Detection of inspiratory resistive loads after anaesthesia for minor surgery

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Summary
We measured the ability to detect inspiratory resistances in 22 patients undergoing minor gynaecological surgery, using linear resistances of 1.2–24.4 cm H₂O litre⁻¹ s, and ranked 1–9. The rank that could be detected was measured before surgery, approximately 10 min after recovery from anaesthesia, and then 25 min later. After anaesthesia there was a significant increase \( (P<0.001) \) in the rank number by 2, which returned to preoperative values 25 min later. Minute ventilation was reduced by 25% after operation and this may partly explain this change; however, we believe the transient decrease in ability to sense resistance indicates residual effects of anaesthetics and may explain in part the incidence of hypoxia caused by airway obstruction in patients after anaesthesia. (Br. J. Anaesth. 1997; 78: 308–310).

Key words

Episodes of hypoxaemia are common immediately after operation and are also frequent later, in association with treatment with opioids, where airway obstruction appears to be the cause.¹ Anaesthetic agents and opioids are known to reduce responses to hypoxia and hypercapnia but they may perhaps also reduce responses to other effects of airway obstruction. Such responses may be protective. For example, some patients with severe asthma have a reduced capacity to detect inspiratory resistance and have reduced responses to hypoxia. Such patients are more likely to suffer severe consequences in association with attacks of asthma, such as loss of consciousness or mechanical ventilation.² To assess the possibility that other responses to airway obstruction could be affected after anaesthesia, we tested the ability of patients to detect a threshold inspiratory resistive load before, immediately after and later after minor surgery with general anaesthesia.

Methods and results
After obtaining local Ethics Committee approval and informed written consent, we recruited women undergoing minor gynaecological surgery as day-case patients. We noted any current medication, smoking history, a history of airway disease or exposure to pollutants, and also noted if the patient appeared unduly anxious.

We used a series of resistances constructed using different numbers of glass capillary tubes (6 cm long, of the type used for measuring erythrocyte sedimentation rate) mounted in parallel in a supporting tube. They were linear within 5% for flow rates of up to 30 litre min⁻¹ and had resistances of 1.2, 1.7, 2.5, 2.8, 3.8, 8.3, 10.3, 11.5 and 24.4 cm H₂O litre⁻¹ s. Calibration was performed with a laboratory manometer system (for the purposes of statistical analysis, however, they were numbered 1–9 and considered as ranked values on an ordinal rather than an interval scale). Each resistance could be fitted in turn to a respiratory system.

The patient lay recumbent and wore a noseclip, and breathed from a mouthpiece connected to a unidirectional low resistance anaesthetic valve (Ambu Hesse). Valve resistance was 2 cm H₂O litre⁻¹ s at an inspiratory flow rate of 3 litre s⁻¹. The inspiratory side of the valve was connected to a large bore three-way tap by means of a wide bore tube, 70 cm in length, so that a resistance could be added for a single inspiratory effort, unknown to the subject. She was asked to indicate by hand movement if she felt a change in the effort of breathing. The resistances were added each in turn until the patient reliably indicated it had been felt. The patient also breathed from the mouthpiece while minute volume and frequency were measured over 1 min with an electrical Wright respirometer (Ferrari) connected to the expiratory limb of the circuit. This device had been calibrated recently by the manufacturer.

The anaesthetic induction agent was restricted to propofol, but anaesthesia and surgery were not otherwise standardized. During and after surgery analgesics were given as judged necessary and all agents used were noted, including a plot of end-tidal concentration of the volatile agent vs time.

The minimum resistance detected, and minute volume and ventilatory frequency, were measured again immediately after recovery from anaesthesia, soon after the patient could co-operate with the test,
and also after the patient had fully recovered and had returned to the ward. If the patient could not detect a resistance after recovery from anaesthesia, this response was scored as 10.

Data were analysed with the sign test and sign confidence limits to compare changes within patients with time, the Mann–Whitney test to compare subgroups, and descriptive plots using Minitab release 8 run in MS-DOS 6.2.

We recruited 25 patients but three did not complete the study. Of the remaining 22, one patient did not have measurements made in the ward. Mean age was 30 yr, height 160 (SD 7) cm and weight 62 (14) kg. Four patients reported mild asthma but none was currently receiving treatment. Nine were smokers and two were exposed to dust. Before anaesthesia minute volume was 8.7 (7.4–9.6) litre min\(^{-1}\) and ventilatory frequency was 17 (13–19) bpm (median, quartiles). After recovery from anaesthesia the corresponding values were 6.3 (4.9–7.7) litre min\(^{-1}\) and 15 (13–17) bpm, and on testing in the ward these values were 8.3 (6.4–9.2) litre min\(^{-1}\) and 15 (14–17) bpm. Changes in ventilation were statistically significant (\(P<0.001\) and 0.05, respectively), but changes in frequency were not.

The propofol dose used was 200 (165–300) mg, and duration of anaesthesia and surgery was 17.5 (10–21.5) min. The responses to loads were measured 11 (10–13) min after the end of anaesthesia. The tests in the ward were performed at 36 (30–42) min after the end of anaesthesia. Surgical procedures ranged from examination under anaesthesia to laparoscopic procedures. The anaesthetic techniques included intubation of the trachea, administration of neuromuscular blocking agents and morphine analgesia. However, the maximum duration of anaesthesia was only 25 min. Three patients received desflurane and the remainder enflurane, all in nitrous oxide, and the expired volatile agent concentrations were almost equipotent with no large variations in the concentrations administered. Seven patients received either fentanyl or alfentanil in small amounts and seven received morphine during operation.

The resistances detected are shown in figure 1. In each circumstance, patients were consistently able to identify a single load. Before operation the rank of resistance was median 2.5 (95% confidence intervals 2–4) and this increased to 5 (4–7) after operation and decreased to 3 (2–4) after return to the ward. The increase in resistance for detection after operation was significant (\(P<0.001\)) and there was no difference between the rank values before operation and after return to the ward.

There was no relationship between changes in minute volume and ventilatory frequency in individual patients, and the changes in detection of the load. The loads detected by patients who had received morphine in the intraoperative period (seven patients, median dose 15 (8–15) mg) were only greater by one rank, and 95% confidence limits for change in rank were almost identical. Two patients received topical anaesthesia to the trachea before intubation using lignocaine. They did not show excessive changes in load detection, the load detected increasing by two rank values in each case. No other factors associated with anaesthesia and surgery appeared to affect the resistance threshold. In particular, duration of surgery and time between i.v. induction agent and testing were not related to change in the rank of resistance detected.

**Comment**

Appreciation of resistive loads is altered by changes in inspiratory flow rate.\(^3\) After operation, mean inspiratory flow rate is likely to have been reduced by approximately 25% as expired minute volume was reduced by a median of 2.1 litre min\(^{-1}\) from 8.6 litre min\(^{-1}\), with no significant change in ventilatory frequency. This may have reduced the sensation of the resistance slightly but it is unlikely that this factor alone was responsible for the increase in load rank of 2.5. In addition, ventilation increased by only 1.1 litre min\(^{-1}\) in the ward 30 min later and at this time load detection had returned to values found before operation. The resistive loads we used were not great: the greatest load in the series causes moderate dyspnoea in normal subjects (on a standardized linear verbal rank scale). Detection of a resistive load seems to be proportional to the proportional increase in resistance it generates (the Weber law)\(^3\) and thus comparison of loads between subjects is of less value than changes in perception within subjects.

We measured the conscious appreciation of the load by the patient. This response may not reflect the response of the respiratory muscles to increased inspiratory resistance: there may continue to be an increase in muscle activation or force generation in response to the load, but conscious appreciation of the presence of the load is impaired.

The anaesthetic and surgical procedures that we studied were not standardized other than in the use of a single, relatively short-acting induction agent. In many respects the patients were heterogeneous, in

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**Figure 1** Rank load number detected by each patient. Values are for before anaesthesia, immediately afterwards and after return to the ward. Patients who received morphine during surgery are shown separately and within each category each patient is represented by a different symbol.
terms of anxiety, postoperative pain and management (use of different opioids with various durations of action, and airway management by face mask, laryngeal mask or tracheal intubation). Further study of more uniform groups may allow factors responsible for the changes to be more established clearly, but this study did show a clear effect in a common clinical setting. The time course of the resolution of the effect suggests that the cause was residual anaesthetic agents. If this were the induction agent, the effect would be greater in patients who had short procedures: if it were the volatile agent then the effect would be more marked after the longer procedures. No such effects were evident.

Factors that protect patients from hypoventilation and resultant hypoxia, apart from vigilance of their attendants and oxygen therapy, are not well understood. The awake subject breathes with more force when inspiration is impeded but the influence of residual anaesthetic agents or opioids on detection and response to this response is not clear. Despite experimental evidence for responses to airway pressure, mediated by sensory information from the airways, these responses seem to be inactive in sleeping humans. In sleeping subjects, airway obstruction causes increases in tongue muscle activity only after a complete respiratory cycle of obstruction. Then, airway muscle activity increases in parallel with activity in other respiratory muscles such as the diaphragm. In fact, detection of airway resistance appears to be mediated by changes in intrapulmonary pressure.

We conclude that after short periods of anaesthesia with relatively short-acting agents such as propofol, enflurane, and isoflurane, patients responded less well to minor transient airway obstruction, even when awake. The use of moderate amounts of opioids (morphine) did not appear to affect impairment, although we did not formally investigate this factor and patients who received morphine may have been systematically different from those who did not.

References