EFFECT OF INTRAOPERATIVE INSPIRED GAS MIXTURES ON POSTOPERATIVE NOCTURNAL OXYGEN SATURATION

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SUMMARY
Continuous postoperative nocturnal pulse oximetry was performed in 20 patients undergoing elective cholecystectomy to examine if the composition of anaesthetic gas mixtures affects postoperative gas exchange. The patients were allocated randomly to receive either nitrogen or nitrous oxide during anaesthesia, and oximetry was performed on the night before operation and the first and third nights after operation. Considerable oxygen desaturation was seen in both groups. During the first night after operation the proportion of the night during which oxygen saturation was less than 85% was greater in the nitrogen group than in the nitrous oxide group, but there was no significant difference between the mean overnight saturation values of the two groups. (Br. J. Anaesth. 1993; 71: 476-480)

KEY WORDS

It has been known for many years that upper abdominal surgery is associated with an impairment of gas exchange in the lung and that this persists for several days after operation [1-3]. Recent studies using pulse oximetry have shown that many patients have some nocturnal arterial oxygen desaturation for up to 5 nights after operation, and that this is often associated with episodes of obstructive or central apnoeas [4].

A major cause of arterial desaturation is believed to be the development of atelectatic areas in dependent zones of the lung. These develop shortly after induction of anaesthesia and persist into the postoperative period [5, 6]. It is believed that the lung changes are associated with the reduction in functional residual capacity (FRC) which occurs on induction of anaesthesia and persists for several days after operation. However, it has also been suggested that anaesthesia with a gas such as nitrous oxide, which has a relatively great blood solubility, may increase the development of atelectasis in alveoli with critically low ventilation:perfusion ratios [7]. This theory is supported by the observation that substitution of nitrogen for nitrous oxide during anaesthesia and thoracotomy for repair of hiatus hernia decreased the magnitude of collapse in the dependent lung as assessed by postoperative radiographs [8].

Because overnight pulse oximetry recording provides more comprehensive data than repeated blood-gas analysis after operation, we decided to use this technique to compare the effects of intraoperative administration of nitrogen or nitrous oxide on the development of nocturnal hypoxaemia after a standardized upper abdominal operation.

PATIENTS AND METHODS
The study was approved by the Hospital Ethics Committee. Twenty-three patients scheduled to undergo routine cholecystectomy via a standard transverse right subcostal incision gave informed consent to inclusion in the study and were allocated, according to a randomized order drawn up before the start of the study, to receive either nitrous oxide or nitrogen as a component of their intraoperative inspired gas mixture.

In accordance with Ethics Committee requirements, the patients were free to leave the study at any stage. Of the 23 from whom baseline measurements of oxygen saturation were recorded on the night before operation, 20 (ASA I or II) were studied again on the first night after operation and 11 on the third night (table I).

On the night before operation, the patients were permitted to receive night sedation, if required, in the form of temazepam 10 mg; this was taken by four

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<th>TABLE I. Patient characteristics (No. or mean (SE) [range])</th>
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<td>Nitrous oxide group</td>
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<td>No. of patients (M/F)</td>
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patients. All patients received temazepam 20-30 mg 1 h before operation. One patient was also given pethidine 100 mg i.m. for gall bladder pain.

Anaesthesia was induced with thiopentone 4-5 mg kg\(^{-1}\), fentanyl 1.5-2.5 \(\mu\)g kg\(^{-1}\) and vecuronium 0.1 mg kg\(^{-1}\). All patients underwent manual ventilation of the lungs before tracheal intubation was performed, the nitrogen group receiving 2 % enflurane in oxygen and the nitrous oxide group 2 % enflurane with 68 % nitrous oxide in oxygen. Subsequently, all patients underwent mechanical ventilation of the lungs by a Nuffield 400 ventilator and circle absorber system delivering tidal volumes of 10 ml kg\(^{-1}\); frequency was adjusted to produce an end-tidal carbon dioxide concentration of 4.5 %. All patients received 30 % oxygen and 1-2 % enflurane, depending on depth of anaesthesia and cardiovascular response, plus either nitrous oxide or nitrogen (nitrogen delivered in the form of medical air; oxygen concentration measured with a calibrated fuel cell analyser).

Use of i.v. fluids was standardized as far as possible. One litre of Hartmann's solution was given over the first 1 h and an additional 1 litre over the next 4 h. This was followed by glucose—saline 1 litre over each of the subsequent 8- and 12-h periods. Four patients (three in the nitrogen group) also received Dextran 70 500 ml in normal saline as part of one surgeon's antithrombotic regimen.

All patients received papaveretum 15-20 mg and prochlorperazine 6.25 mg i.m., 5-10 min before the end of the operation. They were then prescribed the same analgesia as required 4-hourly. The last four patients studied (two in each of the study groups) also received 0.5 % bupivacaine 10-20 ml injected into the wound edges during skin closure.

Data collection and analysis

Continuous nocturnal oxygen saturation recordings were made before operation and on the first and third nights after operation, using an Ohmeda 3740 pulse oximeter which measured arterial oxygen saturation (\(S_{\text{pO}_2}\)) via a finger probe secured to the patient's finger with an adhesive dressing. Data were stored and analysed using a specially designed computer program running on a Toshiba 1600 computer.

A computer analysis program [9] performed second-by-second sampling of \(S_{\text{pO}_2}\) and heart rate and calculated average saturation for the entire duration of the recording, mean values and SD of \(S_{\text{pO}_2}\) during each 10- or 30-min epoch and percentage of recording time during which \(S_{\text{pO}_2}\) was less than 90, 85 or 80 %.

Whenever the finger probe was detached from the finger, to allow patient mobility, to facilitate nursing intervention or accidentally, zero data points were recorded and automatically rejected from further analysis by the computer program. An average of about 7 % of the potentially available data were excluded in this way.

The mean total duration of overnight recording was 9.8 h, representing 35280 potential items of data. Individual saturation recordings were examined to ensure that those collected were satis-

factory. Successive 10-min continuous recordings revealed an overall pattern of reasonably stable oxygen saturation with intermittent, brief periods of desaturation and recovery.

Our computer program did not record signal amplitude. We were therefore unable to measure signal artefact as described recently elsewhere [10]. A small amount of movement artefact was seen in all patients but, given the overall duration of the recordings and number of observations obtained, we assumed this would have had little effect on the mean overnight oxygen saturation or cumulative analysis of overnight oxygen saturation. A visual analysis of the number of periods of transient oxygen desaturation (decreases in oxygen saturation of 4 % or more with prompt recovery of at least 3 %) was made for each overnight recording obtained.

RESULTS

Individual mean overnight oxygen saturations are shown in figure 1. Mean \(S_{\text{pO}_2}\) in each patient decreased by 0.9-14.3 % on the first night after operation. The greatest individual overnight mean \(S_{\text{pO}_2}\) on the first night after operation was 95.4 % and the least 78.5 %.

Mean overnight oxygen saturation was greater in the nitrous oxide group than in the nitrogen group (ns: Student's \(t\) test, \(P = 0.05\)) (table II). The
The mechanisms and time course of postoperative hypoxaemia have been reviewed extensively [4]. It is believed that physiological changes occurring as a consequence of anaesthesia may persist after operation and be modified by postoperative events. This study was designed to examine if the nature of the anaesthetic gas mixture might have any effect on postoperative respiratory function.

It is well known that functional residual capacity (FRC) decreases immediately after the induction of anaesthesia [11]. This is thought to be the result of a reduction in the resting tone of the inspiratory muscles of the rib cage and diaphragm, which normally oppose the intrinsic elastic recoil of the lung tissue. At the level of the alveolus, such a reduction in FRC should produce a reduction in the size of individual alveolar units. On a theoretical basis, this may predispose to alveolar collapse [7]. The likelihood of such collapse occurring should be increased by the presence of gases with high blood/gas solubility coefficients, such as nitrous oxide, leading to “absorption atelectasis”, and should be decreased by less soluble gases, such as nitrogen.

This effect was demonstrated clinically in a study which examined the effect of substituting nitrogen for nitrous oxide in the anaesthetic gas mixture given to a group of patients undergoing thoracotomy for hiatus hernia [8]. The investigators found a 51% incidence of radiologically-evident postoperative atelectasis in patients who had received nitrous oxide during this procedure, compared with a 24% incidence in a similar group of patients who were given nitrogen. However, the nitrous oxide patients...
were studied retrospectively and the nitrogen patients prospectively, so that the two groups may have been affected by other variables. Another study examined the effects of nitrogen and nitrous oxide on venous admixture during anaesthesia in a group of patients undergoing minor surgery [12] for which neither anaesthetic technique nor surgical procedure was standardized. Venous admixture was calculated from measurements obtained during spontaneous or controlled ventilation with 30% oxygen in nitrogen and a fixed difference of oxygen content between arterial and mixed venous blood was assumed. The calculated shunts were compared with those calculated for a group of patients who received oxygen in nitrous oxide and no significant difference in venous admixture during anaesthesia was observed between the two groups.

Both the above studies may be criticized on the grounds that they were not planned as randomized controlled trials, therefore we studied patients subjected to a standardized anaesthetic and operation, the only variable being the administration of nitrogen or nitrous oxide during the operation. Although the number of patients we studied was relatively small, our data failed to confirm the suggestion that ventilation of the lungs with nitrogen decreases the incidence of atelectasis resulting from anaesthesia and surgery. This leads us to conclude that critical ventilation:perfusion collapse and absorption atelectasis are not of major importance during anaesthesia for this type of operation, or that the effects of these mechanisms may be hidden as a result of other factors.

Apart from the sometimes severe desaturation which we observed in patients on the first night after operation, the most surprising aspect of our study was the fact that patients in the nitrogen group tended to have more desaturation than those in the nitrous oxide group—a finding opposite to that expected on theoretical grounds. One possible explanation is that the patients in the nitrogen group seemed to suffer more pain immediately after operation and tended to require more postoperative analgesia than those in the nitrous oxide group. It is recognized that postoperative pain and analgesia affect ventilatory function [13] and it is possible that the increased postoperative pain inhibited deep breathing and re-expansion of collapsed alveoli, or that the increased requirement for opioids produced central respiratory depression. However, there was no statistical correlation between the amount of opioid administered and the degree of oxygen desaturation.

Whereas absorption atelectasis depends, theoretically, on the gaseous content of critically unstable alveoli, a second possible mechanism of producing intraoperative atelectasis might be by a reduction in the forces tending to hold alveoli open, allowing "compression collapse". We have mentioned already that reduction in thoracic cage tone allows intrinsic elastic recoil of the lungs to produce a reduction in lung volume. Other extrapulmonary forces also may influence this process. General anaesthesia has been seen to be associated with a cranial shift in the resting position of the diaphragm [14]. This appears to result from a shift of blood from the thorax and extremities to the abdomen. In addition, the reduction in the resting tone of the diaphragm during anaesthesia allows the contents of the abdominal cavity to exert a pressure gradient on the adjacent lung tissue [5], producing the "compression atelectasis" in dependent regions of the lung which has been demonstrated by computed tomography of the thorax during anaesthesia [15, 16]. These effects would appear to be a consequence of the state of anaesthesia itself and have been shown to be independent of the mode of induction of anaesthesia and the nature of ventilation of the lungs (spontaneous or controlled) [5] although, with controlled ventilation, the changes might be exaggerated by increased intrathoracic pressure.

Another factor which might have an effect on postoperative respiratory function is the nature of the surgery undergone by patients studied. Surgical manipulation of the abdominal contents can have a profound effect on the function of the diaphragm. In certain circumstances, and particularly after gall bladder surgery [17], diaphragmatic dysfunction may occur for 7–10 days after operation [18, 19]. The overriding cause of this dysfunction would seem to be a reduction in phrenic nerve output, since diaphragmatic contractility can be shown to be normal on direct stimulation of the phrenic nerve [20]. It would appear that inhibition of the phrenic nerve occurs as a consequence of the stimulation of unmyelinated splanchnic nerves [21]. The gall bladder and other regions of the gastrointestinal tract are supplied with receptors responding to compression, distension and movement [17]. Mechanical stimulation of the gall bladder may thus produce a selective inhibition of the diaphragm which may continue into the postoperative period. The resultant reduction in diaphragmatic tone could then contribute to the persistence of intraoperative atelectasis.

We conclude that the substitution of nitrogen for nitrous oxide during general anaesthesia for gall bladder surgery does not influence the development of nocturnal arterial oxygen desaturation during the postoperative period.

REFERENCES


