J Anesth (1998) 12:17–20

Journal of
Anesthesia

Middle-ear pressure variations during total intravenous anesthesia with propofol, fentanyl, and ketamine

TAKESHI KUBOTA[1], KAZUYOSHI HIROTA[2], NORIAKI OTOMO[1], TADANOBU YASUDA[1], AKIMASA MIYATA[1], ASAHEI MAEDA[1], HIRONORI ISHIHARA[2], and AKITOMO MATSUKI[2]

[1] Department of Anesthesia, Hachinohe City Hospital, 1 Bishamondaira, Tamukai, Hachinohe, 031 Japan
[2] Department of Anesthesiology, University of Hirosaki School of Medicine, Hirosaki, 036 Japan

Abstract

Purpose. As the middle-ear cavity is one of the noncompliant gas-filled cavities, an increase in middle-ear pressure (MEP) instead of volume expansion is observed with inhalation of nitrous oxide (N_2O). Changes in MEP cause many complications, such as ear pain, temporary hearing impairment, and postoperative emesis. Therefore, we investigated changes in MEP during total intravenous anesthesia (TIVA) with propofol, fentanyl, and ketamine (PFK) and inhalation of N_2O.

Methods. Twelve patients were anesthetized with PFK until 60 min after the induction of anesthesia, and then N_2O (60%) inhalation was started. MEP was measured by impedance audiometry (ranging from −300 daPa to +200 daPa) at 10-min intervals during PFK, and at 2-min intervals after the inhalation of N_2O.

Results. MEP gradually but significantly increased from the preanesthetic value of 16 ± 8 to 34 ± 12 (SEM) daPa 50 min after the induction of PFK. However, MEP did not exceed the normal limit. The values of MEP in all patients were more than 200 daPa within 36 min after the start of inhalation of N_2O in oxygen.

Conclusion. PFK had a minimal effect on MEP, whereas addition of N_2O to PFK increased MEP dramatically. Therefore, TIVA, or at least PFK, would be a better choice for patients with middle-ear or upper-airway diseases.

Key words: Middle-ear pressure, Total intravenous anesthesia, Propofol, Fentanyl, Ketamine

Introduction

Nitrous oxide (N_2O) is known to produce changes of volume in complaint gas-filled cavities. When 50% N_2O is present in the alveoli, the body gas space must be doubled; similarly, at an alveolar concentration of 75% N_2O, the gas space must increase fourfold [1]. The volume change occurs because the rate of diffusion of N_2O from the blood into the gas-filled cavities exceeds the rate of loss of nitrogen from the gas-filled cavities into the blood.

In the middle-ear cavity, a noncompliant gas-filled cavity, an increase in middle-ear pressure (MEP), instead of volume expansion, is observed on inhalation of N_2O. Changes in MEP cause many complications, such as ear pain, temporary hearing impairment, rupture of the tympanic membrane, graft displacement after tympanoplasty, stapes displacement, window rupture [2–8], and postoperative emesis [9,10].

In contrast, we expected a minimal effect of total intravenous anesthesia (TIVA), on MEP because no inhalational anesthetic is used in this method. In this study we determined changes in MEP during TIVA with propofol, fentanyl, and ketamine (PFK).

Materials and methods

Twelve patients (six men and six women, mean age 55 ± 5 [SD] years, range 40–77) undergoing elective surgical or gynecological operations were studied. We obtained informed consent from the patients. The patients had an ear examination by an otolaryngologist the day before operation to confirm that their tympanic membranes and tympanograms were normal. A normal tympanogram (tympanogram type A) was defined as one that gave a characteristic sharply defined peak by an impedance audiometer, with MEP between 0 and 100 daPa (102 mmH_2O).

MEP was measured with an RS-31 Impedance Audiometer (Rion, Tokyo, Japan). The theoretical basis of impedance audiometry has been described by Blackstock et al. [11]. The impedance audiometer functions by forming an airtight seal in the external auditory

Address correspondence to: T. Kubota
Received for publication on May 9, 1997; accepted on October 6, 1997

canal. A pure tone is emitted while the pressure in the external canal is varied stepwise from −300 to +200 daPa (−306 to +204 mmH_2O), and measurements of reflected sound are made at each pressure. When the pressures in the middle ear and the external auditory canal are equal, the tympanic membrane is free to vibrate. At this point of minimal impedance, the pressure is considered to be the MEP as long as the tympanic membrane is intact. Hence, the limit of the measurement of MEP by the impedance audiometer ranges from −300 to −200 daPa.

All patients inhaled 40% oxygen at a total flow rate of 4 l·min^{-1} via a mask for 3 min before induction of anesthesia. Anesthesia was induced with divided doses of fentanyl 100 μg, propofol 1 mg·kg^{-1}, and ketamine 0.5 mg·kg^{-1}, and maintained with a continuous infusion of propofol 4–6 mg·kg^{-1}·h^{-1} and ketamine 1 mg·kg^{-1}·h^{-1}, and intermittent bolus administration of fentanyl. Tracheal intubation was facilitated with intravenous vecuronium 0.1 mg·kg^{-1}. The lung was ventilated with 40% oxygen to maintain end-tidal carbon dioxide ($ETCO_2$) at 35–45 mmHg. Muscle relaxation was obtained with vecuronium.

MEP was assessed just before the induction of anesthesia and at 10-min intervals until 60 min after induction. The inhalation of 60% N_2O in oxygen was then started. MEP was measured at 2-min intervals. When MEP exceeded 200 daPa, the upper limit of the impedance audiometer, the measurement was completed.

Data analysis

The data were analyzed by repeated-measures analysis of variance followed by Fisher's partial least-squares difference test. *P*-values less than 0.05 were considered significant. All values were expressed as mean ± SEM.

Results

All patients had normal tympanograms and tympanic membranes. MEP gradually increased, and the increase reached a significant level 50 min after the induction of PFK (Table 1 and Fig. 1). After inhalation of 60% N_2O in oxygen, MEP rapidly increased and exceeded 200 daPa, the upper limit of the audiometer, within 36 min in all patients (Fig. 1). The values of MEP even at 2 and 4 min after the start of N_2O administration (60 ± 16 and 87 ± 19 daPa, respectively) were significantly higher than before N_2O administration (38 ± 12 daPa). The mean rate of increase of MEP was 0.4 daPa·min^{-1} during PFK, whereas it was 12.3 daPa·min^{-1} after the start of N_2O administration. Because MEP in three patients was already above 200 daPa, the data after 6 min of N_2O administration were not statistically analyzed.

Table 1. Changes in middle-ear pressure during total intravenous anesthesia with propofol, fentanyl, and ketamine

Time	Middle-ear pressure (daPa)
Pre	16 ± 8
Ane10	23 ± 7
Ane20	16 ± 7
Ane30	22 ± 11
Ane40	23 ± 12
Ane50	34* ± 12
Ane60	38* ± 12

Pre, Before induction of PFK; Ane, minutes after induction of PFK.
* $P < 0.05$ *vs* Pre. All data are mean ± SEM.

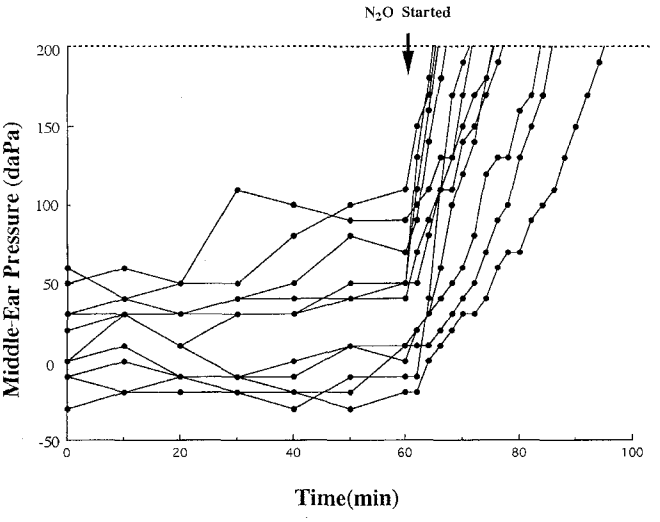

Fig. 1. Individual changes in middle-ear pressure (daPa) during total intravenous anesthesia with propofol, fentanyl, and ketamine and during inhalation of N_2O

Discussion

Armstrong and colleague [12] demonstrated the detailed relationships between the positive pressure of the middle ear cavity and symptoms of the ear. Briefly, positive pressures in the middle ear from 40.8 to 68 mmH_2O are consciously perceived by most people as a feeling of fullness in the middle ear. At about 136–204 mmH_2O, the feeling of fullness is distinct and annoying, and hearing impairment occurs. Pressures between 204 and 408 mmH_2O usually cause increased discomfort and tinnitus. In some individuals, there may be actual pain and vertigo of a mild nature. Above 408 mmH_2O MEP, there is unbearable pain, tinnitus, and vertigo. Thomsen et al. [13] reported that MEP increased by 340 mmH_2O 32 min after inhalation of 80%

Table 2. Diffusion rates of anesthetics relative to that of nitrogen

Gas	Molecular weight	Blood/gas partition coefficient	Diffusion rate relative to that of nitrogen
Nitrogen	28	0.016	1.0
Oxygen	32	0.03	1.7
Nitrous oxide	44	0.47	23.5
Halothane	197	2.3	54.2
Enflurane	185	1.8	43.8
Isoflurane	185	1.4	34.0
Desflurane	168	0.42	10.7
Sevoflurane	200	0.69	16.1

Relative diffusion rates (RDR) of the anesthetic agents (AAs) with respect to nitrogen (N_2) are calculated by the following formula: RDR = D_{AAs}/D_{N_2} = ($\lambda_{AAs}/\lambda_{N_2}$) × (square root of [$MW_{N_2}/MW_{AAs}$]). λ, Blood/gas partition coefficient; MW, molecular weight (see ref. 15).

N_2O, 220 mmH_2O 38 min after inhalation of 60% N_2O, and 290 mmH_2O 66 min after inhalation of 40% N_2O. Perreaults et al. [3] also showed that MEP reached 400 mmH_2O 30 min after inhalation of 66%–70% N_2O and 0.5%–1.0% halothane. At these levels of MEP, the patient (if conscious) would complain of discomfort and tinnitus.

In normal subjects, passive venting of the middle ear by opening the Eustachian tube to relieve the pressure would occur at pressures of 150–300 mmH_2O [13]. However, the pressure may not be relieved when there is Eustachian tube dysfunction due to inflammation, infection, or scar contracture. Venuti and colleagues [14] reported that patients with mild upper-airway or middle-ear disease were likely to have impaired tubal function, and passive opening of the Eustachian tube was impaired during inhalation of N_2O in 47% of such patients. Therefore, inhalation of N_2O should be avoided in patients with otitis media or upper-airway inflammation. In this study, although all patients had normal tympanograms and tympanic membranes, passive venting did not occur at pressures less than 200 daPa. Similarly, Davis et al. [15] reported that passive venting occurred at 225–420 mmH_2O during inhalation of N_2O. Moreover, there is a report of rupture of the tympanic membrane during anesthesia with N_2O in a patient without middle-ear disease [6]. These findings suggest that inhalation of N_2O may not be desirable for any patients.

If anesthetics whose solubility in blood exceeds that of nitrogen were administered at high concentrations, they would be expected to cause increased MEP [1]. Davis et al. [15] reported that under identical conditions, the rates of diffusion (D) of the anesthetic agents (AAs) are proportional to the blood/gas partition coefficient (λ) and inversely proportional to the square root of its molecular weight (MW). The relative diffusion rates (RDR) of anesthetic agents for nitrogen (N_2) shown in Table 2 are calculated by the following formula: RDR = D_{AAs}/D_{N_2} = ($\lambda_{AAs}/\lambda_{N_2}$) × (the square root of [MW_{N_2}/MW_{AAs}]) [15]. In spite of its high relative diffusion (23.5), N_2O is usually administered at high concentrations. Since volatile anesthetics have higher diffusion rates than nitrogen (some of them higher than N_2O), the relative rates of transfer into the middle-ear cavity would be quite rapid. However, the increases in MEP with these agents are minimal, as these agents are usually administered at low concentrations.

The increase in MEP after N_2O administration might be due not only to N_2O but also to PFK, as MEP significantly increased during PFK without N_2O. However, the rate of increase of MEP after inhalation of N_2O was 31-fold higher than it was before inhalation of N_2O. Moreover, the rate of increase after the start of N_2O inhalation that we obtained (12.3 daPa·min^{-1}) is similar to that reported by Davis et al. [15] (13 $mmH_2O \cdot min^{-1}$). Therefore, PFK may make little contribution to the increase in MEP after the start of N_2O inhalation.

In the present study, MEP slightly but significantly increased during PFK. However, its maximum level (38 ± 12 daPa 60 min after the start of PFK) was lower than the previously reported level 55 min after the inhalation of 1%–2% halothane in oxygen (97 daPa) [15]. Although the cause of this increase in MEP during PFK is still unclear, it may be due to inhalation of 40% oxygen, whose diffusion rate is 1.7-fold that of nitrogen. Moreover, dysfunction of the Eustachian tube due to the lack of swallowing and the supine position during general anesthesia may also be involved [16]. In contrast, Karabiyiki et al. [10] reported that MEP significantly decreased until 20 min after induction of TIVA with propofol and alfentanil. However, their patients were managed with a lower $ETCO_2$ of 4.5 ± 0.5 kPa (33.8 ± 3.8 mmHg) and a lower F_IO_2 of 0.33, as compared with our patients ($ETCO_2$, 35–45 mmHg; F_IO_2, 0.4). Changes in $PaCO_2$ would significantly affect MEP, as Shinkawa et al. [17] demonstrated that raising the $PaCO_2$ level by hypoventilation increased MEP. Therefore, the difference in $ETCO_2$ and F_IO_2 may cause the discrepancy with the MEP data.

In conclusion, we found that PFK had a minimal effect on MEP, whereas the addition of N_2O to PFK increased MEP dramatically. Therefore, TIVA, at least PFK, could be a better choice for patients with middle-ear or upper-airway diseases, in order to avoid ear complications. Moreover, it is better to avoid the use of N_2O even for patients without middle-ear or upper-airway diseases.

References

1. Munson ES (1974) Transfer of nitrous oxide into body air cavities. Br J Anaesth 46:202–209
2. Owens WD, Gustave F, Sclaroff A (1978) Tympanic membrane rupture with nitrous oxide anesthesia. Anesth Analg 57:283–286
3. Perreault L, Normandin N, Plamondon L (1982) Tympanic membrane rupture after anesthesia with nitrous oxide. Anesthesiology 57:325–326
4. Man A, Segal S, Ezra S (1980) Ear injury caused by elevated intratympanic pressure during general anaesthesia. Acta Anaesthesiol Scand 24:224–226
5. Srivastava S (1980) Tympanic membrane rupture during nitrous oxide anaesthesia. Br J Anaesth 52:961
6. White PF (1983) Spontaneous rupture of tympanic membrane occurring in the absence of middle ear disease. Anesthesiology 59:368–369
7. Waun JE, Sweitzer RS, Hamilton WK (1967) Effect of nitrous oxide on middle ear mechanics and hearing acuity. Anesthesiology 28:846–850
8. Patterson ME, Bartlett PC (1976) Hearing impairment caused by intratympanic pressure changes during general anesthesia. Laryngoscope 86:399–404
9. Palazzo MGA, Strunin L (1984) Anaesthesia and emesis. I: Etiology. Can Anaesth Soc J 31:178–187
10. Karabiyik L, Bozkirli F, Çelebi H, Göksu N (1996) Effect of nitrous oxide on middle ear pressure: a comparison between inhalational anaesthesia with nitrous oxide and TIVA. Eur J Anaesthesiol 13:27–32
11. Blackstock D, Gettes MA (1986) Negative pressure in the middle ear in children after nitrous oxide anaesthesia. Can Anaesth Soc J 33:32–35
12. Armstrong HG, Heim JW (1937) The effect of flight on the middle ear. JAMA 109:417–421
13. Thomsen KA, Terkildsen K, Arnfred I (1965) Middle ear pressure variations during anesthesia. Arch Otolaryngol 82:609–611
14. Venuti FS, Curatolo M, Chillé G, Bellinghieri F, Galletti C, Galletti B, Messina G (1991) Pressure changes in the middle ear during nitrous oxide anesthesia. Minerva Anesthesiol 57:57–62
15. Davis I, Moore JRM, Lahiri SK (1979) Nitrous oxide and the middle ear. Anaesthesia 34:147–151
16. Shinkawa H, Okitsu T, Kaneko Y (1987) Positive intratympanic pressure in the morning and its etiology (in Japanese with English abstract). Nihhon Jibiinkouka Gakkai Kaihou (J Otolaryngol Jpn) 90:695–699
17. Shinkawa H, Okitsu T, Yusa T, Yamamuro M, Kaneko Y (1987) Positive intratympanic pressure in the morning and its etiology. Acta Otolaryngol (Stockh) 435:107–111