Effects of surgical skin incision on respiration in patients anaesthetized with enflurane†

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Summary

We measured ventilation in 12 subjects anaesthetized with enflurane (end-tidal concentration 1.25–1.45 %) and nitrous oxide to assess the effect of surgical stimulation on ventilation in humans. Tidal volume and respiratory timing were measured by pneumotachograph before and just after a standardized surgical skin incision. Surgical stimulation increased ventilation by increasing tidal volume, which increased progressively over the first five breaths after incision. The first breath after the stimulus was prolonged, but the timing of the subsequent breaths returned rapidly to the duration observed before incision. Ventilation increased from median 3.6 (quartiles 2.9, 4.3) to 5.4 (3.8, 7.0) litre in⁻¹ (P < 0.01). The increased tidal volume was not associated consistently with shortening of inspiratory duration. (Br. J. Anaesth. 1996; 76: 777–779)

Key words


Traditional teaching is that depth of anaesthesia can be assessed by a variety of clinical signs [1]. Anaesthesia affects tidal volume and frequency of ventilation, and these variables are commonly used in clinical practice to assess depth of anaesthesia, although ventilatory frequency is influenced more by the volatile anaesthetic agent used than by depth of anaesthesia [2].

Spontaneous ventilation during surgery is influenced by both anaesthetic and stimulus. During anaesthesia with halothane or isoflurane, surgical skin incision is followed by an increase in the depth and rate of breathing [3, 4]. Stimuli in experimental animals cause similar increases in ventilation [5]. An increase in tidal volume, associated with a decrease in the duration of the respiratory cycle, suggests that respiration could be controlled in a manner proposed by Clark and von Euler [6]. The aim of this study was to demonstrate more exactly the pattern of response of respiration in anaesthetized patients after surgical skin incision, and to use this response to investigate the relationship between tidal volume and timing of the respiratory cycle.

Patients and methods

After obtaining local Ethics Committee approval and written informed consent, we studied 12 patients (eight female), ASA I or II, undergoing elective surgery for varicose veins. Patients were aged 30–63 yr and weighed 45–85 kg. No premedication was given. With the patient supine, anaesthesia was induced with propofol 2.5 mg kg⁻¹ i.v., a laryngeal mask airway was inserted and anaesthesia maintained with enflurane and 66 % nitrous oxide in oxygen. End-tidal enflurane concentrations were measured by sidestream sampling from a connector attached to the laryngeal mask airway using a Hewlett-Packard M1025 gas analyser, calibrated according to the manufacturer’s instructions. The inspired enflurane concentration was adjusted to obtain a stable end-tidal value, within 0.1 % for each patient, at 1.25–1.45 %. Patients breathed spontaneously via a screen pneumotachograph (Mercury F100L) from a circle anaesthesia system. The pneumotachograph was calibrated on each occasion with a standard air syringe. The pneumotachograph output was digitized using a laptop computer fitted with an analogue-to-digital converter and recorded with Cardas software (version 6.1) (Oxcams, Oxford), which allowed instantaneous display of flow against time.

After surgical skin preparation and draping (skin clips were not used), each patient was allowed to breathe undisturbed for at least 2 min before a skin incision was made over the femoral vein in the groin for exploration of the saphenofemoral junction. The time of the start of incision was recorded onto the computer flow display. No attempt was made to synchronize the incision with the respiratory cycle.

The flow signal was later replayed and integrated using custom-written software to obtain a breath-by-breath volume signal, which was analysed to measure inspiratory duration (Tᵢ), expiratory duration (Tₑ) and tidal volume (Vᵢ). Instantaneous ventilation was calculated from tidal volume and respiratory cycle duration, and mean inspiratory flow rate was calculated as Vᵢ/τᵢ.

Comparisons were made of these variables within individual subjects by paired comparison using the median values obtained from 10 breaths before

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incision for each patient, with the median of 10 breaths after incision in the same patient (Wilcoxon paired test). P < 0.05 was considered significant. In addition, the first breath after incision was compared with the median value obtained from 10 breaths before incision, to assess the immediate responses. Individual \( V_T \) and \( T_I \) values in each subject before and after incision were plotted to assess the changes in breathing pattern. Statistical analysis was with Minitab release 8.2 using MS DOS version 6.2. Results are reported as median (quartile) values.

**Results**

Immediately before incision, mean end-tidal enflurane concentration was 1.39 % (sd 0.06 %). No patient showed limb movement on skin incision, but skin incision had an immediate effect on breathing pattern (fig. 1).

**TIDAL VOLUME**

Before stimulation, median tidal volume was stable with no significant trend in magnitude. Tidal volume of the first breath after stimulation had a greater volume (297, 178–437 ml) than the median of the values of the 10 preceding breaths (195, 151–211 ml). There was then a progressive increase in tidal volume over the next five breaths. Median tidal volumes for the 10 breaths after incision were significantly greater than the tidal volumes for the preceding 10 breaths (195, 151–211 ml; \( P < 0.01 \)).

**MEAN INSPIRATORY FLOW RATE (\( V_T/T_I \))**

Surgical stimulation was associated with a small immediate increase in \( V_T/T_I \) in the first breath, and a progressive further increase over the next five breaths. The marked increase in tidal volume at the first breath was clearly not the result of an increase in inspiratory flow. The 10 breaths after stimulation had a significantly greater \( V_T/T_I \) than the preceding 10 breaths (10.6, 8.6–15.3 litre min\(^{-1}\) compared with 7.4, 6.9–9.0 litre min\(^{-1}\); \( P < 0.01 \)).

**RESPIRATORY TIMING**

The first breath after stimulation had a longer \( T_I \) (1.99, 1.55–2.42 s) and \( T_E \) (1.95, 1.63–2.28 s) than the preceding 10 breaths (1.49, 1.35–1.70 s and 1.60, 1.40–1.85 s, respectively) (\( P < 0.05 \)). The timing of the subsequent breaths reverted to that before stimulation.

Minute volume had a pattern similar to mean inspiratory flow rate, and increased progressively for the first five breaths after stimulation. The 10 breaths after stimulation had a significantly greater minute volume (4.9, 3.8–7.0 litre min\(^{-1}\) compared with 3.6, 2.9–4.3 litre min\(^{-1}\) before stimulation; \( P < 0.01 \)).

**RELATIONSHIP BETWEEN \( V_T \) AND \( T_I \)**

There was no clear relationship between \( V_T \) and \( T_I \). Stimulation was associated with an increase in \( V_T \) in 10 subjects, of whom six showed a reduction in \( V_T \) and four an increase. Inspiratory duration decreased significantly in only two patients.

**Discussion**

Volatile anaesthetics in general, and enflurane in particular, are respiratory depressants [7]. Surgical stimulation can antagonize this depression, but little work has been done to assess the features of the increase in ventilation [3, 4]. Difficulties with standardizing the degree of stimulation have hampered studies of the effects of surgical stimulation on respiration. The intensity of the stimulus depends on the site of surgery and the degree of trauma. We attempted to standardize the stimulus by using a single skin incision in one body site. We also restricted our observations to breaths shortly after stimulation, so that secondary effects caused by changes in ventilation would have only small effects. The laryngeal mask may have caused some stimulation but our experience is that it is well tolerated at even very light levels of anaesthesia, in many instances better than the manipulations associated with conventional mask anaesthesia.

We found that stimulation had an immediate effect on the timing of respiration, followed by reversion to the timing before the stimulus. The speed of this response resembled the rapid responses in respiratory timing to stimuli mediated by nervous afferent activity, such as limb nerve stimulation [8], intercostal nerve stimulation [9], vagal stimuli [10] and peripheral chemoreceptor stimulation [11]. However, it is remarkable how respiratory timing reverts to the previous pattern after one breath. The remainder of the response to surgical stimulation was similar to that seen with a stimulus such as hypercapnia, which has a great influence on tidal volume, and less on respiratory timing [12].

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**Figure 1** Time course of mean inspiratory flow rate (●), tidal volume (□), and duration of inspiration (○) and expiration (●), showing the median (interquartile range) for each breath in 12 subjects, for 10 breaths before and after the start of skin incision.
Our findings do not support the possibility that lung volume affects inspiratory duration in these circumstances [2, 6].

Surgical stimulus caused a progressive increase in mean inspiratory flow rate over the first five breaths after the stimulus. Noxious stimuli result in extensive activation of brainstem reticular neurones [13], which can cause an increase in respiratory output, particularly in the rate of increase of inspiratory activity [8]. However, increased reticular system activity causes increases in both tidal volume and frequency [14], whereas in this study only drive increased. This resembles the response to a chemoreceptor stimulus [12].

Studies of other volatile anaesthetic agents with different effects on ventilatory frequency, such as halothane, would be of interest. It might also be that other forms of stimulation, in particular those carried from the viscera by the vagus, may result in different patterns of response, because vagal input in the nucleus tractus solitarius is related closely to respiratory reflex control.

In summary, we found that surgical stimulation during enflurane anaesthesia caused a slow increase in central respiratory drive and minute volume, but only a transient change in respiratory timing. Depth of respiration, or mean inspiratory flow rate, is therefore a useful index of respiratory response to surgical stimulation during anaesthesia with enflurane, but ventilatory frequency not.

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References