Insufflation Anesthesia for Suture Removal after Cheiloplasty

To the Editor.—Although suture removal after cheiloplasty in infants and young children is typically a brief procedure (2–5 min), motionlessness of the patient is desirable because of the small size of the suture. In the past, we have administered halothane in oxygen via face mask, with the patient breathing spontaneously. When the level of anesthesia was considered adequate, the mask was removed, and the surgeons proceeded with the suture removal. However, on occasion, the procedure took longer than the anticipated time, resulting in a light level of anesthesia. When this occurred, the risk of laryngospasm or patient movement was greatly increased. To avoid this problem, we have employed an old but still useful technique, insufflation, which allows us to remove the mask and still maintain a deep level of anesthesia.

Patients are monitored with an electrocardiogram, a noninvasive automated blood pressure measuring device, a pulse oximeter, and a precordial stethoscope. A mask induction with halothane in nitrous oxide/oxygen is used to establish a deep level of anesthesia. The mask then is removed, and a 4.0-mm ID oral RAE tube with approximately 8 cm cut off the distal end is placed from the corner of the patient’s mouth into the oropharynx (avoiding the surgical field). Care is taken to curve the tube short enough so that it does not impinge upon the larynx. Through this we administer 2% halothane in oxygen at a high flow rate (5–8 l/min).

The absence of a reservoir during insufflation of anesthetic agents hinders assessment of the depth of respiration and precludes the ability to assist ventilation. Other problems are variable dilution of anesthetic by inspired room air; drying of tracheal mucus; loss of heat and water; anesthetic waste; and difficulty in scavenging the expired gases. However, the technique is simple and unobtrusive and provides the necessary depth of anesthesia. Tracheal intubation is unnecessary, and recovery is faster than after an equivalent amount of intravenous sedation. By delivery of flow rates that approximate the peak inspiratory flow rates of these patients,1 dilution of the anesthetic by room air is minimized. Because of the short duration of the procedure, pollution of the operating room is not a significant problem.

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Reference
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Nitrous Oxide Abuse Presenting as Premature Exhaustion of Sodosorb

To the Editor.—The recent letter by Yudenfreund-Sujka1 on nitrous oxide abuse as presenting as premature exhaustion of Sodosorb was interesting, but the proposed reason for the exhaustion is unlikely.

The Sodosorb Pre-Pak (Dewey and Almy Chemical Division, W. R. Grace, Co.) absorbs approximately 23 l of carbon dioxide per 100 g. The Sodosorb Pre-Pak has in it 2.5 lb, or 1,131 g. Therefore, the capacity is 23 l/100 g × 1,131 g, or 260 l.

The letter indicates that within 3 days (72 h), five of six Sodosorb canisters needed to be changed. The total capacity of five canisters would be 5 × 260 l, or 1,300 l.

A 70-kg person produces carbon dioxide at a rate of approximately 200 ml/min or 12 l/h.2 Even if the system used was totally closed and required 100% of the carbon dioxide to be absorbed, it would take slightly more than 108 h (1,300 l ÷ 12 l/h) to exhaust the Sodosorb.

Although this is the theoretical absorptive capacity, in practice, less than 50% is consumed by the time it is useless clinically.* This obviously reduces the time needed to exhaust the Sodosorb, but in a semi-closed system, without the requirement to absorb 100% of the carbon dioxide, this increases the time needed.

Thus, it seems highly unlikely one person, in the context of the time frame mentioned in the letter, could have exhausted the Sodosorb canisters.

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References
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In Reply—I do not wait until the Sodasorb is completely exhausted before I change it. I personally change it at the first sign of the indicator showing a change of color. I do this for two reasons. Since we are a small department and have no anesthesia technician, I do not want to need to change the Sodasorb in the middle of a long case. In addition, it has been reported that ethyl violet in Sodasorb can be photodeactivated by the fluorescent lighting in the operating room. Therefore, this indicator of Sodasorb exhaustion may not be 100% reliable.

When the problem of premature exhaustion first arose, I believed it was due to the possible poor quality of the Sodasorb, but since the employee in question was discharged, our present stock of Sodasorb is performing as it had in the past. In lieu of this, I have no other explanation for the premature exhaustion of our Sodasorb except for the nitrous oxide abuse by the employee.

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REFERENCE
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If Ventricular Conduction and Rhythm Disorders Are Caused by Bupivacaine, It Is Doubtful That Intraoperative Hyponatremia and Hyperkalemia Enhance Them

To the Editor—Timour et al., after maintaining constant arterial plasma concentrations of bupivacaine of 2.2–3.7 μg/ml concluded: “To the extent that animal data can be extrapolated to humans, we believe that if significant intraoperative hyponatremia or hyperkalemia are present (or are likely to occur), anesthetic techniques that might lead to high blood concentrations of bupivacaine, e.g., epidural or brachial plexus block, should be used with caution. Hyponatremia or hyperkalemia could add to or even potentiate bupivacaine-induced inhibition of intraventricular conduction and result in serious rhythm disorders.”

Their investigation is markedly dissimilar to the clinical situation. Sustained arterial plasma concentrations of 2.2–3.7 μg/ml do not occur in patients after administration of 125–225 mg bupivacaine 0.5–0.75% for epidural block (lumbar n = 20, caudal n = 6) or from 300 mg 0.5% for brachial plexus block (n = 10). Concentrations as great as 2.4 μg/ml may occur with these blocks in 15–30 min. However, in another 15–30 min, they are less than 1.9 mg/ml, and they decrease with each elapsed minute.

Therefore, allowing time from injection to establishment of operating anesthesia (15 min), draping of the patient (15 min), and the occurrence of hyponatremia or hyperkalemia intraoperatively, it is highly unlikely that the bupivacaine concentrations maintained in dogs could ever occur clinically. Even if they did, and even if hyponatremia or hyperkalemia or both occurred intraoperatively or existed prior to a regional block, our published data do not support the thesis of Timour et al. Do they have clinical data? If so, citing them would be appreciated.

To conclude, anesthesiologists should be aware of the following: Animal data cannot be extrapolated to humans. They are only a rough guide line to the situation in humans. Furthermore, as stated some years previously, “Believing in medicine is not enough, one must know.” “Extrapolating” or “believing” is extremely dangerous and only leads anesthesiologists eventually to state that animal data are facts pertinent to humans, when no clinical proof exists. As a result, techniques and drugs that may be the anesthesia of choice are not used. Perhaps most important is that such extrapolating has in the past, and likely will in the future, result in the twisting of facts in a medicolegal case to serve a purpose for which they may not be intended.

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REFERENCES