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The Measurement of Expired Oxygen as Disconnection Alarm

To the Editor—The method reported by Doctors Knack-Steinpegger and Thomson was long ago described as an unreliable method to detect disconnections, except under ideal conditions. At other times, especially when the disconnection site is covered with drapes, the anesthetic mixture collects there and is drawn back into the expiratory circuit by a falling bellows ventilator, where little loss of oxygen may be detected. With a rising bellows ventilator, flow terminates in the expiratory limb of the circuit with similar results.

Modern ventilation monitors, such as carbon dioxide monitors and exhaled volume monitors, specified for anesthesia systems by the ASTM F 29 standard, are readily available and have proven their reliability as disconnection alarms. Users should not depend on a less-reliable device for this critical function.


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REFERENCES


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In Reply—We do agree with Dr. Spooner that users should not depend on less-reliable devices to detect disconnections within the anesthetic circuit. However, Spooner did not prove our method to be unreliable but only stated that not all disconnections occur under ideal circumstances and that in the case of a tracheal tube disconnection under drapes, detection may not be made with an oxygen analyzer. We have data to show that our method is reliable, even when a disconnection occurs under drapes (table 1).

The data shown in table 1, columns I and III, were the figures that we published. The data shown in columns I* and III* were collected at the same time that we did the original work. In columns I* and III*, we show the data that we received when we disconnected the circle under drapes with an additional covering of a pillow to make the area even more airtight. These circumstances are somewhat difficult to standardize; therefore, we did not intend to publish these data. Nevertheless, the detection of a disconnection is reliable even under these difficult versus ideal conditions.

The data show that the reliability of the method changes considerably when the alarm settings are not close to the expected expiratory oxygen concentration as stated in the Discussion of our paper.

We use the oxygen measurements in the expiratory limb to monitor the amount of oxygen that our patients receive, not only what the machine delivers. In the same way, we use the spirometer in the expiratory limb to monitor the amount of volume that the patients re-
**TABLE 1.** Alarm Times (Mean Seconds ± SD, n = 5) at Different Fresh Gas Flows, Tidal Volumes, and Disconnection Conditions

<table>
<thead>
<tr>
<th>Tidal Volume</th>
<th>10 Breaths per Min</th>
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<tbody>
<tr>
<td></td>
<td>I</td>
</tr>
<tr>
<td>500 ml</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>17.8 ± 0.4</td>
</tr>
<tr>
<td>B</td>
<td>22.1 ± 1.4</td>
</tr>
<tr>
<td>1000 ml</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>11.6 ± 1.3</td>
</tr>
<tr>
<td>B</td>
<td>13.2 ± 1.3</td>
</tr>
</tbody>
</table>

1) Nitrous oxide:Oxygen = 2:1, 3 l/min fresh gas flow. Oxygen concentration: 33%. Alarm limit: 30%.

III) Nitrous oxide:Oxygen = 2:2, 4 l/min fresh gas flow. Oxygen concentration: 50%. Alarm limit: 30%.

A) Measurements with monitor A: Anemone, Draeger.
B) Measurements with monitor B: Mono 2, Kontron, Zurich, Switzerland.

* Disconnection under drapes (see text).

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**REFERENCES**


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**Macroglossia Causing Airway Obstruction following Cleft Palate Repair**

To the Editor—Cleft palate repair is a common procedure in the pediatric population and complications are unusual. There have been three cases of severe macroglossia following cleft palate repair previously reported. We recently encountered two cases of life-threatening airway obstruction due to macroglossia following this repair.

**Case 1.** A healthy 10 kg, 20-month-old girl was scheduled for cleft palate repair. Following iv methohexital, atropine, and succinylcholine, the trachea was easily intubated with a 4.0 ETT. Anesthesia was maintained with oxygen, nitrous oxide, and enflurane. No additional neuromuscular blocking agents were administered. She received penicillin 600,000 U intravenously and her palate was infiltrated with 6 ml of 0.5% lidocaine with 1:200,000 epinephrine. A Dinman retractor was in place during the entire 3 h 45 min procedure. The estimated blood loss was 110 ml and she received 800 ml of D51/2NS. At the conclusion of the procedure the child was awakened and the trachea was extubated. She was unable to breathe immediately after extubation and the lungs were difficult to ventilate by mask due to severe lingual edema. Reintubation was extremely difficult and required five attempts. She was taken to the intensive care unit while sedated and with her trachea intubated. Extubation was not tolerated on the first and fourth postoperative day. Endoscopy revealed a swollen tongue and supraglottic region. Ultrasonography of the tongue showed homogeneous tissue without evidence of abscess or other cystic abnormality. Persistent edema precluded tracheal extubation until the eleventh day postoperatively.

**Case 2.** A healthy 9.5 kg, 13-month-old boy was scheduled for bilateral cleft palate repair. Following an inhalation induction with oxygen, nitrous oxide, and halothane iv infusion was started and the child received atracurium, 5 mg. His trachea was easily intubated with a 4.0 ETT and anesthesia was maintained with oxygen, nitrous oxide, and isoflurane. Shortly after induction, 25 μg of fentanyl and 0.35 mg of droperidol were also given. Other medications administered were cefazolin 125 mg iv and 3 ml of 0.5% lidocaine with 1:200,000 epinephrine infiltrated into the palate. A Dinman retractor was in place during the entire 3 h 40 min procedure. The patient lost 30 cc of blood and received 340 cc of RL. At the conclusion of surgery he received neostigmine 0.8 mg and glycopyrrole 0.1 mg iv. His last atracurium dose had been 2 h earlier. Upon extubation he immediately showed signs of upper airway obstruction. The lungs could not be ventilated despite jaw thrust and traction on the tongue suture but the trachea was easily reintubated. The tongue and floor of mouth were edematous. Dexamethasone 2 mg was given iv and he was taken to the ICU while sedated and with the lungs mechanically ventilated. Nine hours postoperatively the child removed the endotracheal tube. The edema had worsened and the tongue had become hard and immobile, filling the mouth. Again the lungs could not be ventilated despite jaw thrust, traction on a tongue suture, and oral airway insertion. Reintubation was extremely difficult. Multiple attempts at intubation were unsuccessful and he sustained a cardiac arrest. Cricothyroid puncture was also unsuccessful but an endotracheal tube was eventually inserted. While the child survived, and although eventually the trachea was extubated, he never regained consciousness.

The average time for cleft palate repair in our institution is 2½ h. The operative times of our two patients and the others that developed macroglossia were greater than 3½ h. A longer procedure implies longer retractor times, a more difficult repair and perhaps other coexisting pathology. A longer procedure may also increase the effects of fluid administration, blood loss, and position.

While it is difficult to make quantitative comparisons among cases from different institutions, it is our opinion, after reviewing the five cases, that the development of macroglossia is related to the length of surgery. We strongly agree with the recommendations of Bell, Oh, and Loeffler that following cleft palate repair the mouth and tongue should be examined prior to extubation. In lingual or sublingual edema...