects on phase and amplitude must also be considered as sources of experimental error. The phase shift that was observed in our positive pressure model indicated that the pneumotach sensor had a slightly better frequency response than Respitrace. It was not possible to increase frequency to supraphysiological levels (>1 Hz) without overall stiffening of the bag; therefore, this may have contributed to the phase shift because of gas compression or hysteresivity of the bag itself. The delay in VdSum caused by differences in the frequency response of the sensors would serve only to attenuate the phase delays (VdSum vs. Vpn) observed in the horses; therefore, we can assume that our test system underestimates rarefaction and compression occurring in vivo. The decay in VdSum amplitude observed in the physical model at higher frequencies would further amplify this error, although the quantitative contribution of each was not determined. In contrast to our system, Jaeger and Otis (17) did not observe phase delays between the volumetric displacement of a piston and a spirometer driven by that piston when there was no resistance between them. The phase shift in the oscillatory test was qualitatively in the same direction as the step test, supporting the concept that frequency responses differed slightly between the pneumotachograph and RIP.

Another technical problem with our study is the use of an arbitrary method for calibrating the RIP waveforms. We were compelled to do so, as it was not possible to assume that a standard calibration, using pneumotachography as a gold standard, would be valid.
during the various obstructions employed in the experiment. By correcting the $V_{\text{dSum}}$ waveform during inspiration, we removed as much of the differences in $V_T$ as possible. This pragmatic approach presumably contributed to an “overcorrection,” as our animals during obstructions clearly exhibited alterations in their inspiratory waveforms as well, and there was a trend in the correlation between conventional and inspiratory test variables in the horses with heaves. Despite this process of calibration, marked dynamic compressive and rarefactive events were observed early during inspiration and expiration in obstructed horses that caused the test variables to change significantly. Hence, the calibration could not have altered the test variables to the extent that the whole breath was corrected.

Phase and magnitude differences between plethysmographic and pneumotachographic measurement of flow and volume were previously observed (17, 24). These discrepancies are a function of resistance, lung volume, f, breathing pattern, and barometric pressure, as previously modeled by Jaeger and Otis (17), according to the following equation for harmonic motion: $\phi = \tan^{-1} 2\pi f R C$, where $R$ is resistance and $C$ is compressibility of gas. Intrapulmonary gas compression is a normal phenomenon (14, 19, 20) during exhalation that is accentuated by hyperpnea and exercise. Differences in plethysmographic and pneumotachographic measures of flow, due to gas compression, worsen in humans with asthma, chronic bronchitis, or emphysema, especially if compounded by hyperpnea (17, 21). We employed these concepts to generate a hypothesis that gas compression as a result, principally of changes in resistance, would be quantifiable using our methods. However, we did not model our system using the approach of Jaeger and Otis (17). The equation of motion assumes sinusoidal harmonic motion, which, in our slow, irregularly breathing subjects, was not evident.

Despite the plethora of studies that have examined the differences between plethysmographic and pneumotachographic volume and flow measurements, there were no studies that we are aware of that have dynam-
ically compared RIP and pneumotachography (i.e., flows). Jackson and coworkers (16) indirectly approached this problem by comparing the kinetics of RIP and pneumotachographic flows (comparing time to reach peak expiratory flow to total expiratory time). They found significant differences in this variable, particularly in infants who wheeze and older neonates with thoracoabdominal asynchrony, and suggested caution in interpretation of uncalibrated RIP. Pennock et al. (26) later employed piezoelectric bands coupled with spirometry and demonstrated a qualitative difference in the magnitude and phase between these signals in normal humans, which he attributed to gas compression. We have taken these observations one step forward by quantifying gas compression in our horse model using boxless plethysmography.

In our horses, there was clear evidence of gas compression by phase delay and magnitude differences in $V_{dSum}$ and $V_{ps}$ that were associated with changes in resistance and Cdyn. As expected, the horses with severe, natural, lower airway obstruction had markedly elevated values for conventional and the test variables. Examination of the waveforms demonstrated that the expiratory portion of the new test waveforms was altered to a much greater extent than the inspiratory portion. In support, a large discrepancy between RIP and pneumotachographic flows persisted during airway obstruction, even after normalization. During bronchodilation, the changes in test variables during expiration were comparable to parallel changes in $\delta P_{tpmax}$, $R_l$, and Cdyn, both qualitatively and quantitatively, with a statistically significant correlation observed between absolute values for SFE$_{max}$ or SFE$_{int}$ and conventional variables. This would suggest that the system used here could be employed to measure bronchodilator effects, again with the distinct advantage of noninvasiveness (no need for an esophageal balloon catheter). This would greatly facilitate serial examinations, particularly in pharmacological studies that require daily or more frequent measurements, and for studies involving untrained horses. Whether our system provides more sensitive or reproducible data than a clinical scoring technique would require further validation.

This study further demonstrates that the change in SFE$_{max}$ and SFE$_{int}$ during airway obstruction can be used to generate dose-response curves and interpolate those curves to obtain valid indexes of airway reactivity. Further studies are required to determine the reproducibility and feasibility of this system for field measurements. The semilog dose-response curve for the test variables was different in shape from the conventional semilog dose-response (i.e., histamine-Cdyn) curve, with the latter appearing more linear. The dose-related changes in SFE$_{max}$ and SFE$_{int}$ resembled more what is seen in barometric whole body plethysmography, whereby changes occur only one or two doses before the clinical reaction (5, 9, 11). To make a comparison with conventional methods, we chose to examine a change in the test variables (35% increase) that matched the magnitude of change in the conventional method (35% decrease in Cdyn) by linear interpolation. This may not be the optimal endpoint to evaluate the airway reactivity in horses or to analyze these curves. However, we did not want to confound the comparison by using different methods of interpolation between our test and conventional methods. The excellent correlation in a small number of horses suggests that the system constructed for this experiment could be applied to horses noninvasively to obtain similar information on airway reactivity.

The role of changing lung volume was not revealed by this study, as we did not measure functional residual capacity (FRC). One would expect that histamine-

Fig. 9. Effect of lobeline-HCl (0.2 mg/kg iv) to induce hyperpnea, followed by tachypnea in horses ($n = 6$). There was a significant effect (*) of hyperpnea (but not tachypnea) on SFE$_{max}$ and peak of subtracted final inhalation waveform (SFI$_{max}$) but no significant effect of hyperpnea or tachypnea on area measurements [SFE$_{int}$, integral of subtracted final waveform during first 25% of inspired volume (SFI$_{int}$)].
induced bronchoconstriction and spontaneous lower airway disease, such as heaves, studied here, would be associated with dynamic hyperinflation, adding to the volume of compressed gas measured with our system. Similar effects were observed in humans with emphysema (17, 21). Further evidence that FRC is important to our measurements is suggested by the work of Johnson and Pierce (18) and later Dorsch et al. (8), who showed that gas compression closely correlated with changes in specific airway conductance (which accounts for changes in FRC). We speculate that $\text{SPF}_{\text{max}}$ and $\text{SFI}_{\text{max}}$ are also variables that are sensitive to changes in lung volume, as a component of gas compression, and this was supported by the effects of hyperpnea during lobeline challenges. The use of a volumetric correction factor may be appropriate to decrease the confounding effects of changes in lung volume and body size on absolute values.

In part III, we attempted to answer whether hyperpnea was associated with discrepancies in $V_{\text{dSum}}$ and $V_{\text{pn}}$. During hyperventilation in horses, this phenomenon was visualized as a transient difference between the peak $V_{\text{dSum}}$ and $V_{\text{pn}}$ ($\text{SPF}_{\text{max}}$, $\text{SFI}_{\text{max}}$) at the beginning of inspiration and expiration. The appearance differed remarkably from the waveforms during bronchoconstriction (natural and histamine induced), where differences occurred asymptomatically, i.e., predominantly during expiration. Hyperpnea effects also deviated from the effects of increased frequency alone (tachypnea) on our physical model, whereby $V_{\text{dSum}}$ amplitude decreased relative to $V_{\text{pn}}$. Tachypnea did not have that effect in the horses. The effect of hyperpnea, therefore, is physiological, rather than artifactual, and may result from compression of airways during expiration, inhomogeneities in time constants of emptying in small airways, or increased lung volume, providing a greater compressed mass of air. Jaeger and Otis (17) noted gas compression in some hyperventilating subjects who maintained a sinusoidal breathing pattern. The sinusoidal pattern, they reasoned, increased the compression of tissues. The breathing pattern during lobeline challenge was also more sinusoidal, as seen during exercise in horses (2).

Based on our observations, one could potentially construct flowmetric variables that discriminate obstruction from hyperpnea. Our use of area differences ($\text{SPF}_{\text{int}}$, $\text{SFI}_{\text{int}}$) was one such attempt. These variables indeed were more “refractory” to the effects of hyperpnea and tachypnea.

In conclusion, a method that directly compares plethysmographic and pneumotachographic flow was found to be both feasible and valid in the horse for measurement of relative changes in lung mechanics because of experimental bronchoconstriction or during bronchodilation of horses with severe, recurrent airway obstruction. The effects of lung volume, barometric pressure, and $f$ (when combined) require further observation in a physical model of the measurement system and in the horse. The advantages of this system for testing airway reactivity and bronchodilator effects include its noninvasive platform and the lack of requirement for energy input (pressure, loudspeakers) to drive the system, making the system potentially portable.

REFERENCES


