Letters to the Editor

Failing Jet Ventilator in A Small Premature Infant

A male infant was born at 26 5/7 weeks gestation to a 29-year-old G2P0010. The dates were confirmed with an 8wk ultrasound. Pregnancy was complicated by class B diabetes. The fetal US showed omphalocele and 2 vessel cord. Amniocentesis was negative for FISH and Microarray tests. Due to short cervix, a cerclage was placed. She was started on antibiotics, steroids, and magnesium. While under observation, the fetal heel was found to be protruding from the cervix, so an urgent C-section was performed.

At delivery, the infant was immediately intubated, omphalocele was covered with sterile gauze, and the infant was then transported to the NICU, where she was placed on HFOV. Physical exam

showed a weight of 995 grams, head circumference of 24 cm, and length of 34.5 cm. The infant was vibrating well on HFOV. A large omphalocele was noted. There was no heart murmur. Back showed thoracic scoliosis. No other congenital anomalies were noted. Pediatric surgery, due to the large mass and prematurity, decided to manage conservatively.

The infant was placed on HFOV with settings of MAP 12, amplitude of 30, 15 Hz and IT 33%. He received surfactant twice. On day 4, he was noted to have an increased oxygen requirement. A trial of rescue inhaled nitric oxide was started with minimal benefits. Chest x-ray (CXR) showed persistent right upper lobe atelectasis (Figure 3). On day 7, a decision was made to try HFJV.

A CXR soon after starting Jet showed expansion up to 11 ribs with a resolution of RUL atelectasis (Figure 5). Initial settings were JET: PIP = 18, rate 420, IT 0.02 secs, I:E 1:6.1, CMV: rate 3,

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PEEP 7, PC 10, IT 0.35. Blood gas 20 minutes after placing on these settings was 7.28/PCO₂ 63/PaO₂ 91.

Follow up blood gases showed respiratory acidosis with CO₂ retention. CXR showed RUL atelectasis again (Figure 6). ETT was adjusted with no benefits. A trial of higher exhalation time on Jet (rate 360, I:E 1:7.3) was unsuccessful. Servo pressure remained stable at 2. The infant remained on 100% Oxygen with sats of 94-95%. The infant was then switched back to previous HFOV with the resolution of respiratory acidosis. The infant is currently stable on HFOV.

The questions for experts are (where we went wrong with HFJV):

With expansion up to 11 ribs (Figure 5), should we have tried higher PIP or PEEP? If yes, what is the risk?

Would a higher rate on JET have made a difference?

Should we have tried CPAP on CMV (0 rates)?
Any other suggestions?

Sincerely,
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Dear Dr. Manzar:

Thank you for sending this interesting case for analysis. The transition from one mode of ventilation to another mode is challenging. Although it appears that the presence of non-uniform lung disease and hyperexpansion on the chest radiograph would favor the transition from HFOV to HFJV, the patient did not have a favorable result and was transitioned back to HFOV.

First, let us discuss the HFOV strategy using HFJV physiology arguments. With a mean airway pressure of 12 cm H₂O and amplitude 30 cm H₂O, the patient is at risk for airway collapse or "pinch" during exhalation. Remember, during HFOV, exhalation is active, not passive. Amplitude of 30 exceeds the mean airway pressure of 12 H₂O by 18 cm H₂O. Although the point at which the process becomes a concern is a matter of debate, at greater than twice the mean airway pressure, both airway pressure and flow can become negative at the wye. Increased airway resistance prevents alveolar collapse, but the premature compliant airways are prone

to collapse. This may subsequently lead to alveolar collapse, as noted in the right upper lobe, the most "dependent" lobe, as well as generalized hyperinflation, as the inspiratory flow is trapped by the "pinched" airway. Instead of going to HFJV, an alternative strategy on HFOV might have included "paradoxically" increasing the mean airway pressure to insufflate the airway and prevent negative flow and pressure at the wye, decreasing the amplitude, and proning the patient.

When transitioning to HFJV, one has to remember that the HFJV amplitude is approximately half that of HFOV at similar delta P because the HFOV deflection is both negative and positive. If 7 H₂O is subtracted from 18 H₂O, the HFJV amplitude is 11 cm H₂O; with the correction applied – that is, $30 \times 2 = 60$ peak amplitude negative to positive for HFOV, the relative amplitude supplied by HFJV is approximately one-sixth of HFOV. Although the elimination of active exhalation resolved the right upper lobe atelectasis, there was just not enough delta P to achieve appropriate ventilation. A jet PIP of 40 would have been more appropriate. More time, as opposed to lower or higher mean airway pressure, would have helped the hyperexpansion. CMV is often a surrogate for mean airway pressure in HFJV. A CMV of 0 would not have helped this situation, but in the long term would have exposed the patient to less barotrauma. The jet rate change may not have produced a significant effect in this patient.

Although HFJV can be challenging, a successful application of its strategies can lead to substantive improvements in ventilation. Conversely, HFOV physiology favors higher mean airway pressures, lower amplitudes, and in some cases proning the patient when atelectasis and hyperinflation are present.

Sincerely,

Mitchell Goldstein, MD
Editor in Chief

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