Tympanic membrane rupture following general anesthesia with nitrous oxide: A case report

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Although rare, tympanic membrane rupture during general anesthesia with nitrous oxide has been reported previously in the literature. Nitrous oxide administration and the effects on closed body cavities will be reviewed. Key factors in patient assessment which can determine safe use of nitrous oxide in the clinical setting will also be discussed.

Key words: Closed space expansion, eustachian tube patency, nitrous oxide, tympanic membrane trauma.

Introduction
This case report involves a young female undergoing laparoscopic surgery with a history of a recently resolved respiratory tract infection. Tympanic rupture was noted 1 hour postoperatively which was probably due to administered nitrous oxide.

Case report
A 28-year-old white female weighing 65 kg underwent a dilatation and curettage (D&C) and diagnostic laparoscopy. Chief complaints consisted of lower abdominal cramping and heavy vaginal bleeding for several days. Previous surgical history included appendectomy, laparoscopy, removal of uterine fibroids, tonsillectomy, and two D&Cs. Medical history included gravida IV, para I, abortion II, and spontaneous miscarriage.

The patient had a recent respiratory tract infection which was resolved 10 days prior to surgery. She smoked one pack of cigarettes per day for several years. Physical examination was unremarkable. Patient denied any recent ear discomfort or a past history of ear problems. Mallampati airway was assigned at Class II. She had a significant anxiety disorder for which she took clonazepam and imipramine hydrochloride. Complete blood count was within normal limits, urinalysis was negative except for trace of protein, and chest x-ray was negative.

Premedication consisted of fentanyl 100 μg and midazolam 2 mg intravenously. Induction agents included lidocaine 60 mg, propofol 140 mg, succinylcholine 100 mg, fentanyl 50 μg, and oxygen. Intubation was atraumatic but required three attempts due to an anterior larynx. Maintenance of anesthesia consisted of nitrous oxide 50%, oxygen 50%, isoflurane 1%, and mivacurium 12 mg and was without incident. Ketorolac 60 mg was given intramuscularly within 10 minutes after intubation.

The total length of the case was 20 minutes. Emergence was smooth. Glycopyrrolate .4 mg and pyridostigmine 10 mg were given intravenously for antagonism of muscle relaxation. Minimal blood loss was noted. Total intravenous fluid administration was 1,400 mL.

The patient began spontaneous ventilations, was extubated, moved herself onto the gurney, and was transferred to the postanesthesia care unit.
(PACU) in stable condition. Peripad and surgical laparoscopy sites were dry. Vital signs were stable and oral temperature registered at 98°F. Approximately 1 hour postadmission into the PACU, the patient complained of left ear pain with altered hearing. Small amounts of blood were noted coming from the left external auditory canal. The patient was immediately referred for ear, nose, and throat (ENT) consultation.

ENT findings included perforation of the eardrum due to barotrauma otitis. The patient was placed on oral antibiotics for 3 weeks to relieve the eardrum and canal congestion.

The patient was sent for auditory testing after complete antibiotic therapy. The results of the auditory studies revealed a mild conductive hearing loss rising to within normal limits for the test frequency of 2,000-8,000 Hz in the left ear. The right ear studies were within normal limits. The ENT specialists felt that complete hearing would return within 2-3 months.

Discussion

Hearing loss following nitrous oxide anesthesia has been reported as a potential complication of general anesthesia. Authors Miller and Barash have both reported acute hearing loss and tympanic membrane rupture following nitrous oxide use. Studies done by Mann and associates demonstrated a correlation between nitrous oxide use and auditory complications. A study concerning nitrous oxide use and tympanic membrane rupture was performed by Waun and colleagues who noted that they have seen spontaneous rupture of tympanic membranes but did not describe types of patients or nitrous oxide concentrations.

Both compliant and noncompliant body spaces are subject to pressure-volume changes with nitrous oxide transfer. The compliant spaces, such as the bowel and pneumothorax, are especially subject to volume changes with nitrous oxide transfer. Noncompliant body cavities include the eye (when a vitreal bubble has been added) and the middle ear. Middle ear and paranasal sinuses are normal air cavities and represent open, nonventilated air cavities.

These spaces normally contain nitrogen, a gas whose blood gas partition coefficient is .014. In contrast, nitrous oxide has a blood gas coefficient of .47. This differential solubility means that nitrous oxide can leave the blood to enter an air-filled cavity 34 times more rapidly than nitrogen can leave the cavity to enter the blood. This transfer of nitrous oxide results in increased pressure or volume of the air-filled cavity. The entrance of nitrous oxide into an air-filled cavity surrounded by a compliant wall (intestinal gas, pneumothorax, pulmonary blebs, or air embolism) causes the gas space to expand. Conversely, entrance of nitrous oxide into an air-filled cavity surrounded by a noncompliant wall (middle ear or cerebral ventricles) causes an increase in pressure.

The magnitude of volume or pressure increase is influenced by the alveolar partial pressure of nitrous oxide, blood flow to the air-filled cavity, and duration of nitrous oxide administration. An alveolar concentration of 50% nitrous oxide might double the gas space volume while a 75% concentration can produce a four-fold increase. When inhaling high concentrations, nitrous oxide will enter these cavities faster than nitrogen removal. In a fixed cavity, such as the middle ear, this results in increased pressure.

Normally, the eustachian tube drains the middle ear and allows for its ventilation by a ball-valve-like action and movement of cilia. The direction of flow through the ball valve is from the middle ear to the nasopharynx. Contraction of dilator muscles of the eustachian tube occurs with yawning and swallowing and allows for equalization of atmospheric and middle ear pressures. Passive opening of the tube occurs when the middle ear pressure reaches approximately 200-350 mm H2O pressure, thus relieving tympanic cavity pressures.

In anesthetized patients, the flow of gases between the middle ear and eustachian tube depends upon the passive action of the ball valve mechanism. Many patients have narrow eustachian tubes, due to inflammation, infection, or scar tissue after an adenoidectomy, which can lead to inability to relieve the middle ear pressure increase passively. These patients are potentially susceptible to pressure-related middle ear damage due to nitrous oxide administration.

Patients with normal eustachian tube function who undergo general anesthesia with nitrous oxide experience venting of the auditory tube at pressures of 200-350 mm H2O pressure. If the auditory tube is traumatized by surgery, disease, or inflammation, middle ear pressure may reach 375 mm H2O within 30 minutes of nitrous oxide exposure. Because of the rapid pressure change, stapes disarticulation, serous otitis media, and impaired hearing can result. Additionally, nitrous oxide can be hazardous to patients with previous middle ear surgery who have concurrent problems with eustachian tube patency.

Hearing impairments have been reported after nitrous oxide anesthesia. Waun and colleagues investigated compliance of the tympanic membrane during nitrous oxide anesthesia. This
study demonstrated hearing loss of 5-10 decibels at lower frequencies in patients after adenoidectomies. Patterson and Bartlett reported hearing impairments due to developed oval window fistula, hemotympanum, and disruption of ossicular reconstruction. Cases of actual rupture of the tympanic membrane have been reported and attributed to the use of nitrous oxide. Tympanic membrane grafts can also be dislodged through the use of nitrous oxide.

Cessation of nitrous oxide after closure of an open body cavity, such as the ear, produces a negative pressure due to the rapid efflux of nitrous oxide. A negative pressure of 275 mm H₂O can be present 75 minutes after nitrous oxide cessation. When a patient begins to breathe room air during the recovery period, the rapid diffusion of nitrous oxide from the middle ear cavity into the blood and a slower diffusion of nitrogen for its replacement causes a negative middle ear pressure. This sustained subatmospheric pressure could cause tympanic membrane injury, trauma, etc.

Mann and associates demonstrated a relation between nitrous oxide and middle ear pressure. The effect of three different anesthetic carrier gases on middle ear pressure in the operative and postoperative periods were assessed. Patients receiving oxygen and oxygen-enriched air did not have significant changes in middle ear pressure. Changes in middle ear pressure were associated with the use of nitrous oxide as the carrier gas. These changes were reversed after inhalation of 100% oxygen or with return of pharyngeal reflexes.

**Summary**

Nitrous oxide has been shown to diffuse into the middle ear during anesthesia causing pressure increases with the middle ear. Alternatively, at the conclusion of anesthesia, the rapid efflux of nitrous oxide results in a negative inner ear pressure. These pressure changes tend to equalize out postoperatively in most cases. However, reports and studies have demonstrated that in patients with recent respiratory tract infection, sinusitis, previous ear surgery, hearing problems, acute and chronic otitis media, and enlarged adenoids, increases in middle ear pressure can result. This increase in pressure can result in postoperative hearing loss and/or tympanic membrane rupture.

In conclusion, caution should be exercised with nitrous oxide use in patients with known disease processes that may affect the eustachian tube structure and patency. Anesthetists should consider avoiding nitrous oxide in patients with tympanic membrane grafting, in those with a history of previous surgery of the middle ear, and in those with acute or chronic processes which may adversely affect eustachian tube patency.

**REFERENCES**


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