Abdominal pressure during laparoscopy: effects of fentanyl

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Background. In patients breathing spontaneously during anaesthesia, expiration is active and opioids enhance this effect. The mechanical consequences are not well characterized.

Methods. We studied 14 patients undergoing laparoscopy for minor gynaecological procedures, anaesthetized with isoflurane in nitrous oxide, and breathing spontaneously through a laryngeal mask airway. We made direct recordings of intra-abdominal pressure and respiratory flow before and after giving fentanyl 25 μg i.v.

Results. Satisfactory records were obtained in 11 patients. Before fentanyl, the abdominal pressure changes were small and had an inconsistent pattern, increasing in inspiration in seven patients and during expiration in five. After fentanyl, an increase in pressure during inspiration was seen in only two patients, and the intra-abdominal pressure during expiration was increased. The mean value of maximum abdominal pressure (which always occurred during expiration) increased from 17 (sd 5) cm H2O before to 25 (9) cm H2O after fentanyl (P<0.01).

Conclusions. Direct measurements support previous findings that opioids stimulate active phasic expiratory activity and can cause large increases in abdominal pressure.

Br J Anaesth 2002; 88: 384–8

Keywords: analgesics, opioid, fentanyl; muscles, respiratory

Accepted for publication: November 8, 2001

Materials and methods

After ethics committee approval, we obtained written informed consent from 14 women, ASA I or II, about to have gynaecological laparoscopy as a day-case procedure. We noted their age, height and weight. A venous cannula was placed in the dorsum of the hand and anaesthesia was induced with up to 50 mg of propofol, followed by inhalation of sevoflurane in nitrous oxide and oxygen. No other drugs were administered at induction and no patient received premedication. After an adequate depth of anaesthesia was attained, an LMA was inserted. Anaesthesia was maintained with sevoflurane in nitrous oxide and oxygen, breathed from a circle system with a carbon dioxide absorber. At the end of the surgical procedure, all ports and instruments were left in place and the intra-abdominal pressure was measured from a connection to the carbon dioxide insufflator tubing using a differential pressure transducer (Gaeltec TC50, Gaeltec, Dunvegan, Isle of Skye, UK). The insufflator was switched off so that the tubing acted as a passive connection to the abdominal cavity. Gas was not vented from the abdomen until the study.

Patients are often anaesthetized for laparoscopic gynaecological procedures breathing spontaneously with a laryngeal mask airway (LMA†). In these circumstances, opioids have to be given in small doses to avoid excessive respiratory depression. We noted that even when a small dose of fentanyl was given, abdominal pressure increased and the high-pressure alarm on the carbon dioxide insufflator was often activated. Previous studies have shown that large doses of fentanyl frequently cause rigidity, but generally reported truncal rigidity, rather than a specific effect on abdominal muscles. Morphine in large doses can increase abdominal muscle activity, but the actual dose required to cause this phenomenon and the resultant mechanical effects were not evident. We have measured the effects of a small dose of fentanyl (25 μg) on intra-abdominal pressure.


‡LMA® is the property of Intavent Limited.
recordings were completed. The pressure signal was digitized by a data acquisition system (1401 interface and Spike 2 software, CED, Cambridge, UK) and Spike 2 software. We also recorded a signal indicating respiratory flow by measuring the pressure drop across a second breathing filter at the patient connection using a differential pressure transducer (Furness FC 10, Furness Controls Ltd, Bexhill, East Sussex, UK). This signal allowed the changes in intra-abdominal pressure to be related to the phase of respiration.

The respiratory rate, inspired concentration of oxygen, and end-tidal concentrations of carbon dioxide, nitrous oxide and isoflurane were recorded from the digital display of a Capnomac monitor, which sampled continuously at the patient connection.

When the breathing pattern and inspired and expired gas compositions were stable, abdominal pressure and respiratory flow waveforms were recorded for 30 s. Fentanyl, 25 μg in 5 ml normal saline, was given rapidly intravenously, followed by 5 ml normal saline. Pressure and flow were recorded for a further 2 min. At the end of this time measurements were stopped, abdominal gas was released, the cannulae were removed and clinical management was continued. We took readings of inspired and end-tidal values from the gas analyser just before and 2 min after administration of fentanyl. Recordings were also made in two patients in whom only the saline flush was administered.

Pressure and flow signals were converted into text files and analysed using a spreadsheet program on a personal computer. The pressure transducer was calibrated using a water manometer.

We analysed three complete respiratory cycles, immediately before and 100 s after giving fentanyl. We displayed these data graphically and measured the following values in each cycle: the minimum intra-abdominal pressure during inspiration; the intra-abdominal pressure at onset of expiration; the maximum intra-abdominal pressure during expiration; and the intra-abdominal pressure at the end of expiration. We also measured the time from the start of each respiratory cycle to each chosen value. We took means of

<table>
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<th>Patient</th>
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<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>$F_{O_2}$ Before</th>
<th>$P_{CO_2}$ Before</th>
<th>% N₂O Before</th>
<th>$F_{iso}$ Before</th>
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Table 1 Patient characteristics and respiratory measurements before and after giving fentanyl.

Fig 1 Time course of change in respiratory flow (upper trace) and abdominal pressure (lower trace) after fentanyl in a representative patient. The spikes of pressure on the trace represent artifact caused by movement by the surgeons.
the measurements from the three cycles and constructed an
average respiratory pressure cycle for each patient, before
and after fentanyl.

Statistics
Group results are expressed as mean and SD and data were
analysed by paired t test (Minitab v 12.1, Minitab Ltd,
Coventry). Statistical significance was assumed if \( P<0.05 \).

Results
We recruited 14 women, and present data from 11. Three
patients were excluded, two because of gas leakage at port
sites causing loss of the pneumoperitoneum during record-
ing, and one because the exact time of fentanyl adminis-
tration was not recorded. For the remaining 11 patients,
mean age was 34 (range 18–45 yr), mean weight (SD) was 65
(5) kg and mean height 165 (6) cm. These and other
characteristics are shown in Table 1. As expected, after
giving an opioid, \( P_{\text{E}}\text{CO}_2 \) increased from 5.4 (0.7) to 6.0 (0.7)
kPa and respiratory rate decreased from 32 (8) to 23 (7)
bpm. A record of pressure and flow from a typical patient is
shown in Figure 1, demonstrating a progressive change in
breathing pattern with prolongation of expiration, and a
simultaneous, marked increase in abdominal pressure
changes during expiration.

Before fentanyl was given, we noted three patterns of
abdominal pressure change within the respiratory cycle: (i)
pressure increase during inspiration only (six patients); (ii)
pressure increase during expiration (four patients); (iii)
biphasic changes in pressure, increasing at the end of
inspiration and again towards the end of expiration. This
pattern was seen in only one subject before giving fentanyl,
but was noted after fentanyl in two patients. Examples are
given in Figure 2.

After fentanyl, in all but one patient (Patient 3), a large
increase in intra-abdominal pressure during expiration was
noted. A small increase during inspiration was seen in two
other patients, as noted above. Minimum abdominal pres-
sure (which occurred during inspiration) increased from 13
(4) to 14 (4) cm H\( _2 \)O after fentanyl. Maximum abdominal
pressure (which occurred during expiration) increased from
17 (5) to 25 (9) cm H\( _2 \)O after fentanyl and the duration of
expiration was increased from 1.1 to 1.8 s (both \( P<0.01 \)).
Figure 3 summarizes these changes for the whole group,
showing the mean pressures and times during an average
respiratory cycle before and after fentanyl. The pressure
changes over the period of expiration increased significantly
after fentanyl \( (P<0.01) \).

Discussion
In normal quiet breathing in the conscious subject, inspira-
tion is an active process involving contraction and descent
of the diaphragm. The pressure in the abdomen increases. If
the subject is upright, the abdominal muscles are more
active than in the supine position, and the increase of
pressure in the abdomen associated with contraction of the

![Fig 2 Examples of different pressure waveforms. Broken line: respiratory
flow (inspiration downward deflection.) (A) A small increase during inspiration;
(B) increase during expiration; (C) increases during both inspiration and expiration.
](image)

![Fig 3 Mean (sd) pressure and time during the respiratory cycle in all
patients studied. The end of inspiration is marked by the vertical broken
line. Broken line: before fentanyl; solid line: 100 s after fentanyl.](image)
Fentanyl – effects on abdominal pressure

Diaphragm is greater than when the subject is supine.\textsuperscript{4} Expiration is usually passive, and as the diaphragm relaxes, intra-abdominal pressure decreases. However, during anaesthesia with spontaneous respiration, activity of the abdominal muscles has been recorded during expiration,\textsuperscript{5, 6} and opioids enhance this activity.\textsuperscript{3} Thus abdominal pressure rises in expiration.\textsuperscript{5} In normal conscious subjects, little or no activity is found in the recti or external oblique muscles, but activity is usually present in the transversus abdominis and internal oblique muscles, indicating that expiration is always active to some extent.\textsuperscript{7} A progressive increase in abdominal muscle action during pentobarbital anaesthesia has been reported in dogs.\textsuperscript{8}

In some of the patients we studied, abdominal pressure increased during inspiration, caused by contraction of the diaphragm. In normal conscious subjects, this is the major influence on abdominal pressure. However, in our subjects this pressure change was small, and in some patients no increase in pressure during inspiration was detected, suggesting that either the diaphragm was contracting poorly or that this pressure change was concealed by the actions of other muscles. For example, relaxation of the abdominal muscles or, less likely, inspiratory activity of the rib-cage muscles could reduce the increase in abdominal pressure during inspiration and conceal any pressure change generated by contraction of the diaphragm.

Pressure changes consistent with some expiratory activity of the abdominal muscles were noted before opioid administration but these changes increased markedly and consistently after fentanyl. The close temporal relationship of these changes to the injection, and the characteristics of fentanyl, make other causes very unlikely. There were no changes in breathing pattern, gas measurements, or pressure after saline administration. We made measurements of the effects after 100 s because this would reliably give three breaths to analyse before the recording stopped at 120 s. We did not have ethical permission to prolong the recording further, and it is possible that the pressure changes were not maximal when we made our measurements. However, because ventilation is reduced, other confounding factors such as carbon dioxide tension, anaesthetic depth, and perhaps cerebral blood flow, will be changing too. Early measurements allow these factors to have less impact, and interpretation of changes noted after prolonged observations would have to be limited. Other studies have shown that the effects of fentanyl in the conscious subject take considerably longer to cause rigidity,\textsuperscript{9} and some even suggest that loss of consciousness may be a necessary condition for the feature to develop.\textsuperscript{10} Such observations are consistent with the cerebral kinetics of fentanyl,\textsuperscript{11} but animal studies suggest that the rigidity may be mediated by brainstem structures and it is evident that the respiratory effects of fentanyl are equally prompt.

Although these effects of opioids have been noted before, our study shows that a modest dose can cause a considerable increase in abdominal pressure change during expiration, to well above the limits usually set for gas insufflation during laparoscopy. However, the pressures generated by maximum expulsive efforts can be at least 10 times the values we noted.\textsuperscript{12} Distension of the abdomen by gas insufflation during laparoscopy would increase the length of the abdominal muscle fibres and might augment the tension the fibres could generate and the pressure changes produced. However, animal experiments suggest that the abdominal muscles, in contrast to the diaphragm, normally work over a limited range of fibre length.\textsuperscript{13}

The effects of opioids on the abdominal muscles may be increased during anaesthesia. In healthy conscious volunteers, morphine 2 mg kg\textsuperscript{-1}, caused abdominal muscle activity in expiration and this activity was greatly increased by 70\% nitrous oxide, which caused ‘board-like’ rigidity and prevented mechanical ventilation.\textsuperscript{3} Abdominal muscle activity of this degree is potentially harmful, reducing the lung volume at end expiration, which could impair gas exchange by causing airway closure.\textsuperscript{14} During laparoscopy, abdominal muscle contraction will reduce the volume of gas that can be introduced before the pressure limit of the insufflator is reached and this could impair surgical access. During pelvic floor surgery, increased venous pressure caused by increased abdominal pressure may cause excessive bleeding. Abdominal muscle action can cause abnormal patterns of respiratory movement, which could be difficult for the inexperienced anaesthetist to distinguish from movements seen when airway obstruction is present, and lead to misdiagnosis and mismanagement of the airway.

There are several agents that could potentially reduce this effect. Although \(\mu\) opioid effects are responsible for increased muscle tone, the effects are reduced by \(\delta\) or \(\kappa\) opioid agonists.\textsuperscript{15} Opioids increase muscle activity by action at several central sites, including the peri-aqueductal grey matter and the locus coeruleus. Lesions in the latter site reduce the ability of opioids to increase muscle tone\textsuperscript{16} and it is possible that other agents that act at this site, such as \(\alpha_2\) adrenergic agonists, could modulate this process. The control of muscle tone from the locus coeruleus is by \(\alpha_1\)-adrenergic and glutaminergic pathways, probably arranged in series.\textsuperscript{17} The \(\alpha_1\) antagonist prazosin antagonizes the central effects of opioids whereas the spinal \(\alpha_2\) blocker yohimbine does not.\textsuperscript{18}

In summary, respiration is active in both expiration and inspiration but in the normal conscious subject expiratory activity in the abdominal muscles is small. Both anaesthesia and opioids can increase this activity. Even a small dose of opioid during anaesthesia increases abdominal pressure during expiration. In our patients, the expiratory pressure changes dominated the pressure waveform, suggesting a radical change in the mechanics of breathing in the anaesthetized patient given an opioid. During anaesthesia with spontaneous breathing, anaesthetists should be aware of the marked changes in abdominal pressure that can be brought about by even small doses of fentanyl.
References


