Effects of airway occlusion on breathing muscle electromyogram signals, during isoflurane anaesthesia, with and without the effects of fentanyl and hypercapnia

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Editor’s key points

- Airway occlusion pressure may be a useful measure of expiratory muscle activation during anaesthesia.
- This small study investigated the effects of airway occlusion on muscle activity itself.
- Airway occlusion had no effect on electrical activity of the diaphragm or external oblique muscles.
- This implies that this technique can be used to measure respiratory function during anaesthesia.

Background. Expiratory muscle action is prominent during anaesthesia and can impair lung function. This activity is exaggerated by the use of opioids. Airway pressure during occlusion of expiration would be a valuable measure in the study of expiratory muscle activation. However, this would only be valid if the imposed occlusion did not itself alter muscle activation. This possibility can be checked by directly assessing muscle activity by electromyography; varying arterial carbon dioxide tensions and opioid action should be considered.

Methods. We studied seven spontaneously breathing patients, anaesthetized with nitrous oxide and isoflurane, in four conditions: during an infusion of fentanyl and after naloxone, breathing normally and with breathing stimulated with CO₂. We compared diaphragm and external oblique abdominal electromyogram (EMG) signals during normal and occluded breaths. We also measured chest wall volume and compared airway occlusion pressure, during inspiration and expiration, with the EMG results.

Results. Inspiratory occlusion increased the duration of inspiration during hypercapnia by 20%, but not the rate of electrical activation of the diaphragm, indicating that occlusion does not cause a reflex increase in diaphragm contraction. In contrast, expiratory occlusion did not affect either the duration of expiration or the electrical activity of the external oblique muscles.

Conclusions. In these conditions, except for a change in inspiratory duration, respiratory muscle activity is unaffected by airway occlusion. Airway occlusion will permit valid measures of muscle activity in inspiration and expiration and provide simple measurements of respiratory muscle function during anaesthesia.

Keywords: anaesthetics, inhalation; analgesics, opioid; electromyography; respiratory muscles; respiratory physiological processes

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Measurement of airway occlusion pressure was developed to assess respiratory drive. If airway pressure is measured during occlusion of the airway of a spontaneously breathing subject, then the pressure developed indicates the neural activation of the respiratory muscles.¹ The first part of the inspiratory attempt occurs before the subject becomes aware of the impediment to inspiration. The force developed early in the inspiratory attempt is taken as an index of central drive, because isometric muscle contraction generates a pressure proportion to neural activation. During anaesthesia, the method is particularly useful for two reasons: first, the entire process of pressure development can be followed from the start of the breath, because the subject is unaware of the occlusion, and second, if ventilation is also measured, then the factors that generate force, indicated by occlusion pressure, and those that affect translation into flow and volume change, indicated by ventilation, can be distinguished.² However, the method is only valid if the manoeuvre itself has no effect on neural output to the respiratory muscles. For
example, stretch receptors could be activated when muscle shortening is prevented, causing reflex effects on muscle activity. The electrical activity of the muscle [electromyogram (EMG)] is the most appropriate signal to assess possible reflex events. Reflexes can also affect the actions of expiratory muscles, but these have been much less studied in anaesthetized man.

We intend to investigate the effects of expiratory muscles because they are frequently active during anaesthesia. Occlusion pressure measurements could provide a practical means to distinguish the forces generated when the expiratory muscles relax from the force generated when the inspiratory muscles contract. To be a valid measure, however, occlusion should not have reflex effects on muscle activity. We tested the proposition that occlusion would not affect the pattern of EMG activity, either in magnitude or in timing, in anaesthetized subjects. Chemical drive (hypercapnia) and pharmacological agents (opioids) affect the pattern and timing of muscle activation, particularly of the abdominal muscles. We therefore measured EMG signals while manipulating these factors to allow conclusions which could be applied elsewhere.

**Methods**

The study was approved by the Biomedical Research Committee of the Henri Mondor Hospital. After informed consent, we recruited patients about to undergo superficial or peripheral surgical procedures, excluding those with obesity or clinical evidence of cardiac or respiratory disease. Anaesthesia was maintained with nitrous oxide, isoflurane, and i.v. supplements of fentanyl, up to 750 μg. Neuromuscular blockers (NMBs) were used if necessary during the surgical procedure. When surgery was complete, residual NMB activity was antagonized and a full reversal ensured by monitoring neuromuscular transmission by ulnar nerve stimulation with supramaximal train-of-four stimuli. The patients breathed spontaneously from a breathing system with a low resistance one-way valve (Fig. 1). An infusion of fentanyl was started to replace the intermittent dosing used during anaesthesia and surgery, and adjusted to maintain a stable respiratory rate, close to the value present when spontaneous respiration commenced. Patients breathed 70% nitrous oxide in oxygen. Inspired isoflurane (Drager Vapor 19.3) was adjusted to maintain an intended end-tidal isoflurane concentration between 0.65% and 0.7%.

Respiratory gas flow was measured with a pneumotachograph (Mercury FC10, Mercury Instruments, Glasgow, UK) and a differential transducer (Furness FC044, Bexhill-on-Sea, UK) calibrated with flows of 70% nitrous oxide in oxygen. Pressure at the airway opening was measured with a Validyne DP 45 transducer (Northridge, CA, USA). Large-bore taps were used to intermittently occlude single episodes of inspiration or expiration (Fig. 1). Gas was sampled from the centre portion of the valve for continuous analysis of CO₂ (Normocap 200) and isoflurane concentration (Normac, both Datex Instrumentation, Finland). Gas sampling was discontinued during occlusion manoeuvres, so that the volume of the isolated system remained constant. The inspired and expired gas tubing was connected via a wide-bore sidearm so that when the fresh gas supply to the inspiratory tubing was reduced, some rebreathing of exhaled gases could occur, to allow economy in fresh gas use during CO₂ administration. The breathing system was repeatedly checked to ensure that there were no leaks.

We used percutaneous fine-wire electrodes (150 μm diameter) (Xomed, Jacksonville, FL, USA) placed 1 cm apart, in the diaphragm and in the external oblique muscle to measure the EMG. For the diaphragm, insertion was via either the sixth or the seventh intercostal space in the anterior axillary line on the right side, and for the external oblique muscle, in the left upper quadrant of the abdomen. Fine needles were used for insertion, which were then withdrawn, leaving the wire in place in the muscle. During insertion, we aided placement of the needles by stimulating the motor nerves to the target muscles. We used unipolar needle electrodes to stimulate the phrenic nerve in the neck and the ninth intercostal nerve in the intercostal space in the mid-axillary line to stimulate the external oblique. Satisfactory electrode positioning was confirmed by repeated nerve stimulation after the electrodes had been placed. After needles had been placed in the diaphragm, we used 2% lidocaine to block three appropriate intercostal nerves in the mid-axillary line on that side, to reduce contamination of the diaphragm EMG signal by EMG activity from the overlying intercostal muscle. EMG activity was amplified (Nihon Koden AM6016), filtered (low-frequency time constant 0.03 s, high-frequency cut-off 3 kHz), and integrated with a time constant of 100 ms (Nihon Koden E1 6010, Nihon Koden, Cachan, France). The amplifier had an input impedance of 5 MΩ, a common mode rejection ratio of >60 dB from DC to 65 Hz, and a peak to peak interval noise of 6 μV. A calibration signal of 1 mV was available. By noting the amplifier settings and recording, the response to a standard square-wave input, the integrated EMG signal could be expressed in standard units of μV s. Both the raw EMG and integrated EMG signals were recorded on paper (Gould TA 2000, Gould SA, Longjumeau cedex, France), and the integrated EMG, pressure, and flow signals were recorded with a Dash IV recorder/logger system (Dash Instruments, AstroMed House, Slough, UK).

Chest wall movement was measured using an optical measurement system. A laser light is formed into a wide but narrow beam that appears as a stripe when it falls on the measured surface. With a precisely controlled mirror, the beam is rapidly moved to five different known positions on the surface. The shape of these lines of light depends upon the contour of the body surface and is detected by a video camera located in an accurately known position. The contour of the each line, relative to a reference position, is calculated and each stripe of the video image is assembled to form sections of the object. From the area of the sections and the distance between each section, absolute chest wall
volume is computed and the changes in volume during respiration can be calculated. These measures of respired volume are unaffected by drift, which is a weakness of systems that use flow integration.

Measurements were made when respiration was stable. Inspiratory and expiratory phases were occluded alternately. Each episode of occlusion was separated by a randomly selected number of breath cycles, but not less than six breathing cycles apart. During each occlusion, the relevant valve was closed during the preceding phase of the breathing cycle for a single phase of respiration, to allow flow, EMG activity, and airway pressure to be measured for the unoccluded breath before and then during the respiratory phase of the attempt at inspiration or expiration (Fig. 2). As soon as the occlusion phase was complete, the occlusion was removed by re-opening the tap. Inspiration and expiration were each occluded at least four times for each condition studied. We studied quiet breathing (the time period called Opiate, Quiet Breathing) and then reduced the fresh gas flow until rebreathing of exhaled gas occurred. CO2 was added to the fresh gas until ventilation was approximately doubled. After stabilization for 10 min, measurements were repeated (Opiate, Stimulated Breathing). CO2 flow was then stopped, fresh gas flow was increased, the infusion of fentanyl discontinued, and naloxone 0.8 mg given i.v. A further dose of naloxone 0.4 mg was given if the measurements were not completed in 20 min. When respiration was stable, the measurements were repeated (Naloxone, Quiet Breathing) and then stimulation with CO2 was repeated, and a final set of measurements were made (Naloxone, Stimulated Breathing). At the end of the procedure, the electrode wires were removed from the muscle and placed together under an ECG electrode on the skin of the patient. The resulting signal was recorded to provide a baseline for the integrated EMG signal. Records were replayed from the logger, sampled at 0.1 Hz, and transferred to computer for further analysis.

**Measurements**

For each breath measurement, we used the preceding breath as a control for timing, flow, and volume. Measurements of breath timing were taken from the integrated EMG signal. We noted the duration of the inspiration for control and occluded breaths using the initial increase and the maximum value as the onset and offset of ‘electrical’ inspiration. The amplitude of the EMG signal was also noted at these times, for both the occluded breaths and the immediately preceding normal breath. We also noted the times of the start and end of inspiration in this breath. Because the expiratory pressure generated in the airway was often not constant during the occlusion of expiration, we measured the mean pressure between 25% and 75% of the expiratory time, and the mean EMG value of the abdominal muscles over the same time. The duration of expiration was not measured from the pressure events; this was measured from EMG signals as the time between the offset of diaphragm activity in the preceding inspiration and the onset of the diaphragm activity for the next inspiration. This ensured that all breath timing was measured with reference to diaphragm electrical activity.

**Statistical analysis**

Differences within the time periods were tested using paired t-tests, and differences between the time periods were generally tested using two-way analysis of variance. The differences between control and occluded breaths for respiratory timing and electrical activity were not normally distributed, so the Wilcoxon rank-sum test was used. For summary purposes, means of values (±SD) from each breath during each of the four time periods were taken for each subject.
Results
We approached 14 patients for this study. Ten gave their consent and were studied, but satisfactory recordings of both muscle groups throughout the study were only obtained in nine. In one subject, in whom we did not obtain satisfactory recordings, a small pneumothorax was detected after the study, which resolved spontaneously. There were no other complications or adverse effects in any of the subjects. In two additional patients, we had to reposition the diaphragm electrodes during the course of the study because of a sudden reduction in signal amplitude, presumably caused by displacement of an electrode tip. These changes occurred during stimulation of breathing with CO₂. Consequently, we did not compare their EMG results between time periods and restricted analysis to comparisons between breaths (normal and occluded) within each time period.

Details of the seven patients for whom we present complete results are shown in Table 1. End-tidal isoflurane concentration was maintained at between 0.5% and 0.75% throughout the measurements. In one subject, the concentration had to be reduced to allow adequate ventilation, and one subject required an increase in concentration during the final period of measurement because of signs of inadequate anaesthesia.

An example of the breathing pattern and muscle activities under the effects of fentanyl, and changes after giving naloxone, are shown for a representative subject in Figure 3. As expected, respiratory frequency and diaphragm activity increased after naloxone. Abdominal muscle activity decreased markedly after naloxone.

Neither naloxone nor CO₂ stimulation affected inspiratory time, but the duration of expiration was markedly reduced by naloxone (P<0.001) (Table 2). CO₂ increased tidal volume (P<0.05) and had a pronounced effect on mean inspiratory flow (VT/TI) (P<0.001). Inspiratory occlusion pressure responses were strongly affected by CO₂ stimulation (P<0.01), but not by naloxone. Expiratory pressures were increased by CO₂ (P<0.01) and reduced by naloxone (P<0.05).

Effects of occlusion
Timing
The timing of the normal and occluded breaths is shown in Figure 4A and B. The duration of inspiration was increased...
Table 1  Details of patients studied (fentanyl dosage is summarized as median and quartile values)

<table>
<thead>
<tr>
<th></th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Height (m)</th>
<th>Weight (kg)</th>
<th>Fentanyl dose during surgery</th>
<th>Fentanyl dose during study (µg)</th>
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<tr>
<td>1</td>
<td>M</td>
<td>35</td>
<td>1.82</td>
<td>119</td>
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<td>275</td>
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<tr>
<td>2</td>
<td>F</td>
<td>46</td>
<td>1.62</td>
<td>57</td>
<td>750</td>
<td>100</td>
</tr>
<tr>
<td>3</td>
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<td>31</td>
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<td>79</td>
<td>625</td>
<td>125</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>44</td>
<td>1.79</td>
<td>82</td>
<td>300</td>
<td>125</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>67</td>
<td>1.63</td>
<td>66</td>
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<tr>
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<td>40</td>
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<td>58</td>
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<td>50</td>
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<tr>
<td>7</td>
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<td>59</td>
<td>1.78</td>
<td>75</td>
<td>250</td>
<td>50</td>
</tr>
<tr>
<td>Mean</td>
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<td>171</td>
<td>77</td>
<td>625</td>
<td>100</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>9</td>
<td>20</td>
<td></td>
<td>325, 660</td>
<td>62, 125</td>
</tr>
</tbody>
</table>

Fig 3  Changes in airway flow and muscle activity after giving naloxone. The first and last parts of the trace are at a faster timescale to show the waveform and the central part shows the trend in values.
Table 2  Experimental conditions. Data presented as mean (SD). Duration of expiration was reduced by naloxone ($P<0.001$). Carbon dioxide increased tidal volume ($P<0.05$) and mean inspiratory flow ($V_t/T_i$) ($P<0.001$). Inspiratory occlusion pressure increased by carbon dioxide ($P<0.01$). Expiratory pressures increased by carbon dioxide and reduced by naloxone ($P<0.01$ and 0.05, respectively).

<table>
<thead>
<tr>
<th>Condition</th>
<th>End-tidal CO₂ (kPa)</th>
<th>End-tidal isoflurane (%)</th>
<th>Duration of inspiration (s)</th>
<th>Duration of expiration (s)</th>
<th>Tidal volume (ml)</th>
<th>Mean inspiratory flow rate (ml s⁻¹)</th>
<th>Occlusion pressure (inspiration) (cm H₂O)</th>
<th>Occlusion pressure (expiration) (cm H₂O)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fentanyl</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No CO₂ added</td>
<td>7.09 (0.93)</td>
<td>0.6 (0.13)</td>
<td>1.08 (0.24)</td>
<td>4.41 (2.32)</td>
<td>324 (111)</td>
<td>256 (64)</td>
<td>-13.9 (4.8)</td>
<td>10.0 (4.8)</td>
</tr>
<tr>
<td>CO₂ added</td>
<td>8.16 (0.69)</td>
<td>0.62 (0.11)</td>
<td>1.06 (0.22)</td>
<td>4.36 (2.32)</td>
<td>534 (217)</td>
<td>400 (102)</td>
<td>-26.2 (11.9)</td>
<td>15.7 (6.5)</td>
</tr>
<tr>
<td><strong>After naloxone</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No CO₂ added</td>
<td>5.8 (0.59)</td>
<td>0.64 (0.13)</td>
<td>0.96 (0.24)</td>
<td>1.90 (0.40)</td>
<td>294 (123)</td>
<td>247 (73)</td>
<td>-12.3 (2.8)</td>
<td>7.3 (3.4)</td>
</tr>
<tr>
<td>CO₂ added</td>
<td>7.87 (0.85)</td>
<td>0.68 (0.09)</td>
<td>0.96 (0.19)</td>
<td>1.80 (0.62)</td>
<td>571 (256)</td>
<td>483 (115)</td>
<td>-24.6 (8.1)</td>
<td>14.1 (5.7)</td>
</tr>
</tbody>
</table>

**Fig 4** Comparison of timing and EMG amplitudes for normal breaths and occluded breaths during the four treatment periods. Each panel shows the same sequence: fentanyl action, without and then with CO₂, followed by naloxone administration, without and then with CO₂. The lower part of each panel shows the absolute values, related to the left-hand axis. The upper part shows the changes associated with occlusion, plotted in relation to the right-hand axis. (a) Comparison of inspiratory duration for normal and occluded breaths. The duration of inspiration was increased by occlusion ($P<0.001$, ANOVA). Post hoc tests showed a significant effect in the period when breathing was stimulated during fentanyl administration ($P<0.05$). (b) Comparison of expiratory duration for normal and occluded breaths. (c) Maximal diaphragm integrated EMG signal for normal and occluded breaths. (d) Mid-expiratory abdomen integrated EMG signal for normal and occluded breaths. There were no significant differences between normal and occluded breaths.

by occlusion ($P<0.001$, ANOVA). Post hoc tests showed a significant effect only when breathing was stimulated during fentanyl administration, changing from 1.06 (0.85, 1.19) to 1.25 (0.87, 1.43) s (Bonferroni’s test, $P<0.05$).

In contrast, the duration of expiration was not affected by occlusion of expiration. Since the range of expiratory duration values before naloxone was considerable, there could be concerns that the statistical power of this conclusion might
be inadequate. However, the inter-quartile range of the changes caused by occlusion was small (0.52 and 0.35 s, with and without stimulation) and the study had 80% power to detect a 20% change in the duration of expiration.

**Electrical activity**

**Diaphragm**
The effects of occlusion on EMG are shown in Figure 4C and D. The amplitudes of the integrated diaphragm EMG at the end of a normal and an occluded breath were not different (two-way ANOVA, $P=0.072$). However, because the duration of inspiration was increased (by subgroup analysis in the time period when patients were receiving fentanyl and CO₂), we corrected integrated EMG amplitude for inspiratory duration. After this correction, occlusion was associated with a significant reduction in amplitude (two-way ANOVA, $P<0.01$). The mean difference for all the groups combined was 7.5% and seemed to be consistent for all the measurement periods. However, *post hoc* Bonferroni’s tests did not identify any time periods when there was a significant difference in activity after occlusion.

**External oblique**
Abdominal activity was much greater in expiration (comparison with inspiration, $P<0.0001$). Abdominal activity in expiration was related to the conditions of anaesthesia. Naloxone reduced activity ($P<0.05$) and CO₂ stimulation increased it ($P<0.01$). There was a weak interaction between CO₂ stimulation and opioid effect (two-way ANOVA, $P=0.054$).

Importantly, there was no effect of occlusion on integrated EMG activity ($P=0.335$). This last finding is an important part of the purpose for the study, so the power of this conclusion was tested. The SD of the differences of integrated EMG activity between control and occlusion is 0.0337 μV s. Assuming that an increase in integrated EMG activity by even 5% might be biologically relevant, the power of the study to detect such a difference, if it were present, was 99%.

**Relationship between EMG and occlusion pressure**
The relationship between integrated EMG and occlusion pressure is shown in Figure 5. During inspiration, there is a very similar relationship between these signals, and a similar increase in both values occurs when stimulation with CO₂ is applied. However, the abdominal muscles show a different pattern. When fentanyl is active, abdominal muscle activity is considerable, and when opioid activity is antagonized, the activity decreases. CO₂ stimulation increases abdominal action when opioid activity has been antagonized, but has no additional effect when fentanyl is active.

**Discussion**
We found that airway occlusion has small effects on the timing, but no influence on the magnitude of activation of two representative respiratory muscles. The only important difference found was a prolongation of inspiration, which was significant when ventilation was stimulated. We found a limited effect on the electrical activation of the diaphragm, and no effect at all on either timing or magnitude of activation of the external oblique. We conclude that airway occlusion will provide a generally valid and useful measure of expiratory muscle activity in anaesthetized subjects. Because occlusion had no effects on the abdominal muscles, occlusion should be particularly useful to investigate abdominal muscle activity during anaesthesia.

The relationship in Figure 3 could be interpreted as showing that opioids affect abdominal muscle action. This requires qualification, since CO₂ may confound this interpretation. Muscle activities before and after naloxone administration were measured at different levels of carbon dioxide. A study of abdominal muscle activity, with and without opioid, would require isocapnic conditions to separate the effects of opioid and CO₂.

**Effects on timing**
The only important modification of the respiratory pattern caused by occlusion was that the duration of inspiration was increased. One study, of adult humans anaesthetized with enflurane, reported that occlusion of inspiration...
pproprinoceptive regulation of fibres to be from muscle spindles. Studies of airway occlusion of the diaphragm found about one-third of the supply of the diaphragm, more recent studies of the afferent nerve supply found that diaphragm afferents could interfere with activation of the respiratory muscles during anaesthesia. There could be two potential interactions, involving either muscle afferents, or signals from the lung. Respiratory muscles are subject to reflex responses to loads, both inhibitory (probably mediated by tendon organs) and excitatory (via spindle afferents). In conscious subjects, brief inspiratory occlusion causes transient inhibition of inspiratory intercostal muscles, not mediated by pulmonary afferents and possibly mediated by different information from the muscles themselves. However, these reflexes are probably abolished by clinical depths of anaesthesia. Activation of the diaphragm is not affected by adding elastic loads to breathing in anaesthetized animals, and in human studies, adding a load to inspiration does not affect the diaphragm activity. A mechano-receptor reflex from muscles could either augment or inhibit the motor output. Intercostal muscles have a rich supply of muscle spindles, and the stretch reflex is considered an important component of their control. For example, parasternal intercostal muscle activity is increased by airway occlusion. Although early physiological and histological studies did not support proprioceptive regulation of the diaphragm, more recent studies of the afferent nerve supply of the diaphragm found about one-third of the fibres to be from muscle spindles. Studies of airway occlusion suggest that diaphragm afferents may have a role in the responses to load. Our findings do not support the presence of possible reflex actions during isoflurane anaesthesia.

**Abdominal muscle effects**

Holding the lung at end-inspiration could increase pulmonary stretch receptor activity, and thus augment the expiratory action of the abdominal muscles, either by a direct increase in motor output or by prolonging expiration (an inspiratory–inhibitory Hering Breuer reflex). Prolonged expiration could then also allow the expiratory pressure to augment. Both of these potential interactions, and the effects of inspiratory occlusion, were tested by comparing EMG signals during normal and occluded breaths. The effects of expiratory occlusion were convincingly negative. Thus, occlusion of expiration can be used to assess the force of contraction of the abdominal muscles, without triggering reflex increases in muscle action.

Some technical aspects of our study require consideration. Measurements of breath timing from EMG can show considerable differences from those made from physical measurements such as flow and pressure. We chose to use EMG measurements exclusively to avoid any systematic variation, for example, between pressure and volume signals, which has been described previously.

We blocked the local intercostal nerves to reduce the activity of the muscles surrounding the site where we sampled diaphragm activity. Accurate timing of the cessation of inspiratory activity is difficult if expiratory activity is present. The intercostal blocks prevented local extraneous muscle activity extremely well, and no activity was present in expiration.

Nitrous oxide (N2O) is known to affect muscle activity. N2O has spinal opioid actions and causes cerebral endogenous opioid release. Skeletal and respiratory muscle function is affected by N2O. Warner and colleagues studied the effects of adding N2O to humans and dogs anaesthetized with halothane. N2O reduced ribcage muscle action and increased phasic expiratory muscle activity, particularly in the dog. In the human volunteers, N2O reduced rib cage motion, and although there was no intercostal muscle activity present before nitrous oxide was given, N2O may interact with opioids in causing muscle rigidity, although this may be a non-specific effect of N2O associated with loss of consciousness.

Abdominal muscle contraction is an almost universal finding in patients breathing spontaneously during and after surgery and can reduce lung volume, with adverse effects on gas exchange. For these reasons, measuring this phenomenon, and investigating how it may be reduced, is an important practical goal. The pressure generated by active expiration needs to be distinguished from pressure generated by the passive elastic forces of the respiratory system, but this can be done if the passive elastic properties of the respiratory system are known or estimated. Our study shows that occlusion of expiration is a valid method for investigations of expiratory activity. We intend to use this finding to allow comparison of the forces generated by inspiratory and expiratory muscles and assess the contribution of expiratory activity to breathing during anaesthesia.

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Mr S. McGowan wrote the computer software for the optical measurement system.

**Conflict of interest**

None declared.
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