Tympanic Membrane Rupture after Anesthesia with Nitrous Oxide

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Increase in middle ear pressure during anesthesia with nitrous oxide and oxygen has been reported by many authors,¹-⁵ and may be the primary cause for complications such as temporary or permanent hearing impairment, hemotympanum, stapes disarticulation, tympanic membrane rupture, serous otitis, and displacement of tympanic membrane graft.²,⁴,⁶,⁷

As part of an on-going study,⁸ we were measuring middle ear pressures in several patients. One such patient experienced very high middle ear pressures intraoperatively and significant negative pressures in the recovery room that apparently caused a rupture of her tympanic membrane. Consistent with our case, negative middle ear pressures have been observed following elimination of nitrous oxide by other authors.⁴,⁹

REPORT OF A CASE

A 46-year-old woman, was selected randomly to participate in this study. An informed consent was signed to undergo all the pre- and postsurgery ear and hearing evaluations, and to have middle ear pressure measured with an impedance bridge during surgery. She had arterial hypertension caused by an atrophic kidney and was to undergo a right nephrectomy.

She had frequent discharges in both ears during childhood. Prior to surgery, she complained of hearing loss, occasional ringing in the ears, and pain when exposed to cold temperatures. Audiologic evaluation showed hearing to be within the normal range for her age at the frequencies tested (125 Hz to 8,000 Hz). Tympanometric examination of the left ear, revealed high amplitude tympanograms for both probe tones (220 Hz and 660 Hz) and the presence of a W-shaped tympanogram for the 660 Hz susceptibility measurement which is compatible with a flaccid tympanic membrane.¹⁰ Eustachian tube dysfunction was evidenced by the use of the inflation-deflation technique of Williams.¹¹ Eardrum examination showed a strip of tympanosclerosis (5 mm X 1.5 mm) on the anterior border of the right ear and a large central "neo-membrane" of about 6 mm of diameter; in the left ear a central "neo-membrane" of 3 mm of diameter was present.

Anesthesia was induced with 20 mg diazepam, 0.05 mg fentanyl, 6 mg pancuronium, and 200 mg thiopental, iv. The trachea was intubated and anesthesia maintained with 66-70% nitrous oxide and 0.3 to 1.0% inspired halothane.

During anesthesia, the left ear was submitted to indirect measurements of middle ear pressure with the impedance-bridge Grason-Stadler, 1723, Version II.¹² After thirty minutes, middle ear pressure increased to over 40 cmH₂O and remained at this level for almost the entire course of anesthesia (fig. 1). After emergence from anesthesia in the recovery room, her middle ear pressure decreased rapidly to −45 cmH₂O after thirty minutes (fig. 1). At this time, normal pressure curves disappeared on the right, and left tympanograms which indicated that the tympanic membranes did not respond normally lead us to suspect a tympanic membrane rupture (fig. 1). An otoscopic eardrum examination revealed a perforation of about 2 mm of diameter located in the middle of the neomembrane seen during preoperative examination of the right ear. The tympanic rupture was on the right side, opposite to the site of impedance measurement during surgery. The patient complained of ringing in her ear for two days after the operation. Forty-five days later, a hearing evaluation did not show noticeable change from the first examination on both ears. The patient complained of light pain in the ears which was greater on the right side. Microscopic eardrum examination showed wide neomembrane on both ears.

DISCUSSION

Tympanic rupture has been reported during anesthesia with nitrous oxide.⁷ Tympanic perforations can occur under wide variations in middle ear pressure caused by diffusion of nitrous oxide in and out of the middle ear. Diffusion is mainly due to a great difference in blood solubility between nitrous oxide and nitrogen. Ducke et al.⁵ has mentioned the blood vessels, tympanic membrane, and eustachian tube as the three possible sites where diffusion of gases in and out of the middle ear occurs. There are important increases in pressure during nitrous oxide administration, and sometimes important

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decreases during the recovery period, especially when the eustachian tube is obstructed. Tympanic rupture in this patient occurred while a negative pressure existed in the middle ear.

In our case, two factors may be responsible for the tympanic rupture. First, the eustachian tube was obstructed, resulting in negative pressure during the recovery period from anesthesia. Second, the patient had a healed perforation in both ears. The presence of a healed perforation on the eardrum weakens the tympanic membrane and increases the risk of rupture under the stress of negative or positive pressure. The eustachian tube obstruction may generate wide pressure variations, and in association with a healed perforation on the eardrum, may cause tympanic rupture.

The negative pressure in the middle ear observed in patients with an obstructed eustachian tube leads us to think that equilibration of middle ear during elimination of nitrous oxide is achieved by venting of eustachian tube. Obstruction of the tube does not permit a rapid return of nitrogen because of the great difference in diffusion between nitrous oxide and nitrogen. This differs from Duerer's opinion, who observed diffusion of nitrous oxide through the tympanic membrane. Clinically, we have observed negative pressure for as long as 48 hours following anesthesia.

We conclude that the use of nitrous oxide in high concentration should be avoided in patients having a pathologic problem of the middle ear. A physiologic gas mixture should be used which prevents diffusion into or out of the middle ear. In our opinion, the best mixture is air and oxygen with a volatile anesthetic because they are used in low concentrations and do not establish a high diffusion gradient. This technique obviously may be facilitated by the use of the intravenous anesthetics.

REFERENCES