

Suction-Induced Alveolar Instability: An Underexamined Pathway in Bronchopulmonary Dysplasia

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Although neonatal respiratory care has become increasingly focused on preserving lung volume and minimizing ventilator-induced injury, one common procedure remains a recurring source of physiologic disruption: endotracheal suctioning (ETS).

Bronchopulmonary dysplasia (BPD) remains a major morbidity of very preterm birth, and prolonged invasive ventilation is among its recognized risk factors.^{1,2} Over the last few decades, there has been a significant improvement in perinatal care with the introduction of surfactant and gentle ventilation to reduce lung injury, and survival of extremely preterm infants has increased. But the incidence of BPD has not changed, and it remains the most common late morbidity of preterm birth.²

This paper advances a hypothesis rather than a finding. We propose that repeated ETS may create recurrent episodes of lung derecruitment that accumulate across days or weeks of ventilation. We do not claim that suctioning causes BPD. However, we do ask the question: what is the cumulative effect on the developing lung when suctioning repeatedly removes lung volume and disrupts alveolar stability?

Current practice appropriately focuses on reducing immediate suction-related harm. The AARC guideline recommends suctioning neonates only when indicated, using shallow suctioning when effective, avoiding routine saline instillation, limiting the catheter to less than 70% of the endotracheal tube lumen, keeping applied vacuum below -120 mm Hg, and limiting suction to no more than 15 seconds.³ These safeguards address when suctioning is performed and how the catheter is used. They do not directly control the pressure, flow, or volume loss transmitted to the lung.

In neonatal endotracheal tubes, intratracheal pressure during suctioning is related to the applied vacuum, catheter size, and space between the catheter and tube.⁴ ETS causes acute loss of end-expiratory lung volume and tidal ventilation across lung regions, with catheter size exerting a major influence on the magnitude of the change.⁵ Electrical impedance studies also show that lung responses may remain altered after the procedure and that regional behavior can be heterogeneous even when global lung volumes recover.^{6,7} Thus, a suction event is not simply secretion removal; it is also a brief mechanical disruption of the ventilated lung.

This disruption is biologically plausible as an injury pathway, but it has not been shown to cause BPD. In an experimental surfactant-deactivation model, unstable alveoli developed ventilator-induced injury while stable alveoli did not.⁸ In extremely preterm infants, intermittent hypoxemia is associated with adverse respiratory outcomes, including severe BPD.⁹⁻¹¹ These studies support concern about repeated instability, but they do not establish that suction-related desaturation is equivalent to spontaneous intermittent hypoxemia or that it contributes materially to BPD.

That distinction defines the evidence gap. To our knowledge, no study has time-synchronized suctioning events with continuous oximetry to determine how much of an infant's total hypoxemia burden is attributed to the procedure. Likewise, cumulative suction exposure has not been evaluated as a predictor of BPD. As ventilator days accumulate, so do suctioning procedures; yet the dose, duration, and recovery from this repeated exposure remain largely unmeasured.

The vacuum source is a practical and testable variable. Conventional wall suction is pressure regulated, but the gas flow and negative pressure reaching the airway vary with catheter and endotracheal tube geometry.⁴ A controlled-flow peristaltic system limits gas extraction during secretion removal. In bench testing across endotracheal tubes from 2.0 to 8.0 mm, the controlled-flow system produced lower flow and lower peak and sustained negative pressures than wall suction.¹² In the neonatal-size subgroup (2.0-4.5 mm), secretion removal did not differ statistically ($P = .075$). The small sample and absence of a noninferiority design prevent conclusions about equivalent secretion clearance.

Preliminary clinical findings point in the same direction. A prospective, nonblinded crossover study reported 40 suctioning events in 10 mechanically ventilated preterm and term neonates. Compared with conventional wall suction, controlled-flow suctioning was associated with a smaller mean decrease in SpO₂ (-5.6% vs -11.3%, $P < .0001$) and heart rate (-17.2 vs -26.5 beats/min, $P = .0005$), with better post-procedure recovery.¹³ These significant data are encouraging but exploratory. The small sample, nonblinded design, event-level paired analysis, and absence of a peer-reviewed full report limit conclusions about effect size, repeated exposure, and clinical outcomes.

Important limitations must be considered when evaluating this hypothesis. Existing hypoxemia studies did not examine suction-related events, which are often shorter than the episodes associated with BPD.¹⁰ Disease severity is also a major confounder because sicker infants remain ventilated longer, are suctioned more frequently, and are more likely to develop BPD. In addition, repeated suctioning did not produce histologic injury in a short-term rabbit model.¹⁴ Because BPD is multifactorial, any contribution from ETS may be modest. This hypothesis does not support less suctioning; it supports investigating the impact of necessary suctioning when the procedure is less disruptive.

We believe there are a couple of research priorities. The first research priority should be descriptive, not therapeutic: synchronize suction-event logs with high-resolution pulse oximetry and quantify the proportion of total hypoxemia burden attributable to ETS. If that contribution is negligible, the hypothesis weakens. If it is meaningful, the second priority is a randomized crossover study using electrical impedance tomography that could compare conventional and flow-limited suctioning for end-expiratory lung volume loss, regional ventilation, desaturation burden, and recovery time. Only after the exposure and physiology are established should cumulative suctioning be tested against BPD outcomes using methods that address time-varying disease severity and confounding by indication.

Suction-induced derecruitment is frequent, iatrogenic, and potentially modifiable, but its cumulative burden has not been quantified. We do not claim that ETS causes bronchopulmonary dysplasia. We propose that repeated suction-related instability is a plausible, measurable exposure that deserves focused investigation.

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