The Journal of Laryngology and Otology
October 1982. Vol. **96.** pp. 883–892

Middle-ear pressure variations during general anaesthesia

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Introduction

IT is well known that significant transient changes in middle ear pressure occur during general anaesthesia and it has been shown that the primary reason for this is the use of nitrous oxide as an anaesthetic agent. Thomsen *et al* (1965) first documented the pressure changes in 1965 when administering nitrous oxide to normal volunteers. They showed that in any individual the rate of middle ear pressure change increased with increasing concentrations of nitrous oxide. Matz *et al* (1967) showed a significant correlation between pressure rise and the concentration of nitrous oxide in the middle ear.

The changes in pressure are due to the relative rates at which nitrous oxide, nitrogen and oxygen exchange between the gas in the middle ear and the gases dissolved in the blood perfusing the mucosa of the middle-ear cleft. The rates of exchange are a function of the blood/gas partition coefficients, the partial pressure difference between the blood and the gas and, to a small extent, the molar masses of the gases. For similar conditions nitrous oxide will exchange thirty times faster than nitrogen because its blood/gas partition coefficient is thirty times greater (Steward *et al.*, 1973). On induction of anaesthesia, nitrous oxide will diffuse into the middle ear in greater quantity than nitrogen will leave, producing an increase in pressure. On emergence from anaesthesia, the reverse occurs; nitrous oxide will leave the middle ear in greater quantity than nitrogen will enter, producing below ambient or 'negative' pressure.

During middle ear surgery, some otologists request that the anaesthetist discontinue the nitrous oxide prior to grafting a tympanic membrane or replacing a tympanomeatal flap. It is still far from clear what is happening to the middle ear pressure at this time, or in the immediate post-operative period. This study was designed to quantify the pressure variations in an intact middle-ear cleft during and immediately after general anaesthesia, and to relate these changes to the pattern of post-operative pressure changes in an operated ear.

https://doi.org/10.1017/S0022215100093245 Published online by Cambridge University Press

Materials and methods

This study comprised twenty-three adult E.N.T. patients who were scheduled for surgery under general anaesthesia. The patients were 'randomly' selected in that no particular regard was paid to the operation which was undertaken. Middle ear pressure was recorded before, and at intervals during and after, the procedure. Anaesthesia was induced in eighteen patients by thiopentone and suxamethonium and maintained with a gas mixture of 32 per cent oxygen, 66 per cent nitrous oxide and 2 per cent halothane delivered via an endotracheal tube. Ten patients were breathing spontaneously, eight were ventilated. In five patients induced in the same manner as above, anaesthesia was maintained with oxygen and halothane only. All five of these were ventilated. The duration of anaesthesia varied from 15 to 200 minutes.

Middle ear pressure was measured by impedance audiometry (Sheehy and Hughes, 1974), using a modified Madsen Zo 70 electroacoustic impedance bridge. A U-tube manometer and pressure pump were incorporated into the instrument, increasing the range of measurement to ± 600 mm. H_2O. The range of most clinical impedance bridges is $+200$ to -400 mm. H_2O, which is inadequate to measure the pressure changes encountered in the course of this study. Middle-ear pressure was assessed by varying the pressure in the external auditory meatus by means of the manually operated pump, and by noticing the pressure at which a reverse deflection of the compliance needle occurred. This deflection was checked by both increasing and decreasing the pressure in the external auditory meatus.

Results

There is a definite pattern of pressure variation in all patients receiving nitrous oxide. Fig. 1 shows a typical recording. A gradual rise in pressure after administration of nitrous oxide is followed by a sudden decrease to a lower but still positive pressure. The cycle continued for as long as nitrous oxide was administered. On cessation of the nitrous oxide, the middle ear pressure still continued to rise in most cases for some minutes (Range 0–40 minutes with a mean of 12.9 mins. and s.d. of ± 11.9 mins.), after which the pressure gradually started to fall. Post-operative sub-atmospheric pressures developed in 17 out of the 18 patients who received nitrous oxide, with 10 developing pressures below -200 mm. H_2O.

Certain parameters were chosen to describe the pattern of pressure changes.

I.M.E.P. Initial middle-ear pressure before nitrous oxide administration (mm. H_2O)

(dp/dt)I —Rate of initial pressure increase following intubation (mm. H_2O/min.)

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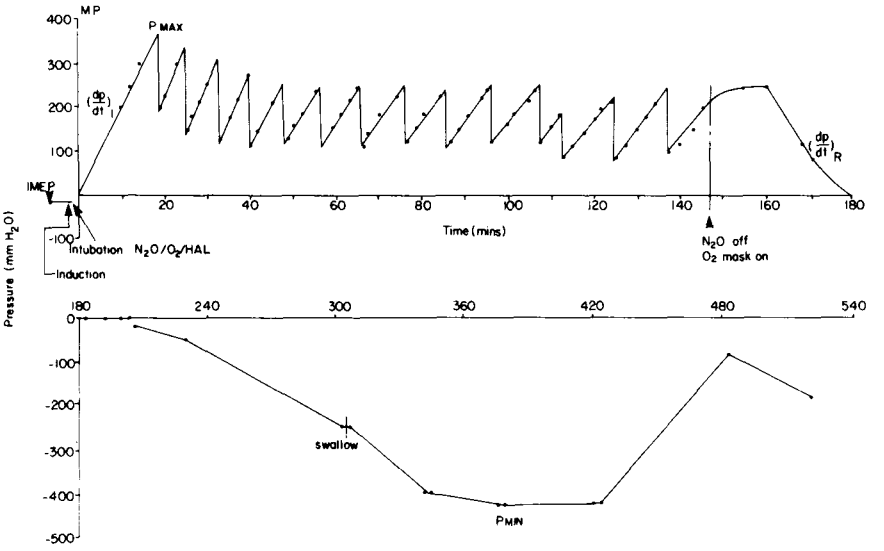


FIG. 1

Middle ear pressure variation during nitrous oxide anaesthesia.

PMAX —Maximum pressure attained (mm. H_2O)

(dp/dt)R —Rate of pressure decrease after cessation of nitrous oxide (mm. H_2O/min.)

PMIN —Minimum pressure attained (mm. H_2O)

These are illustrated in Fig. 1, and the results for all 18 patients are shown in Figs. 2a and 2d.

The parameter (dp/dt)I was determined for each individual by taking the average rate of pressure increase during the initial pressure rise after intubation. PMAX refers to the maximum pressure attained during the initial pressure rise. The rate of pressure decrease (dp/dt)R was determined by calculating the average pressure drop over the first 30 minutes of fall in pressure after cessation of nitrous oxide.

Post-operative swallowing and coughing rarely resulted in ventilation of the middle ear. Development of pressures below −200 mm. H_2O occurred for several hours in over 50 per cent of cases.

The five patients who received only oxygen and halothane again followed a set pattern of pressure change, as shown in Fig. 3. However, the pressures developed were not of the same magnitude as with nitrous oxide, and the cyclical rise and fall was not seen. A slight negative pressure was seen post-operatively in four out of the five cases, ranging from 0 to −200 mm. H_2O with a mean of −90 mm. H_2O.

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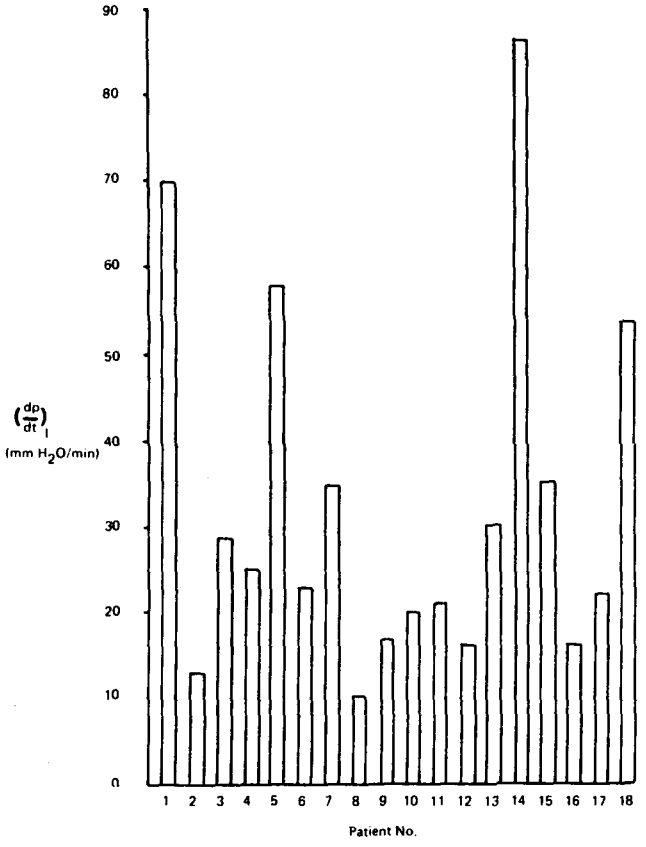


FIG. 2a

Rate of initial pressure increase in middle ear during nitrous oxide anaesthesia.

Discussion

A comparison of the results of this investigation with those of other workers is shown in Table I. The percentage of nitrous oxide used was approximately the same in all five studies. There seems to be a large degree of variation between the studies and the small number of patients in three of the four other studies no doubt contributes to this. With regard to the minimum pressure developed, Patterson and Bartlett (1976) appear to have studied this in only one case. Singh and Kirk (1979) took just one measurement twenty-four hours post-operatively in each of their twenty patients.

The cyclical pattern of pressure change seen with nitrous oxide anaesthesia has been discussed previously by Rasmussen (1967), Patterson and Bartlett (1976) and more recently by Davis *et al* (1979). They concluded that it was due to passive opening of the eustachian tube.

From the results of Thomsen *et al* (1969) we know that in any one individual the rate of pressure increase during inhalation anaesthesia is

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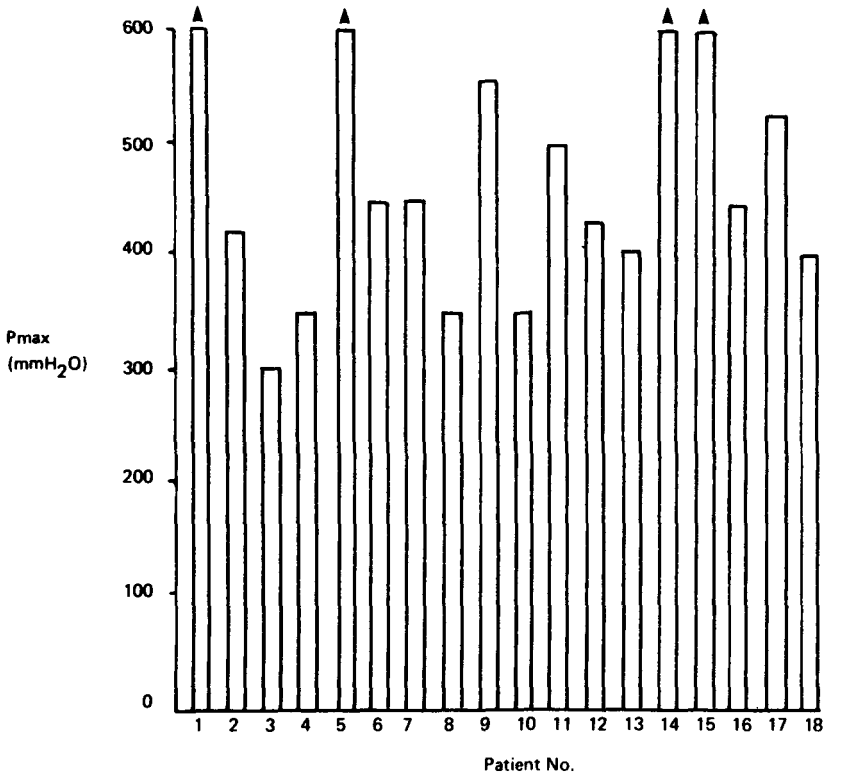


FIG. 2b

Maximum pressure developed in middle ear during nitrous oxide anaesthesia.

related to the concentration of nitrous oxide in the inspired gas. However, from the results of this study it is evident that the same concentration of nitrous oxide creates different rates of pressure increase in different individuals. This large inter-subject variation needs to be explained. We suspect that it is due to variations in perfusion of the middle-ear mucosa and the volume-to-area ratio of the tympanomastoid air spaces. It is interesting that the four highest values of (dp/dt)I occurred in patients who had radiologically sclerotic mastoids of the ear in which the pressure changes were recorded.

The large values for the rate of pressure increase reported by Singh and Kirk (1979) may possibly be explained by their patient sample, which was exclusively children, as compared to the other studies where the samples were exclusively adult. In a separate study we have examined this parameter in a group of fourteen children undergoing surgery under general anaesthesia with nitrous oxide and have found significantly greater rates of pressure increase than in our group of adults.

The smaller middle ear pressure changes found when using halothane

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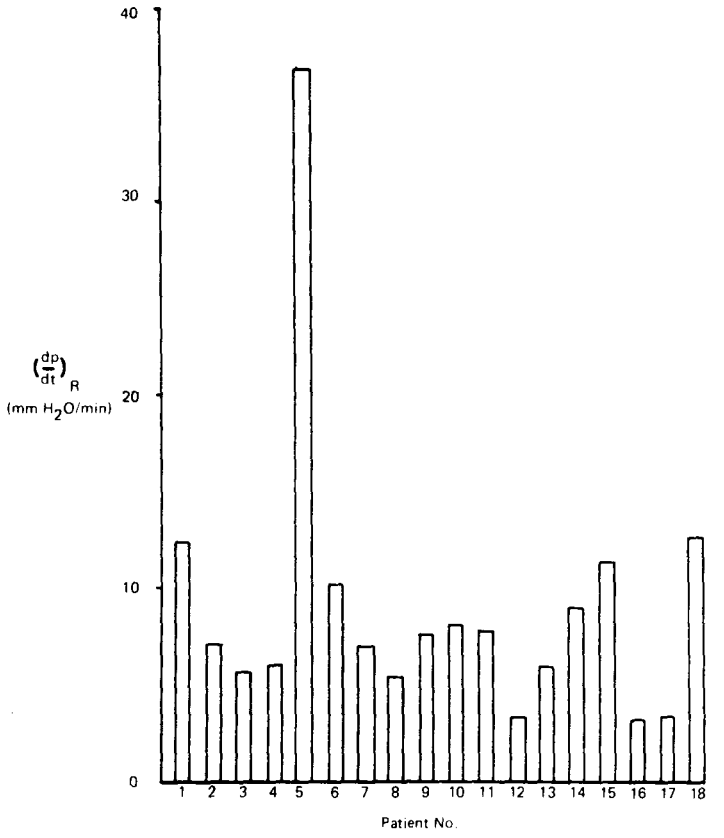


FIG. 2c

Rate of pressure decrease in middle ear after cessation of nitrous oxide.

in oxygen only can, we contend, be explained by the following:

(a) Oxygen has a significantly lower diffusion rate than nitrous oxide.
(b) The difference in the diffusion rates of oxygen and nitrogen is insignificant in comparison to that between nitrous oxide and nitrogen, with the result that nitrogen diffuses from the middle-ear cleft at approximately the same rate as the oxygen diffuses in.

The absence of the rise and fall cycle when using oxygen can be explained by the failure of middle-ear pressure to reach that required to passively open the eustachian tube.

During an operative procedure with an open middle-ear cleft, the middle-ear pressure will obviously be atmospheric, with nitrous oxide diffusing into the cleft and mixing with the ambient air. On closing the cleft by grafting the tympanic membrane or replacing the tympanomeatal flap the subsequent pressure changes will be dictated by the timing of cessation

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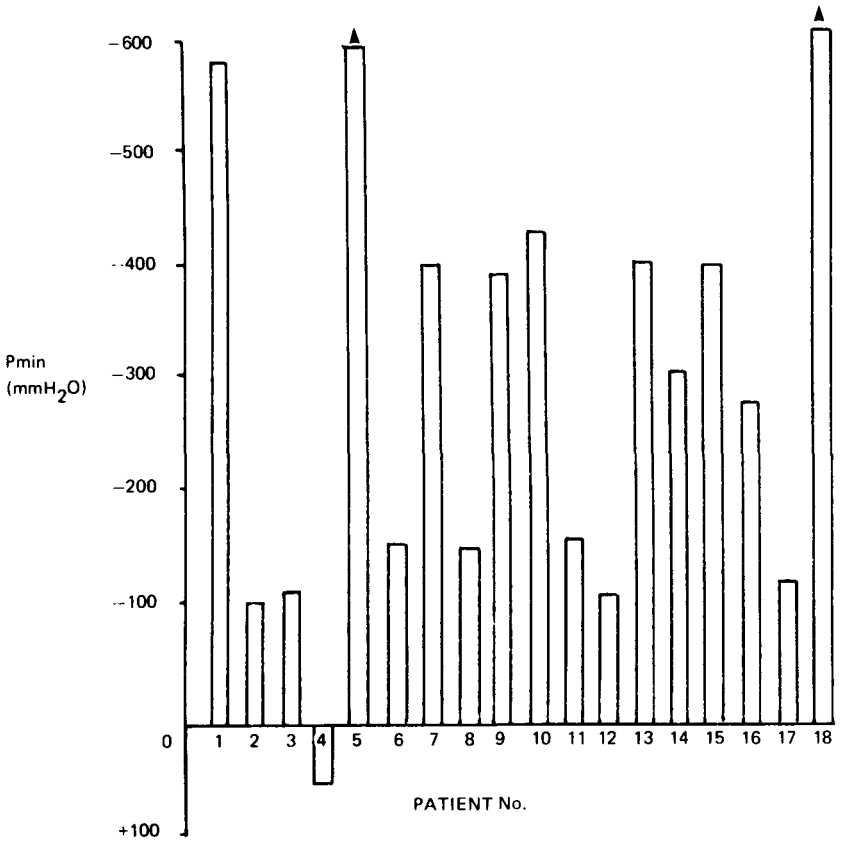


FIG. 2d

Minimum pressure developed in middle ear after nitrous oxide anaesthesia.

of nitrous oxide and the kinetics of nitrous oxide excretion.

This latter point has been studied by Frumin *et al* (1961). They monitored the end tidal nitrous oxide concentrations in eight anaesthetized subjects and constructed a decay curve from the average data. Extrapolation from this curve reveals that it takes 15 minutes from cessation of nitrous oxide for the percentage of nitrous oxide in the end tidal air to fall to 10 per cent of the concentration during anaesthesia.

If the administration of nitrous oxide is not stopped as the cleft is closed, the pressure within the middle-ear cleft will start to rise rapidly, probably sufficiently to cause passive venting of the eustachian tube. If this occurs a significant negative pressure is likely to ensue post-operatively. If cessation of nitrous oxide coincides with closure of the cleft, significant pressure variations may still occur and it seems logical to cease the administration of nitrous oxide at least 15 minutes before closing the middle ear cleft.

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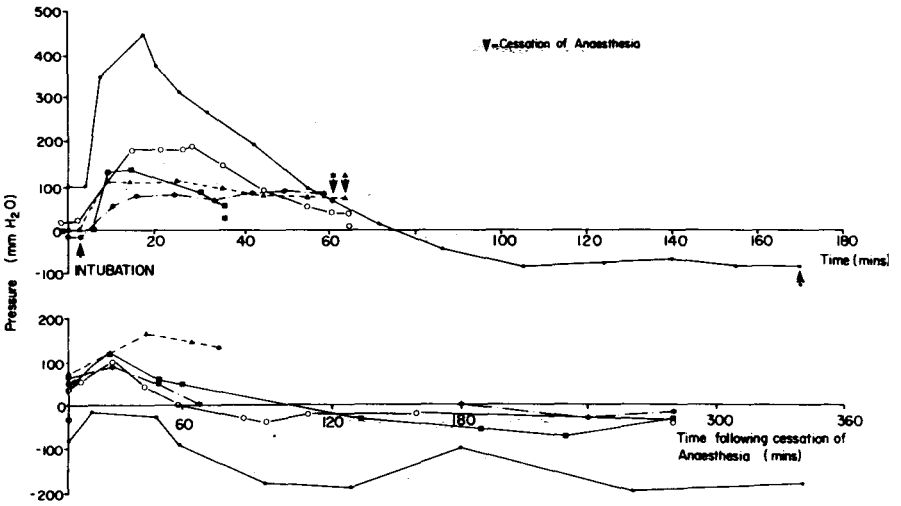


FIG. 3

Middle ear pressure variation during inhalation of oxygen and halothane only.

Obviously from Frumin's study there is a wide inter-subject variation in the excretion rate of nitrous oxide.

To quantify the post-operative pressure changes more accurately in the operated ear, more work of a theoretical nature is necessary.

The surgical implications of a significant middle-ear negative pressure in the immediate post-operative period are considerable and we surmise that it may well be capable of:-

	No. of subjects	$(\frac{dp}{dt})_I$ mm H_2O/min	Pmax mm H_2O	$(\frac{dp}{dt})_R$ mm H_2O/min	Pmin mm H_2O
Thomsen, Terkildsen and Arnfred	2	6	> 340	4	not stated
Patterson and Bartlett	6	15	275 - 450	6 - 10	−285 (only one case followed into neg phase)
Singh and Kirk	20	30 - 120	210 - 500	not measured	−30 - −400
Davis, Moore and and Lahiri	5	9.5	230 - 420	1	not stated
Richards, O'Neill and Wilson	18	10.0 - 86.0 mean 32.2 s.d. 21.1	300 ->600* 461 98	3.3 - 37.0 9.2 7.5	+50 ->−600* −289 195

* All values greater than 600 were considered as being equal to 600 when calculating mean and standard deviation.

TABLE 1

Comparison of results from nitrous oxide anaesthesia studies.

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(i) Detachment of an inlay graft from the inner surface of the tympanic membrane.
(ii) Dislocation of a middle-ear prosthesis.
(iii) Forcing a stapes replacement prosthesis into the vestibule.
(iv) Frustration of early ventilation of the middle ear following tympano-mastoid surgery, promoting the formation of a middle-ear effusion.

Conclusions

Our work confirms the findings of other workers concerning the general pattern of middle ear pressure changes during nitrous oxide anaesthesia, namely: that there is a cyclical rise and fall in pressure which is sustained for as long as nitrous oxide is administered.

We have clearly demonstrated that, when halothane is administered in oxygen only, the pressure changes which are developed in the middle ear are substantially smaller than when nitrous oxide is used, and also the cyclical pattern does not occur.

The negative pressure that develops after cessation of nitrous oxide anaesthesia in a closed middle ear cleft is generally accepted as being due to withdrawal of nitrous oxide from the middle ear without compensatory replenishment by nitrogen from the blood and the lack of ventilation *via* the Eustachian tube. Our findings indicate that this negative pressure is more consistently present and is of greater magnitude and duration than previously suspected. We were surprised to find that it is only rarely relieved by swallowing.

Such a negative pressure post-operatively in an operated ear is obviously undesirable. To reduce the risk of this occurring it is advised that the administration of nitrous oxide be ceased at least 15 minutes before the middle ear cleft is closed by grafting a tympanic membrane defect or replacing a tympanomeatal flap.

Although this measure is likely to prevent a significant negative middle ear pressure post-operatively, we advocate that nitrous oxide is an undesirable anaesthetic agent for otological surgery. Clearly these problems would be completely avoided if all otological procedures were performed under local analgesia. When this is not practical it should be possible to find a more favourable combination of gases. From our research we find it difficult to conceive of any mixture which would have any advantages over a volatile anaesthetic in air.

Acknowledgements

We would like to thank Dr. D. Picken and Professor W. W. Mapleson, of the Department of Anaesthetics, Welsh National School of Medicine, for their technical assistance; and also Mrs. Diana Prichard, for typing the manuscript.

https://doi.org/10.1017/S0022215100093245 Published online by Cambridge University Press

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