The Measurement of Thoraco-abdominal Asynchrony in Infants With Severe Laryngotraceobronchitis*

G. Michael Davis, M.B., Ch.B.; David M. Cooper, M.B., B.S.; and Ian Mitchell, M.B., Ch.B., F.C.C.P.

Retractions of the lower rib cage (chest wall distortion [CWD]) during inspiration are frequently observed with moderate to severe respiratory disease in the infant. Laryngotraceobronchitis (LTB) results in a reversible partial airway obstruction with severe CWD. We wished to measure the motion of the chest wall during distortion to determine the changes in minute ventilation (\(V_e\)) and to evaluate this clinical sign as a means of assessing disease severity. The respiratory inductance plethysmograph was used to determine the distortion of the lower chest wall, and distortion was correlated with \(V_e\), measured at the mouth, in six infants with severe LTB and ventilatory failure. As the conditions of these infants improved, the CWD decreased with decreasing transcutaneous carbon dioxide tension (tc\(\text{CO}_2\)). \(V_e\) increased from 0.27±0.12 L/min kg\(^{-1}\) at a tc\(\text{CO}_2\) of 64 mm Hg to 0.64±0.06 L/min kg\(^{-1}\) when the tc\(\text{CO}_2\) had fallen to 28 mm Hg. Over the same change in tc\(\text{CO}_2\), the tidal volume (\(V_T\)) increased from 4.8±0.5 ml kg\(^{-1}\) to 15.7±1.4 ml kg\(^{-1}\). In the most severe disease state, the excursion of the chest wall (as an inductance) was \(-14±3\) mV in severe obstruction, but increased to 75 mV±4 mV with resolution of the illness. The timing and vector of movement of the abdomen and chest wall were expressed as a Lissajous figure, which is measured as a phase angle. The severity of the disease process, as determined by tc\(\text{CO}_2\), was directly related to the phase relationship, and thus reflected both \(V_e\) and \(V_T\). The severity of the CWD may be assessed rapidly by the use of Lissajous figures. (Chest 1993; 103:1842-48)

\(\Theta_c = \) calculated phase angle; \(\Theta_m = \) measured phase angle; CWD = chest wall distortion; \(I_{ABD} = \) inductance of abdomen; \(I_{CW} = \) inductance of chest wall; LTB = laryngotracheobronchitis; tc\(\text{CO}_2 = \) transcutaneous carbon dioxide tension; tc\(\text{FO}_2 = \) transcutaneous oxygen tension; RIP = respiratory inductance plethysmography; \(V = \) airflow

The assessment of the severity of respiratory disease in infants relies on clinical observation because of the difficulties in measuring ventilation in the infant. There are at least two clinical methods of assessing the severity, both of which include an assessment of chest wall distortion (CWD) during inspiration as an element of the severity score. Because this is a nonspecific respiratory system abnormality, CWD is thus interpreted as reflecting increased pleural pressure swings because of a change in the resistance to airflow within the respiratory system.

Chest wall distortion occurs during inspiration as the abnormal paradoxical inward movement of the lower chest wall, demarcated by the insertion of the diaphragm into the ribcage, representing a collapse of the lower chest wall at a time when the thoracic cavity is increasing in volume (ie, "out of phase" or paradoxic). This uncoupling of the ribcage-abdominal motion may result in a decrease in tidal volume (\(V_T\)), a decrease in alveolar minute ventilation (\(V_e\)), and thus ventilatory failure.

Chest wall distortion is frequently seen in the newborn, although it may be present at any time during the preschool years. Two recent publications have suggested that objective assessment of the severity of respiratory disease in infants is possible by using measurements of the timing of motion of the chest wall and abdominal excursions, to avoid calibrating an inductance plethysmograph for volume.

Viral laryngotracheobronchitis (LTB) is representative of a group of diseases in infants and young children associated with acute partial upper airway obstruction, increased resistance to inspiratory airflow, and severe CWD. In the most severe form, elevation of arterial carbon dioxide tension occurs because of alveolar hypoventilation. Therefore, we studied infants with acute severe LTB to determine the physical displacement of the chest wall and abdominal compartments during the progression of the disease. The study determined during the clinical illness the magnitude of the CWD, the changes in \(V_T\), and the effects on gas exchange.

METHODS

Subjects

Six previously healthy infants, admitted to the Pediatric ICU of the Alberta Children's Hospital because of a clinical diagnosis of severe LTB and ventilatory failure, were included in this study. Infants with preexisting pulmonary or cardiac disease or with acute epiglottitis were excluded from the study. The study was approved by the Ethics Review Committee of the University of Calgary.

At the time of hospital admission, all had severe partial upper airway obstruction and a clinical diagnosis of LTB. All required a fractional inspired oxygen (\(\text{FIO}_2\)) greater than 0.35 to maintain a

*From the Department of Pediatrics and the Pediatric Intensive Care Unit, Alberta Children's Hospital, Calgary, Alberta, Canada. Manuscript received June 10; revision accepted December 8. Reprint requests: Dr. Davis, Suite D-380, 2300 Tupper Street, Montreal, Quebec, Canada H3H 1P3

1842 Measurement of Thoraco-abdominal Asynchrony in Infants (Davis, Cooper, Mitchell)
transcutaneous oxygen tension (tcPO$_2$) >55 mm Hg and each infant had a transcutaneous carbon dioxide tension (tcPCO$_2$) greater than 45 mm Hg to meet the requirements of ventilatory failure. Infants who required immediate endotracheal intubation were excluded from this study. The initial treatment of the ventilatory failure was determined by the attending physician.

When the infant was clinically stable, usually within 2 h of ICU admission, prospective respiratory monitoring was commenced during quiet sleep$^9$ and continued throughout the illness until complete clinical resolution of both the illness and CWD. Throughout the progression of the illness, the tcPO$_2$ was maintained greater than 55 mm Hg by judicious manipulation of the FIO$_2$, necessitating at times up to an FIO$_2$ of 0.85. Racemic epinephrine was routinely administered to these infants, but all recordings were performed at least 1 h after a treatment with this inhaled medication.

The displacement of the chest wall and abdomen was monitored using a respiratory inductance plethysmograph (RIP) (Respitrace, Ambulatory Monitoring, Ardenley, NY) with the ventilatory movements measured each 8 l during the entire illness. The inductance bands for the RIP (Ripsbands) were fixed on the chest at the nipple line and midabdomen as previously described for infants by Duffy et al.$^9$ with the position of the inductance bands not altered between measurements. Volume calibration for the RIP was not performed but the signals are recorded as an inductance (volts). The volume of each breath (VT) was obtained from airflow (V), sensed at the mouth by a mask and pneumotachograph (Hewlett-Packard 21609B) and the V was subsequently integrated to VT (Hewlett-Packard 8815A). All volume measurements were performed during quiet (non-REM) sleep as determined by direct

observation and by electro-oculogram, recorded by a high-gain biologic signal amplifier (Hewlett-Packard 8816A). These epochs of quiet sleep constituted less than 15 percent of total time monitored, and were frequently of short duration, often lasting only 10 to 15 min. Simultaneously the transcutaneous oxygen and carbon dioxide (tcPO$_2$ and tcPCO$_2$) tensions were measured (Kontron Scientific Ltd, Mississauga, Ontario), and heart rate was recorded.

Analysis

Analysis of the pattern of breathing was performed during epochs of quiet sleep when the tcPCO$_2$ was stable for 5 min. Twenty consecutive breaths were analyzed for the displacement of the chest wall (Inc) and abdominal (I Abd) compartments, and for ventilatory parameters, using the measured VT (obtained from V) respiratory frequency to obtain the instantaneous minute ventilation (Ve) and the mean inspiratory flow (Vr/Ti). All results were reported as a mean and standard deviation, with the ventilatory volumes normalized by body weight to allow comparison between the infants.

The pattern of movement of each compartment was analyzed. The relationship of the motion of the two compartments was derived with the outward movement (inspiration) arbitrarily defined as a positive signal to derive the Lissajous figures of relative motion of the two compartments after the method of Allen et al.$^9$ When CWD was present, the inductance of the ribcage (Inc) was derived for the maximal displacement such that a negative Inc was possible (Fig 1) and expressed as the measured phase angle (Om). Furthermore, a calculated phase angle (Oc) was also obtained, after the scalar method of Agostini and Mognini,$^4$ by using the difference in time to peak displacement for the ribcage and abdomen, divided by the

---

**Figure 1.** Recordings obtained by respiratory inductance plethysmography (RIP) and pneumotachograph during an episode of laryngotracheoebronchitis in one infant. The left panel demonstrates synchrony of movement between the chest wall (Inc) and abdomen (I Abd), as well as the tidal volume (VT), with the Lissajous loop of the relative motion of the Inc and I Abd. The center panel demonstrates moderate chest wall distortion with the initial negative deflection of the Inc, and the change in orientation of the Lissajous loop. The right panel illustrates severe distortion of the chest wall throughout inspiration, with a negative deflection in the Inc, and the loop demonstrates the large phase angle between the I Abd and Inc.
total respiratory cycle time, and expressed as degrees. Statistical analysis was performed using regression techniques, and the two methods of deriving the phase angles were compared by the method of Bland and Altman with statistical significance assumed at the 0.05 level.

RESULTS

The mean age of the 6 infants was 10 months (7 to 13 months) and mean weight was 7.1 kg (5.5 to 9.1 kg). Because of the selection of the infants, there was ventilatory failure present for the first recording of each subject. The magnitude of the CWD varied directly with the clinical severity of the disease, as illustrated in Figure 2, although there were three general patterns of chest wall motion in all subjects corresponding to different stages of illness. Firstly, the mildest abnormality, delayed expansion of the chest wall at the beginning of inspiration, was usually seen late in the recovery phase (Fig 1, left panel). This minimal distortion of the normal synchrony of movement of the two compartments was associated with the clinical resolution of the illness. The anticlockwise pattern of the Lissajous loop, and a phase angle (Ωm) of <30°, suggested that the ribcage did expand during inspiration, but was temporally delayed behind the excursion of the abdomen.

Secondly, with more severe CWD, there was early paradoxical inward displacement at the beginning of inspiration, but outward displacement of the chest wall prior to the onset of abdominal expiration (Fig 1, center panel). This stage of illness was associated with an anticlockwise Lissajous figure, and a phase angle ranging between 30° and 90°.

Finally, the most severe impairment was associated with marked CWD with inward motion of the chest wall throughout the entire inspiratory phase of the abdomen, although late minimal expansion of the chest wall occurred during abdominal expiration (Fig 1, right panel). In this case, the Lissajous figure was associated with a phase angle (Ωm) greater than 90°.

In all cases, increasing severity of the disease process was associated with the increasing measured phase angle. There was a direct relationship between the measured phase angle and the severity of disease, as determined by the tcPCO₂, and it is expressed in Figure 3 (r² 0.982, p<0.005). This change in measured phase angle did not differ significantly from the calculated phase angle by regression techniques (r² 0.901, p<0.005), as illustrated in Table 1 and Figure 4, but there was a systematic bias between the measurements with the Ωc being greater than the Ωm (95 percent confidence intervals −11.55 to −6.51) by the method of Bland and Altman.

VENTILATION

The ventilatory volumes obtained for the six infants with LTB were normalized for body weight. The $V_E$ increased from a minimum of 0.28 ± 0.06 L·min⁻¹·kg⁻¹ at a tcPCO₂ of 64 mm Hg to 0.63 ± 0.09 L·min⁻¹·kg⁻¹ at a tcPCO₂ of 28 mm Hg. The relationship of tcPCO₂ to $V_E$ is shown in Figure 2 (lowest panel), and included in Table 1.

Further, the increase in $V_E$ resulted principally through improvement in $V_T$ which increased from 5.6 ± 0.6 ml·kg⁻¹ with the most severe airflow obstruction (tcPCO₂ of 64 mm Hg) to 15.7 ± 0.4 ml·kg⁻¹ with clinical resolution when the tcPCO₂ was 28 mm Hg (Fig 2, center panel). Simultaneous with the increase in $V_T$, there was an initial increase in respiratory frequency from 50 breaths·min⁻¹ at a tcPCO₂ of 64 mm

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{The changes in $V_E$ (bottom panel), $V_T$ (middle panel), and frequency (upper panel) that occurred with changes in transcutaneous carbon dioxide tension throughout the clinical course of recovery from severe laryngotracheobronchitis. Each point represents the mean and standard deviation of 20 consecutive breaths. All regressions were significant at p<0.05.}
\end{figure}
Hg to a maximum of 69 breaths·min⁻¹ at 42 mm Hg, and then subsequently decreasing to 50 breaths·min⁻¹ at 28 mm Hg (Fig 2).

This fall in tcPCO₂ and increase in VE was due, primarily, to the progressive recruitment of outward (positive) chest wall excursion during inspiration. The change in Iinc increased from a negative impedance change of -4 mV ± 3 mV to 75 mV ± 4 mV over the same range of tcPCO₂. The excursion of the abdominal compartment, measured as the IABD, mimicked that of respiratory frequency. The IABD fell from the initial value of 70 mV ± 7 mV at 64 mm Hg, to a nadir value of 43 mV ± 4 mV at 50 mm Hg, and then subsequently rose to the maximum of 82 mV ± 6 mV at a tcPCO₂ of 28 mm Hg. The changes in tcPCO₂ with V̇ and frequency were significant by a second-order polynomial regression (r² = 0.87, p<0.05, and r² = 0.60, p<0.05, respectively) but correlated linearly with V̇ (r² = 0.932, p<0.005). As was expected, there was an inverse linear relationship between the tcPCO₂ and the rib cage inductance (r² 0.82, p<0.005), and directly with the phase angle (r² 0.842, p<0.005) as illustrated in Figure 3.

**DISCUSSION**

This study has shown that a predictable change in CWD occurs during the recovery from ventilatory failure in infants with severe LTB. The pattern of CWD consisted of asynchronous displacement of the chest wall and abdominal compartments during inspiration, resulting in a decrease in the V̇, and a decrease...
in Vt, principally through the loss of the chest wall excursion during inspiration. The chest wall motion, measured as Inc, was paradox to the abdomen in the most severe state, but with resolution of the clinical illness, the motion became temporally displaced rather than paradoxical.

In this study, the progression of the CWD was quantitatively associated with an increasing phase angle, thus indicating that both the increase in asynchrony of chest wall motion (phase angle) and the decrease in chest wall displacement (Inc) contributed to alveolar hypoventilation. This is in agreement with previous observations and clinical scores where deterioration in the clinical condition was associated with more obvious inward movement of the lower part of the chest wall. The loss of the chest wall contribution led to a fall in Vt, a fall in Ve, and thus alveolar hypoventilation with elevated tcPCO2.

The two previous studies reported have not found a direct relationship between the phase angle and the tcPCO2. The different results in this study may be due, in part, to the narrow age range studied (7 to 13 months), leading to a very homogeneous group of previously healthy infants with a single disease process. The observations were homogeneous with the six infants demonstrating a similar response in phase angle and rib cage inductance to a fall in the alveolar ventilation (Fig 5). These results were in contrast to the study of Sivan et al who studied 30 infants, only 8 of whom had LT6, and were examined short-term following the administration of racemic epinephrine (Vaponefrin). Similarly, the group of infants studied by Allen et al were between 2 and 13 months, had chronic lung disease, and were assessed within 15 to 20 min following administration of a bronchodilator.

Furthermore, under conditions of respiratory failure, the intercostal muscles may well be fatigued, such that the compliance of the chest wall (Cw) will be principally determined by the passive characteristics. In turn, the passive Cw is more likely to be an age-determined factor, although evidence to support this conjecture is minimal. In this manner, the close relationship between phase angle and tcPCO2 is most likely a function of the group selection rather than a generally applicable statement.

The analysis of relative motion of the two compartments using a Lissajous figure allows a rapid means of measuring the degree of asynchrony. Because the motion included in the Lissajous figure is dependent on the time relationship between two sine waves, it is an appropriate means of rapidly assessing the timing of chest wall movement and the vector. When there is inward displacement of the chest wall throughout the whole of the inspiratory phase, then the loop changes quadrants, the phase angle is between 90° and 180°, and this change can quickly be recognized using either an oscilloscope or X-Y plot. Previous authors have noted that, despite the limitation that the IRC and IABD displacement curves do not fit as perfect sine waves, the error in using the Lissajous loop for analysis is small. Our own work confirms this observation in that the comparison of the two different methods of analysis, one of which is dependent on the assumption of a sine wave, demonstrates there is no significant error associated with the phase angle by measurement.

There is normally a compensatory response to a decrease in Ve by an increase in either respiratory frequency or an increase in abdominal volume displacement, as measured by the IABD. An increase

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, mo</th>
<th>Weight, kg</th>
<th>tcPCO2, mm Hg</th>
<th>Vt, m/kg⁻¹</th>
<th>Ve, L/min</th>
<th>Phase Angle, Measured</th>
<th>Phase Angle, Calculated</th>
<th>Inc, mV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7.0</td>
<td>5.5</td>
<td>52</td>
<td>5.8±0.7</td>
<td>0.37±0.06</td>
<td>110±12.2</td>
<td>90±11.6</td>
<td>15±8</td>
</tr>
<tr>
<td>2</td>
<td>9.0</td>
<td>7.6</td>
<td>52</td>
<td>6.0±0.4</td>
<td>0.36±0.09</td>
<td>100±7.9</td>
<td>90±13.6</td>
<td>15±6</td>
</tr>
<tr>
<td>3</td>
<td>9.5</td>
<td>8.4</td>
<td>55</td>
<td>5.8±0.7</td>
<td>0.34±0.07</td>
<td>110±10.3</td>
<td>100±13.5</td>
<td>9±5</td>
</tr>
<tr>
<td>4</td>
<td>10.0</td>
<td>8.8</td>
<td>60</td>
<td>4.8±0.5</td>
<td>0.29±0.06</td>
<td>120±10.2</td>
<td>110±11.6</td>
<td>-8±2</td>
</tr>
<tr>
<td>5</td>
<td>11.5</td>
<td>9.0</td>
<td>62</td>
<td>5.4±0.3</td>
<td>0.27±0.09</td>
<td>120±15.3</td>
<td>100±10.9</td>
<td>-8±4</td>
</tr>
<tr>
<td>6</td>
<td>13.0</td>
<td>9.1</td>
<td>64</td>
<td>5.6±0.6</td>
<td>0.28±0.06</td>
<td>120±7.5</td>
<td>110±8.6</td>
<td>-14±9</td>
</tr>
</tbody>
</table>

*tcPCO2 = transcutaneous carbon dioxide tension; Vt = tidal volume; Ve = minute ventilation; Inc = inductance of chest wall.
The factors determining CWD in these infants were not specifically studied. We speculated that inspiratory CWD was the result of an imbalance in the opposing forces applied to the chest wall during inspiration. Normally, the chest wall is supported during inspiration by the bony structures and the intercostal muscle groups that act to both anchor and expand the chest wall in synchrony with the diaphragmatic contraction during inspiration. In the infant, the static chest wall compliance is greater than lung compliance, implying that support by contraction of intercostal muscle groups is necessary to augment the rigidity for the chest wall to function normally. Diaphragmatic contraction would tend to collapse the lower chest wall because the large horizontal vector from its insertion into the lower ribs must favor CWD. As well, the large swing in negative pleural pressure associated with partial upper airway obstruction must increase the transmural pressure to augment collapse of the chest wall. Finally, fatigue of the small intercostal muscles of the chest wall may occur, leading to failure to fixate or expand the chest wall during inspiration. Alteration of one or more of these determining factors would potentially result in the failure of the chest wall to withstand the inward forces during inspiration, and thus promote development of CWD.

In summary, this study shows that, during the recovery from severe LTB, chest wall movement shows a predictable progression in pattern of distortion, associated with a decreased Ve and alveolar hyperventilation. The normal compensatory mechanisms of increasing Ve and frequency do not suffice to defend Ve when there is severe distortion, resulting in an increase in tcPCO2 secondary to alveolar hyperventilation. Furthermore, in this restricted group of patients, within a very narrow age range, the temporal relationship of the chest and abdominal wall results in a phase angle of motion, rapidly measured from the Lissajous figure, that is directly related to the Ve and the tcPCO2. We therefore find that the repeated measurement of phase angle provides a simple and reliable technique of objectively assessing both severity and progression of LTB, and postulate that the phase angle can be used as a noninvasive means of following the severity of obstruction within the same patient.

REFERENCES
2 Westley CR, Cotton EK, Brooks JG. Nebulised racemic epi-
3 Ringel EB, Lincoln SH, Mead J, Ingram RH. Chest wall
distortion during resistive inspiratory loading. J Appl Physiol
1848

Measurement of Thoraco-abdominal Asynchrony in Infants (Davis, Cooper, Mitchell)

1989; 65:1063-98

Downloaded From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21672/ on 05/31/2017