Tracheal suctioning is associated with prolonged disturbances of cerebral hemodynamics in very low birth weight infants

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Objective
Examining the effects of tracheal suctioning on cerebral hemodynamics of normotensive ventilated very low birth weight (VLBW) infants with normal cranial ultrasounds; determining the factor(s) influencing changes in mean cerebral blood flow velocity (CBFv) after suctioning.

Methods
Seventy-three VLBW infants had continuous monitoring of mean arterial blood pressure (MABP), PaCO₂, PaO₂ and mean CBFv before, during, and after 202 suctioning sessions during the first week of life. Peak (or nadir) and relative changes of the four variables for 45 min after suctioning were calculated. Multiple linear regression was used to determine the factor(s) influencing changes in mean CBFv after suctioning.

Result
Birth weight was 928 ± 244 g; gestational age was 27.0 ± 2.0 weeks. Mean CBFv increased to 31.0 ± 26.4% after suctioning and remained elevated for 25 min. PaCO₂ was highly associated with mean CBFv (P < 0.001), whereas MABP and PaO₂ were not.

Conclusion
We observed prolonged increases of mean CBFv following suctioning in ventilated VLBW infants that were previously unrecognized. This is concerning since disturbances of CBF may be associated with subsequent brain injury.

Keywords: cerebral blood flow velocity; carbon dioxide; oxygen; blood pressure; cerebral autoregulation

Introduction
Tracheal suctioning is a common neonatal intensive care procedure that is performed to clear tracheobronchial airways of secretions and maintain patency of the endotracheal tube in intubated infants. This necessary procedure, however, is associated with a number of transient disturbances of systemic hemodynamics and gas exchange that may be associated with impaired cerebral blood flow (CBF) regulation and development of subsequent neonatal brain injury. Elevated blood pressure (BP), 1–8 bradycardia, 2–4,9,10 hypoxemia and arterial desaturation, 1–5,9–11 and hypercapnia, 11 as well as increased intracranial pressure1,3,6,7,12 have been observed during and briefly after tracheal suctioning. Abrupt increases in BP may cause cerebral hyperperfusion and intraventricular hemorrhage (IVH) in infants with or without intact cerebral autoregulation. Disturbances of arterial blood gases during suctioning may also have deleterious effects on CBF, since carbon dioxide and oxygen are potent mediators of cerebrovascular tone. 13

Very brief increases of CBF measures in premature infants have been reported (using both Doppler ultrasound and near-infrared spectroscopy) during tracheal suctioning; 1,4,5,11,14 two additional studies also observed increases in CBF that were preceded by transient decreases in cerebral blood volume15 and CBF velocity (CBFv).16 This is concerning because increases in CBF in premature infants and neonatal animals have been shown to play an important role in the development of IVH.17,18 Other investigators, however, observed decreased cerebral blood volume following tracheal suctioning. 9,10 Inconsistency in these observations may be due to the following reasons: use of different suctioning and ventilation techniques (open vs closed; use of pre-oxygenation, various ventilator manipulations and/or saline vs non-application of these procedures and so on); use of different CBF measurement methods, and indirect and intermittent physiological monitoring systems; inclusion of heterogeneous populations (wide range of gestational and postnatal ages, different behavioral states, different respiratory diseases, infants with and without hypotension or brain injury and so on); differences in sedation and paralysis protocols; and measurements either during clinically indicated suctioning procedures or during convenient periods. Thus, consensus is lacking regarding the effects of tracheal suctioning on cerebral hemodynamics; additionally, the duration of effects of tracheal suctioning on the cerebral circulation is not known.
The main objectives of this observational study were to use a continuous physiological monitoring system (1) to examine the acute effects of clinically indicated suctioning procedures on cerebral hemodynamics of normotensive ventilated very low birth weight (VLBW, birth weight \( \leq 1500 \) g) infants with normal cranial ultrasounds and (2) to assess whether changes in mean CBFv were associated with changes in mean arterial BP (MABP), PaCO\(_2\), or PaO\(_2\). Based on our observations that surfactant administration was associated with increases of mean CBFv due primarily to increases in PaCO\(_2\),\(^{19}\) we hypothesized that mean CBFv would also increase during and after tracheal suctioning and was primarily related to increases in PaCO\(_2\).

**Methods**

**Study sample**

VLBW infants born at the University of Arkansas for Medical Sciences between July 2002 and June 2005 were eligible for this observational study if they required mechanical ventilation and had a 3.7 Fr umbilical arterial catheter placed during newborn stabilization. Infants with major congenital anomalies were excluded. All infants had normal cranial ultrasounds throughout their hospitalization and did not require treatment for hypotension during the first week of life. Infants included in this study were part of a larger investigation to determine the pattern of development of cerebral autoregulation in VLBW infants. Neonatal characteristics (time and date of birth, birth weight, gestational age, race, gender, cranial ultrasound findings, administration of vasopressors, and Apgar scores at 1 and 5 min) were abstracted from the clinical research database. Gestational age was estimated based on the best obstetrical and neonatal criteria. If there was a discrepancy of >2 weeks between the obstetrical and the neonatal estimates, the neonatal estimate was used.

Routine intensive care procedures were left to the discretion of the attending neonatologist who was present at all newborn resuscitation of premature infants. Typically, intubated VLBW infants at our institution received prophylactic surfactant in the delivery room \( \leq 20 \) min of life. Time cycled, pressure limited, high-frequency ventilators were used during newborn resuscitation with initial settings: peak inspiratory pressure 16 to 20 cm H\(_2\)O; positive end-expiratory pressure 4 to 5 cm H\(_2\)O; inspiratory time 0.25 s; respiratory rate 50 to 60 breaths per min; and FIO\(_2\) to maintain arterial saturations >90%. Once transferred to the neonatal intensive care unit, some infants were transitioned to high-frequency flow-interrupter ventilators. The decision to use high-frequency ventilation was neonatologist-dependent, and generally, more immature infants were provided with high-frequency ventilation. Mean airway pressure was usually set 1 to 2 cm H\(_2\)O above the mean airway pressure on conventional mechanical ventilation,\(^{20,21}\) amplitude was increased until appropriate chest movement was noticed, and the frequency was set between 10 and 12 Hz. During the study period, a permissive hypercapnic ventilation strategy (with a PaCO\(_2\) target range of 45 to 55 mm Hg) was used.\(^{22}\)

It was our practice to maintain MABP (in mm Hg) equal to or greater than gestational age (in weeks). When MABP fell, premature infants were typically administered a fluid bolus followed by dopamine infusion. Additional doses of surfactant were typically administered every 6 h (up to a maximum of four doses on the first day of life (DOL)) to infants with continued radiographic evidence of respiratory distress syndrome and >30% FiO\(_2\). A majority of the cranial ultrasounds were interpreted by one senior radiologist.

**Monitoring equipment**

Continuous BP monitoring was performed with an umbilical arterial catheter (Diametrics, St Paul, MN, USA; Argyle/Tycor Healthcare/Kendall, Mansfield, MA, USA) attached to a BP transducer (Transpac IV, Abbott Critical Care Systems, North Chicago, IL, USA).

Continuous blood gas monitoring was performed with a Neotrend-L (Diametrics Medical Ltd, St Paul, MN, USA) fiber optic sensor that was placed in the umbilical arterial catheter. The sensor was connected to a satellite monitor (Diametrics Medical Ltd, St Paul, MN, USA) that continuously displayed pH, PaCO\(_2\), PaO\(_2\), temperature, bicarbonate, base excess and oxygen saturations in real-time. Digital output from the blood gas monitor was converted to an analog signal by a digital-to-analog converter.

Continuous measurements of right middle cerebral artery CBFv were made using a transcranial Doppler ultrasound system (Nicolet Biomedical Pioneer, Madison, WI, USA). A lightweight 2-MHz pulsed-wave button transducer was placed transtemporally anterior to the external ear and above the zygomatic arch and held in place by an appropriately sized crocheted hat (courtesy of the Arkansas Extension Homemakers Council). A depth of 16 to 22 mm was used to study the proximal portion (M1) of the middle cerebral artery. A 100-Hz low-pass filter was used to dampen ‘noise’ from the vessel wall. Transducer placement was optimized when the highest intensity acoustic signal was perceived and the highest intensity Doppler spectra were visualized. Fast Fourier analysis was performed on the CBFv signal to determine the systolic, diastolic and mean CBF velocities. The ultrasound intensity (5 to 21 mW cm\(^{-2}\)) was well below the recommendations for continuous monitoring.\(^{25}\) CBFv tracings were consistent \( >1 \) h with minimal or no drift in the signal intensity. The investigator or research assistant was present at the bedside during all monitoring periods.

Analog signals from the BP monitor (112 samples s\(^{-1}\), 10 mV/1 mm Hg), blood gas monitor (1 sample s\(^{-1}\); PaCO\(_2\): 25 mV/1 mm Hg; PaO\(_2\): 4 mV/1 mm Hg), and transcranial Doppler (100 samples s\(^{-1}\), 10 mV/1 cm/s) were collected simultaneously with a data acquisition system (PowerLab 8 channel, ADInstruments, Mountain View, CA, USA). Cyclic waveform analysis...
was performed on the BP data to calculate the MABP (the average amplitude of the BP waveform over 1 cycle) and systolic and diastolic BPs (maximum and minimum values of the BP cycle, respectively).

**Experimental protocol**

Informed consent was obtained from a parent prior to study participation. BP, PaCO₂, PaO₂ and CBFv were continuously monitored from each VLBW infant before, during and after clinically indicated tracheal suctioning procedures during the first week of life. Infants were monitored up to twice daily during the first 3 days and once daily during the next 4 days, if still intubated. Baseline monitoring began ∼15 min before tracheal suctioning, when infants were quiet and not undergoing any other procedures, and continued for ∼45 min post-procedure (to discern duration of disturbances). The University of Arkansas for Medical Sciences Institutional Review Board approved the study protocol.

**Standard tracheal suctioning protocol**

Tracheal suctioning of ventilated VLBW infants during this study was performed only when clinically indicated and was never routinely scheduled or performed for the convenience of the investigators. No pre-oxygenation or hyperventilation was used. The tracheal suctioning protocol was performed only when clinically indicated and was never statistically appropriate to use an ordinary least squares regression modeling approach, which would treat all of the sessions as being independent of one another. Therefore, multiple regression modeling that accounts for the correlation between tracheal suctioning sessions for a given infant was utilized.

Physiological variables included in the model were PaCO₂ (mm Hg), MABP (mm Hg) and PaO₂ (mm Hg) during each infant suctioning session. Other candidate variables considered as potential confounders were birth weight (grams), estimated gestational age (weeks), postnatal DOL (1 to 7), race (black, white, Hispanic), gender and mode of ventilation during the suctioning session (conventional vs high frequency ventilation).

**Results**

**Subject characteristics**

Seventy-one of 75 infants (42 females and 31 males) survived to hospital discharge. Forty were white, 27 were black and 6 were Hispanic. Nineteen (26%) infants were from multifetal gestations. Premature delivery was associated with preterm premature rupture of the membranes (n = 21), pregnancy-induced hypertension (n = 20), preterm labor (n = 18), chorioamnionitis (n = 7), incompetent cervix (n = 4), twin–twin transfusion (n = 1), cocaine abuse with abruption (n = 1) and abdominal trauma (n = 1). Sixty-six (90%) were exposed to antenatal steroids. Fifty-five (75%) were delivered by Cesarean section. The birth weight was 244 g and the gestational age was 27.0 ± 2.0 weeks. The median (and interquartile range) Apgar scores at 1 and 5 min were 4 (3 to 6) and 7 (6 to 7), respectively. The infants received 2.6 ± 1.0 doses of surfactant; all infants received ≥1 dose. None of the infants received vasopressors or had anatomical abnormalities on cranial ultrasounds during the first week of life.

**Tracheal suctioning**

There were a total of 202 tracheal suctioning sessions: 28 on DOL 1; 54 on DOL 2; 47 on DOL 3; 27 on DOL 4; 26 on DOL 5; 15 on each infant with stable baseline data, a locally weighted regression (LOESS)²⁴ technique was used to assist in revealing the behavior of each of the four variables over time.

The first objective was to obtain descriptive measures of peak (or nadir) values for each of the variables for the infant sample. The mean ± s.d. values for the time (min) to reach the peak (or nadir) and the change (mm Hg or cm sec⁻¹) from baseline to peak (or nadir) after tracheal suctioning were calculated. The second objective was to calculate the mean percent change from baseline and a corresponding 95% CI at every second of monitoring after tracheal suctioning for 45 min for each of the four variables for the infant sample.

Next, a multiple linear regression analysis was performed where the dependent variable was mean CBFv (cm sec⁻¹). Since more than half of the infants had >1 tracheal suctioning session, it was not statistically appropriate to use an ordinary least squares regression modeling approach, which would treat all of the sessions as being independent of one another. Therefore, multiple regression modeling that accounts for the correlation between tracheal suctioning sessions for a given infant was utilized.
DOL 6; and 5 on DOL 7. Infants had either conventional mechanical ventilation or high-frequency ventilation during 124 and 78 suctioning sessions, respectively. Infants receiving high-frequency ventilation were more immature, weighed less, had lower Apgar scores and received more surfactant doses than infants on conventional ventilation (Table 1). The mean airway pressure and FIO₂ were significantly higher, baseline mean CBFv and PaCO₂ were significantly higher, and percent change in mean CBFv and PaCO₂ were significantly lower in infants on high-frequency ventilation compared to those on conventional ventilation; all the differences, however, were not clinically important.

Since CBF disturbances following tracheal suctioning have been reported to be independent of the mode of ventilation and because mode of ventilation was not an independent predictor of mean CBFv (see multiple regression section below), we combined the data from suctioning sessions of infants on both conventional and high-frequency ventilation for the analysis.

Table 1 Comparison of infants receiving high-frequency vs conventional ventilation

<table>
<thead>
<tr>
<th>Factor</th>
<th>HFV (n = 78)</th>
<th>CMV (n = 124)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational age (weeks)⁴</td>
<td>26.3 ± 2.0</td>
<td>27.1 ± 1.8</td>
<td>0.005</td>
</tr>
<tr>
<td>Birth weight (g)⁴</td>
<td>823 ± 178</td>
<td>983 ± 220</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Apgar 1 min⁵</td>
<td>3 (1, 4)</td>
<td>4 (3, 6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Apgar 5 min⁵</td>
<td>6 (5, 6)</td>
<td>7 (6, 7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Surfactant doses⁴</td>
<td>2.9 ± 0.9</td>
<td>2.4 ± 1.0</td>
<td>0.012</td>
</tr>
<tr>
<td>Mean airway pressure (cm H₂O)⁴</td>
<td>7.9 ± 1.9</td>
<td>6.3 ± 0.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Oxygen concentration (FIO₂)⁵</td>
<td>27.9 ± 9.7</td>
<td>24.8 ± 5.3</td>
<td>0.005</td>
</tr>
<tr>
<td>Baseline mean CBFv (cm/sec)⁴</td>
<td>19.4 ± 5.4</td>
<td>18.0 ± 4.9</td>
<td>0.046</td>
</tr>
<tr>
<td>Baseline PaCO₂ (mm Hg)⁴</td>
<td>48.6 ± 8.0</td>
<td>45.8 ± 6.3</td>
<td>0.005</td>
</tr>
<tr>
<td>% Change in mean CBFv⁴</td>
<td>25.2 ± 26.8</td>
<td>34.6 ± 25.5</td>
<td>0.013</td>
</tr>
<tr>
<td>% Change in O₂²</td>
<td>9.5 ± 12.1</td>
<td>14.7 ± 12.0</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Abbreviations: CBF, cerebral blood flow; CBFv, cerebral blood flow velocity; CMV, conventional ventilation; HFV, high-frequency ventilation.

Mean ± s.d.

Median (25th, 75th percentiles).

Mean percent change from baseline

The sample group’s mean percent change from baseline and a corresponding 95% CI at each second after suctioning for 45 min for each of the four variables are illustrated in Figure 2. Between 6 and 7 min after suctioning, mean CBFv reached its maximum mean percent increase of 17%, and gradually returned to baseline values by 25 min. The mean percent increase in PaCO₂ (%) mirrored changes in mean CBFv by peaking between 6 and 8 min and returning to baseline by 25 min. The mean percent change in MABP peaked earlier and reached baseline much sooner. The mean percent change in PaO₂ reached its lowest value of approximately −12% at 3 min, and then increased to its maximum value (9%) at ∼10 min. It is important, however, to realize that the figure illustrates mean percent changes from baseline for the group sample. So, while the average increase in mean CBFv from baseline to peak was 31.0 ± 26.4%, this is not reflected in the figure, since the peaks occurred at different time points for individual infant suctioning sessions.

Multiple regression

The final multiple linear regression model consisted of the following independent predictors of mean CBFv: PaCO₂, MABP, PaO₂, DOL, and estimated gestational age (Table 3). Since the

Table 2 Peak (Nadir) measures for physiological variables following tracheal suctioning

| Variable | Baseline to peak Baseline to peak Baseline to peak |
|---------|------------------------|------------------------|------------------------|
|         | (% Change)⁴ (Change)⁵ (min)² |
| Mean CBF velocity (cm/sec) | 31.0 ± 26.4 | 5.3 ± 4.5 | 11.4 ± 11.0 |
| MABP (mm Hg) | 13.7 ± 13.7 | 5.0 ± 4.8 | 12.2 ± 13.6 |
| PaCO₂ (mm Hg) | 12.7 ± 12.3 | 5.6 ± 5.1 | 13.1 ± 11.6 |
| PaO₂ (mm Hg) | (−22.4) ± 15.9 | (−15.8) ± 16.0 | 12.1 ± 14.4 |

Abbreviations: CBF, cerebral blood flow; CBFv, cerebral blood flow velocity; MABP, mean arterial blood pressure.

Mean ± s.d.
P-value for mode of ventilation was 0.937, it was removed from the multiple regression model.) All prospectively measured physiological variables were kept in the model, because they are conjectured to influence CBF in premature infants.25–29 DOL and estimated gestational age were included in the model, because they have been previously shown to be associated with CBF velocity.30,31 MABP and PaO₂ were not statistically significant predictors of the mean of mean CBFv (P = 0.434 and P = 0.148, respectively).

Whether or not any commonly considered confounding variables were included in the analysis, PaCO₂ (P < 0.001) was the only statistically significant predictor of mean CBFv. Coefficients for PaCO₂ did not change between models (range 0.290 to 0.303). Holding all other variables in the model constant, for a 10 mm Hg increase in the mean of PaCO₂, the mean of mean CBFv increases 2.90 cm s⁻¹ (95% CI (1.91, 3.89 cm s⁻¹)).

### Discussion

Experienced neonatal practitioners have long recognized that necessary routine neonatal intensive care unit procedures and practices, including tracheal suctioning, produce numerous undesirable effects on systemic hemodynamics and gas exchange of VLBW infants, which may also adversely affect the developing brain.1,19,32 Without prolonged and continuous cerebral and physiological monitoring (at least until disturbed variables return to baseline) during clinically indicated procedures, the duration of effects of tracheal suctioning on the cerebral circulation of...
ventilated premature infants was previously unknown. Additionally, while closed system tracheal suctioning,8,9 pre-sedation with phenobarbital,5,7 preoxygenation,4 briefly increasing peak inspiratory pressure, and/or using pressure-regulated volume control ventilation may be superior in mitigating physiological disturbances,9 we studied the effects of the method that we clinically used at the time.

About one-third of the suctioning sessions occurred when infants were receiving high-frequency ventilation. Although there were significant univariate differences between infants receiving high-frequency and conventional ventilation, when we entered mode of ventilation into the multiple regression analysis, it was not a statistically significant predictor of mean CBFv. Furthermore, while the measured mean airway pressure was higher for infants receiving high-frequency ventilation compared to conventional ventilation (7.9 ± 1.9 vs 6.3 ± 0.8 cm H2O), this was likely not clinically important, because our criterion for setting mean airway pressure for high-frequency ventilation is 1 to 2 cm H2O higher than mean airway pressure when on conventional ventilation. Therefore, data from suctioning sessions during both high-frequency and conventional ventilation were combined.

The average peak increase of middle cerebral artery mean CBFv following tracheal suctioning in our study sample was 31.0 ± 26.4%, which agrees with previous studies using Doppler ultrasound.1,5,14,16 Furthermore, all but 7% of VLBW infants had increases in mean CBFv following suctioning (without preceding decreases), which is consistent with observations by Perlman et al.1 Others using near infrared spectroscopy also observed brief increases in cerebral blood volume after tracheal suctioning.4,11 In this study, we observed that mean CBFv peaked at 6 min and remained elevated for approximately 25 min after suctioning. The duration of the CBFv increase that we observed is worrisome and is quite different than what was previously reported. While some investigators observed that CBF changes returned to baseline within 1 to 2 min after suctioning,4,9,10,11 most did not report the duration of CBF disturbances.5,10,14,16 Moreover, while the large difference in duration of CBF changes noted by us and others may be due to differences in population characteristics, inclusion of small sample sizes, different suctioning techniques, use of pre-oxygenation or sedation policies, and so on, we believe that the main difference and strength of our study is that we examined infants who clinically required suctioning and used continuous monitoring until disturbed physiological variables returned to baseline.

Since CBF increases have been shown to play an important role in the development of neonatal IVH,17,18 our results are concerning. On the other hand, since the 31% increase in mean CBFv that we observed following tracheal suctioning was lower than what was reported by others,17,18 we wondered whether our results were clinically significant. While we cannot answer this question because our sample of ventilated VLBW infants likely had intact autoregulation (no relationship between CBFv and MABP) and were free of significant brain injury (that is, VLBW infants at lower risk of adverse outcomes), we are concerned that increases in CBF following repeated suctioning procedures in intubated premature infants with impaired cerebral autoregulation could be associated with the development of IVH. Moreover, some intubated VLBW infants may have endotracheal suctioning performed dozens of times during the first week of life, and there are still centers that perform suctioning on a scheduled basis.

PaCO2 was a statistically significant predictor of mean CBFv, whereas MABP and PaO2 were not. Based on our model, a 10 mm Hg increase in PaCO2 would increase mean CBFv by 2.90 cm s−1; this represents an approximately 15 to 25% increase in mean CBFv (based on resting measurements of middle cerebral artery mCBFv in premature infants with similar gestational ages during the first week of life).31 The pattern and duration of change of mean CBFv following tracheal suctioning most closely mirrored the changes in PaCO2; this corroborates our previous observations following surfactant administration that changes in mean CBFv were highly associated with changes in PaCO2.19 Our results also agree with Skov et al.11 who observed in more mature and larger infants that changes in cerebral blood volume following tracheal suctioning were predominantly related to changes in PaCO2 and not due to changes in MABP or oxygenation. They suggested that the infants in their study may have had intact cerebral autoregulation. Another study in stable newborn ventilated premature infants undergoing intensive care (majority with normal cranial ultrasound findings) also observed that cerebral blood volume was independent of MABP, and was highly related to PaCO2.25 Others have also observed in premature infants that PaCO2 is an important regulator of CBF.26–28

Acute changes in MABP may also affect CBF, especially if premature infants have impaired cerebral autoregulation.29,33 In contrast to Perlman et al.,1 in our infant sample, MABP did not significantly influence mean CBFv, suggesting that cerebral autoregulation was probably intact in a majority of our sample group. We were not surprised by this observation since only normotensive VLBW infants with normal cranial ultrasound findings were enrolled in our study, and previous investigations have reported similar results.11,25 Also, PaO2 did not significantly influence mean CBFv. This was also expected since similar changes in PaO2 have much smaller effects on the cerebral vasculature than changes in PaCO2.34

Despite that a majority of previous studies also observed CBF increases, a few investigators reported decreases,9,10 or brief decreases followed by increased CBF after suctioning.15,16 One of the studies reporting CBF decreases had few patients, and the other had a majority of infants with IVH. In contrast, our study was relatively larger and all infants were free of IVH. Rieger et al.16 recently examined the effects of clinically indicated tracheal suctioning on the middle cerebral artery CBFv of normotensive extremely low birth weight infants. They observed at 16 to 20 s after
suctioning that CBFv decreased an average of 22 to 24% followed by an average increase of 20 to 31% from baseline that occurred at 26.1 to 31.4 s after suctioning. They concluded that the initial decrease may be due to cerebral venous congestion and increased intracranial pressure, but unfortunately did not discuss the subsequent increase. Furthermore, duration of the increase was not studied (S Kuhle, personal communication), baseline PaCO2 in their infants was quite high (66 ± 16 mm Hg), and higher wall suction was used (more than twice) compared to the other studies.

A possible limitation of this study is that we used transcranial Doppler ultrasound to measure CBFv instead of more direct measures of CBF. However, a good correlation between relative changes in CBFv and CBF was previously observed in neonates.[55] Transcranial Doppler ultrasound has the advantage of being noninvasive, easily performed at the bedside, and is ideal for making serial measurements from stable or sick infants. Furthermore, it does not require the use of radioactive materials, patient manipulations or alterations of oxygen concentration. Another limitation of this observational study was that we examined physiological variables only after clinically indicated suctioning (as subjectively determined by the bedside nurse) rather than after predetermined objective criteria. Thus, the baseline period may be somewhat different before each suctioning session and could possibly influence the results. On the other hand, we felt that this was the strength of our study, since we did not subject infants to procedures based solely on the investigators’ schedule or on our notion of when it was appropriate to suction, but left the decision to the ‘real-world’ decision makers.

Using continuous monitoring, we observed prolonged increases of CBFv following tracheal suctioning in relatively low-risk ventilated VLBW infants during the first week of life that were previously unrecognized. This is concerning since increases of CBF in premature infants and neonatal animals have been associated with brain injury,[16,17] especially those with hypotension and impaired cerebral autoregulation. To prevent swings in CBF, changes in PaCO2 during suctioning need to be prevented. It is unclear at this time, for instance, whether closed suctioning would mitigate increases in PaCO2 and reduce changes in CBF. What is clear is that, while necessary, tracheal suctioning should not be routinely scheduled, but performed on an ‘as needed’ basis in vulnerable VLBW infants during the first week of life.

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