Respiratory mechanics after abdominal surgery measured with continuous analysis of pressure, flow and volume signals†

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Summary
We studied 10 patients during the first night after upper abdominal surgery to assess the effect of airway obstruction on chest wall mechanics, by recording nasal gas flow and carbon dioxide concentration, rib cage and abdominal dimensions, abdominal muscle activity, and oesophageal and gastric pressures. The mean duration of study of each subject was 5.8 h, and 5.2 h were analysed. The median proportion of time spent breathing with normal mechanics was 29% (interquartile values 0–57%). Abnormal abdominal mechanical events were common and associated with airway obstruction (P<0.001). Two common patterns of abnormal pressure and movement were found. In the first, abdominal pressure decreased at the onset of inspiration and there was a phase lag in abdominal movement. The incidence was 33 (14–50) %. In the second pattern, abdominal pressure decreased and in addition the abdominal wall moved inwards at the onset of inspiration. This occurred for 34 (0–52)% of the time. Both patterns were associated with evidence of increased activation of the abdominal muscles during expiration, changing the relationship of abdominal and pleural pressure changes and chest wall movements. Such changes have been interpreted previously as evidence of diaphragm dysfunction. (Br. J. Anaesth. 1996;77:317–326)

Key words

Abdominal surgery influences respiratory pattern, but effects on respiratory muscles are not well described. Measurements of respiratory movements and pressures have led some workers to conclude that the activity of the diaphragm is impaired1 2. After surgery, i.v. or extradural administration of opioids can cause episodic airway obstruction and result in periods of hypoxaemia3 4.

We investigated systematically how analgesia, airway obstruction and hypoxia may be related. To select simple but appropriate methods of measurement of these events, we examined a limited number of patients on the first night after operation. We intend in subsequent studies to measure fewer variables, which could be performed more easily, yet still be interpreted correctly. We also examined the possibility of “diaphragmatic inhibition”, which has been inferred in previous studies from the relationship between pleural and abdominal pressure changes.

Patients and methods
THEORY OF ANALYSIS
We used a graphical analysis of respiratory pressures and dimensions. Chest wall movement (fig. 1d) is considered to be in two compartments, rib cage and abdomen, which can move independently to change chest wall and lung volumes, although commonly both expand simultaneously on inspiration and return along the same trajectory on expiration. Action of other muscles, such as the abdominal wall muscles, can separate the inspiratory and expiratory lines. A small amount of asynchrony is normal5. Paradoxical motion can occur, despite inspiratory flow, when one compartment moves inward but not sufficiently to negate the outward motion of the other. With no flow, movement is constrained to the “isovolume” line, where the volume changes of the rib cage and abdomen are equal and opposite. Often, outward motion of the abdomen on inspiration is considered to indicate the action of the diaphragm, and outward rib cage motion to indicate the action of the intercostal and other inspiratory rib cage muscles. However, the diaphragm contributes considerably to rib cage expansion. Its costal fibres elevate the rib margin6. If the abdominal wall is firm, then the relatively incompressible abdominal contents form a “fulcrum” that allows contraction of the diaphragm to increase abdominal pressure, which expands the lower rib cage through the “zone of apposition” where the diaphragmatic and rib cage pleural surfaces are in contact7. The relative amount of rib cage and abdominal displacement caused by diaphragm action depends on the relative compliances of these two compartments5, and also on the action of other muscle groups at the same time8. If the abdominal wall is tense, then the diaphragm mainly expands the rib cage. Observing the motion of the rib cage and abdomen (such as RC/AB ratio) is
not enough to allow changes in activity of the diaphragm to be inferred unless the compliances of the two compartments remain constant.

**ANALYSIS OF PRESSURES**

Measurements of abdominal and pleural pressures (conveniently plotted graphically in fig. 1) allow better discrimination of respiratory muscle action.

**Action of the rib cage muscles**

Inspiratory action of rib cage muscles (such as external intercostals and scalenes) expands the rib cage and decreases pleural pressure $P_{pl}$. If the diaphragm is inactive, transdiaphragmatic pressure is zero ($P_{di} = P_{ga} - P_{pl}$). Consequently, as pleural pressure decreases, abdominal pressure decreases equally. The diaphragm and abdominal contents are drawn cranially, and the abdominal wall moves inwards.

**Abdominal expiratory muscle action**

Expiratory muscle action alone has the opposite effect on $P_{ab}$ and $P_{pl}$. Inward motion of the diaphragm subsequently relax and result in descent of the diaphragm. This action is difficult to distinguish from rib cage activation.

**Diaphragm action**

Contraction of the diaphragm generates a transdiaphragmatic pressure. The relative changes in pleural pressure and abdominal pressure during inspiration depend on the relative compliances of the rib cage and abdomen (fig. 1a). If the compliance of the abdomen were infinite, then pressure change would occur only by a decrease in pleural pressure. In practice, transdiaphragmatic pressure cannot be generated without a corresponding increase in abdominal pressure. If the abdominal compartment has a very small compliance, abdominal pressure increases with a very small reduction in pleural pressure. It follows that the trajectory of the $P_{ab}$ against $P_{pl}$ plot caused by the diaphragm acting alone lies between these lines. If the diaphragm and rib cage muscles act together in inspiration, they can move the pressures in the segment between the line indicating zero abdominal compliance and the line indicating rib cage muscle action alone.
Diaphragm action may also be assessed with a plot of abdominal pressure vs transdiaphragmatic pressure (fig. 1a). Rib cage action decreases abdominal pressure with no change in Pdi and results in a vertically descending plot. Abdominal expiratory muscle action increases abdominal pressure with no change in Pdi (generating a plot opposite to that seen with rib cage inspiratory action, as has been noted in the Pab vs Ppl diagram). Action of the diaphragm against a rigid abdomen results in equal increases in Pab and Pdi. If abdominal compliance is infinite, Pdi increases alone. Hence this plot differs from the previous (Pab vs Ppl plot) because the sector for sole action of the diaphragm is smaller. However, as in the preceding diagram, any combination of diaphragm and rib cage action generates a plot in the sector between the line of zero abdominal compliance and the rib cage line. Although more information on the activity of chest wall muscles may be obtained from such plots than from measurements of compartmental movement, the relative contribution of each group of muscles cannot be distinguished exactly by this analysis: in particular, abdominal relaxation and rib cage action appear similar.

**ABDOMINAL DIMENSION/ABDOMINAL PRESSURE PLOT**

*(FIG. 1c)*

Action of inspiratory rib cage muscles and relaxation of abdominal muscles can be distinguished by plotting abdominal dimension vs abdominal pressure, as in a compliance graph. **Contraction of the expiratory muscles** generates inward movement of the abdominal wall and an increase in abdominal pressure. Inward wall movement can occur only if another part of the abdominal container is displaced, such as by cranial displacement of the diaphragm. If both the diaphragm and abdominal expiratory muscles contract at the same time, abdominal pressure increases with little decrease in abdominal dimension. **Inspiratory action of the rib cage muscles alone** moves the abdominal wall inwards and decreases abdominal pressure. **Diaphragm action** increases abdominal pressure and displaces the abdominal wall outward, and the slope of this plot indicates the compliance of the abdominal wall. Simultaneous inspection of this plot and the Pab vs Ppl plot (fig. 1a) allows inspiratory events associated with abdominal relaxation to be distinguished from those caused by the inspiratory action of the rib cage.

**AIRWAY OBSTRUCTION**

If airway resistance is increased, muscle shortening is reduced and the tension developed by the muscles for a given degree of activation is increased. Pleural pressure decreases more, and gastric pressure increases less, as there is less displacement of the diaphragm. In addition, pleural pressure can no longer be used as an appropriate indicator of lung volume, as lower airway, alveolar and pleural pressures all decrease substantially during the inspiratory effort. In this condition, transpulmonary pressure (Pao - Ppl, where Pao = pressure at the airway opening) differs considerably from the pressure difference between the alveolus and pleural space which is proportional to lung volume.

We developed the hypothesis that prevention of muscle shortening could increase neural activation by reflex mechanisms, notably the stretch reflex mediated by muscle spindles. Rib cage muscles have a clear postural role and are richly supplied with muscle spindles90. Impedance of inspiration may result in their reflex activation, and an increased contribution to inspiratory force. This will alter the trajectories of the Pab vs Ppl, Pab vs Pdi and abdominal volume vs pressure plots in the directions shown in figure 1.

**PROCEDURE**

After obtaining approval from the regional Ethics Committee and written informed consent, we studied patients aged 40 yr or more, undergoing major abdominal surgery that would involve an incision at least partly above the umbilicus, and who would be cared for after operation in the surgical high dependency unit. Exclusion criteria were severe cardiac, respiratory or renal disease, or a body weight more than 130% of that predicted on the basis of sex, age and height. For postoperative analgesia, six patients received patient-controlled analgesia (PCA) with either morphine or diamorphine i.v., and four received a continuous thoracic extradural infusion of 0.125% bupivacaine containing diamorphine 0.033 mg ml⁻¹ at 10 ml h⁻¹.

A nasogastric tube with an integral oesophageal balloon (Mallinkrodt) was modified by attaching a gastric balloon to the tube tip. This was inserted via a nostril during anaesthesia, and placed to allow estimation of oesophageal and gastric pressures (Poes and Pga) using differential transducers (Furness FC10). Poes and Pga are used as measures of Ppl and Pab, respectively. Transdiaphragmatic pressure (Pdi) was calculated as (Pga - Poes). The gas volume in each balloon was checked regularly and adjusted. Pressure calibration was against a water manometer.

Patients received oxygen 2 litre min⁻¹ via nasal cannulae after operation. Pressure fluctuations in the nasal cannulae were measured to allow estimation of gas flow at the nose11. A narrow sampling tube was inserted through the centre of one cannula to allow sampling of carbon dioxide concentration for measurement by an infrared capnometer (Engstrom Eliza). Rib cage and abdominal dimensions were measured by inductance bands placed around the rib cage immediately below the axillae and around the abdomen at the level of the umbilicus, and avoiding the rib margins if possible. Disposable ECG electrodes were attached in the right upper quadrant of the abdomen to measure abdominal muscle EMG12. An Ohmeda 3700 pulse oximeter with a finger probe was used to record oxygenation continuously. The signals were recorded using a high speed analogue to digital converter (RTM8, Digitimer Ltd) which played into a domestic video recorder and allowed up to 8 h of continuous recording, sampling at a rate of 11 kHz per channel. The monitoring apparatus was set up in the evening after operation, and recordings made over the first night. One investigator (A.F.N.) remained with the patient overnight, supervising the quality of the recorded signals. Occasional adjustments were necessary, particularly to the
gastric and oesophageal balloons, and the nasal flow measurement system.

Further analysis involved playing back the videotaped record onto an eight-channel chart recorder (Graphtec WR3600). These records were then analysed by an observer who was unaware of the clinical circumstance of the patient. Each record was divided into segments of 5 min each, and each segment was classified according to preset criteria. First, the segment was classified as analysable, depending on the satisfactory recording of all the signals. Next, the presence or absence of central apnoea or hypopnoea, or obstructive apnoea or hypopnoea, was determined for each analysable segment. Central apnoea was determined by the absence of nasal flow, no changes in oesophageal pressure and no change in rib cage or abdomen dimension for more than 15 s. Central hypopnoea was defined by a reduction in amplitude of gas flow at the nose with a reduction in oesophageal pressure change. Obstructive hypopnoea was defined by a decrease in gas flow at the nose, associated with an increase in oesophageal pressure changes, and obstructive apnoea as no gas flow and increased oesophageal pressure changes.

The records were then re-analysed for the presence of mouth breathing, which was defined as continued chest wall movements, no change in oesophageal pressure swings and loss of nasal flow and carbon dioxide signals. This definition implies that the patient was breathing through the mouth and not through the nose: however at other times the patient may have been breathing through both the nose and mouth.

Finally, each segment was inspected for the presence of abnormalities of chest wall mechanics which were classified according to table 1. An inspiratory attempt was recognized from a decrease in oesophageal pressure. Because the exact time of onset of inspiration on the pressure trace was often indistinct because of cardiac oscillations, the times of onset and end of inspiration were obtained from the tracing of rib cage dimension. In one patient whose rib cage dimension sometimes decreased on inspiration, oesophageal pressure was used to time inspiration. EMG signals were not analysed formally by integration but were used to assess the presence and pattern of abdominal muscle activity.

Statistical analysis of the association between abnormalities present in each time segment of each subject was performed using a log-linear model with the P4F program from the BMDP package (BMDP statistical software, 1988). Partial association was investigated between pairs of the following categorical variables, after allowing for interaction between the other two variables: airway patency (i.e. airway clear or airway obstructed); chest wall mechanics (i.e. normal or abnormal chest wall and pressure changes); route of respiration (i.e. nose or mouth breathing); subject number (i.e. each patient).

To investigate the patterns of changes in the recorded variables, values for each patient were re-digitized from the replayed analogue signals using a commercial software package (Cardas, version 2.07, Oxcams Ltd, Oxford) at 20 Hz and plotted at 10 Hz using Fig P for windows version 1 (Biosoft, Cambridge), with a standard plot as in figures 3–5.

**Results**

Patient details are shown in table 2. Most patients were aged more than 60 yr and none was overweight. Results are given as median (quartile values). The number of 5-min periods of recording for each patient was 71 (68–72) and of these, 7 (5–10) could not be analysed. There was no evidence of airway obstruction in only 10 (3–22) of the periods analysed, and no abnormality of chest wall mechanics in 14 (0–36). Mouth breathing varied considerably between patients, being present in a median of 10 (1–38) periods per patient. The frequency of these abnormalities for each patient is given in table 3. There was no clear difference between patients using PCA and those receiving extradural analgesia. Airway obstruction and abnormal chest wall mechanics were related significantly ($P<0.0001$), but the relationship between mouth breathing and abnormal mechanics was not significant ($0.05<P<0.1$).

Table 3 also gives the relative distribution of the abnormalities of chest wall mechanics. There was no clear difference between the two types of analgesia. Both gastric pressure paradox and abdominal movement paradox were frequent (33 (14–50) % and 34 (0–52) % of the time, respectively). We noted that when these patterns were present, there was increased abdominal muscle activity on the EMG record. Ventilatory frequency over the study varied considerably between patients, with a median of 13.5 (9.5, 14.5) bpm. For each patient, ventilatory frequency also varied considerably within the study, increasing with arousal. There were no episodes of central apnoea. Determination of the incidence of central hypopnoea was difficult. We defined central hypopnoea as a reduction in both nasal gas flow and oesophageal pressure change during inspiration, that is a decrease in both tidal volume and effort. However, such changes were universal during recovery from an episode of airway obstruction, associated with a presumable decline in hypercapnic stimulation. A decrease in both nasal gas flow and oesophageal pressure change was not evident at other times, and we concluded that "central hypopnoea" was not present in these patients.

**Table 1** Summary of changes at the start of inspiration for different breathing patterns

<table>
<thead>
<tr>
<th>Pattern of Change</th>
<th>Change in Gastric Pressure</th>
<th>Movement of Abdomen</th>
<th>Movement of Rib Cage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Increase</td>
<td>Outward</td>
<td>Outward</td>
</tr>
<tr>
<td>Gastric pressure paradox</td>
<td>Decrease</td>
<td>Outward</td>
<td>Outward</td>
</tr>
<tr>
<td>Abdominal movement paradox</td>
<td>Decrease</td>
<td>Inward</td>
<td>Outward</td>
</tr>
<tr>
<td>Rib cage paradox</td>
<td>Increase</td>
<td>Outward</td>
<td>Inward</td>
</tr>
</tbody>
</table>

**Pattern of change in pressure and motion**

Most breathing was either normal, had paradox of gastric pressure or had paradox of both gastric pressure and abdominal movement. An example is given in figure 2, showing progression from a normal pattern, to paradox of gastric pressure, then abdomin-
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nal movement paradox, followed by resumption of normal breathing.

Representative plots of breaths of each type, obtained from the records of another patient, from a period of 30 min, are shown in figures 3–5.

Normal breath (fig. 3)

Plots of $P_{ga}$ vs $P_{oes}$ and $P_{ga}$ vs $P_{di}$ showed “looping”. At the start of inspiration, $P_{oes}$ decreased promptly. The direction of the early inspiratory plot was just in the “diaphragm action” sector. In the second part of the inspiration, gastric pressure increased and remained increased during the first part of expiration. The plot of abdominal dimension against gastric pressure showed that in the first half of inspiration there was little increase in either gastric pressure or abdominal volume. In the second half of inspiration, abdominal dimension increased smoothly as gastric pressure increased. The plot of rib cage and abdominal dimensions showed that early in inspiration the relative proportion of rib cage dimension increase was greater than that of the abdomen, whereas during expiration both dimensions decreased progressively and in the same proportion, so clockwise “looping” is seen. This pattern was seen in all patients.

The changes in this type of breathing can be summarized as oesophageal pressure and rib cage dimension changing in phase in the later part of inspiration. In expiration, gastric pressure and transdiaphragmatic pressure decreased, and oesophageal pressure increased. The plot of abdominal dimension against gastric pressure decreased along a plot that was the reverse of inspiration.

Gastric pressure paradox (fig. 4)

Figure 4 shows a breath sampled in the same patient, approximately 30 s before the example in figure 3. Gastric pressure decreased early in inspiration. All patients showed this pattern of change at some time during the study. The $P_{ga}$ vs $P_{oes}$ and $P_{ga}$ vs $P_{di}$ plots showed an initial downward descent, and then an increase in gastric pressure in the second part of inspiration. The abdominal dimension/gastric pressure plot was most common, but in two patients an increase in abdominal dimension occurred as gastric pressure decreased. On the dimension plot, outward rib cage motion started with no initial change in abdominal dimension. In the second half of inspiration, and in expiration, the shape of this plot remained similar to that seen in a normal breath.

Table 2 Details of patients. RUQ = Right upper quadrant of the abdomen. Opioid doses are given as milligrams of morphine, assuming half the potency of diamorphine. Intraoperative doses include those given on recovery from anaesthesia. All extradural opioids were diamorphine. Intraoperative doses given to patients with extradurals are divided into extradural (e) and i.v. Pre-study amount = Amount given before the study started.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Incision</th>
<th>Analgesia</th>
<th>Opioid administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>46</td>
<td>185</td>
<td>81</td>
<td>RUQ</td>
<td>PCA</td>
<td>Intraoperative 15 28 8</td>
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<tr>
<td>2</td>
<td>M</td>
<td>64</td>
<td>175</td>
<td>74</td>
<td>Transverse</td>
<td>Extradural</td>
<td>Pre-study 13 i.v. 8 3</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>65</td>
<td>165</td>
<td>65</td>
<td>Transverse</td>
<td>PCA</td>
<td>During study 10 20 9</td>
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<tr>
<td>4</td>
<td>M</td>
<td>74</td>
<td>173</td>
<td>60</td>
<td>Midline</td>
<td>PCA</td>
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<tr>
<td>5</td>
<td>F</td>
<td>71</td>
<td>153</td>
<td>67</td>
<td>RUQ</td>
<td>Extradural</td>
<td>3 i.v. 8 4</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>43</td>
<td>173</td>
<td>82</td>
<td>Midline</td>
<td>PCA</td>
<td>12 40 13</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>64</td>
<td>168</td>
<td>83</td>
<td>Transverse</td>
<td>Extradural</td>
<td>2 e 7 4</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>64</td>
<td>163</td>
<td>81</td>
<td>Midline</td>
<td>PCA</td>
<td>10 19 6 4</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>73</td>
<td>157</td>
<td>67</td>
<td>RUQ</td>
<td>PCA</td>
<td>10 26 4 4</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>66</td>
<td>171</td>
<td>74</td>
<td>Transverse</td>
<td>Extradural</td>
<td>5 i.v. + 3 e 5 4 4</td>
</tr>
</tbody>
</table>

Table 3 Incidence and duration of respiratory abnormality in the patients studied

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Analgesic method</th>
<th>Abnormal mechanics</th>
<th>Airway obstruction</th>
<th>Mouth breathing</th>
<th>Incidence of abnormality (%)</th>
<th>Percentage of time spent with different mechanical patterns</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>Gastric pressure paradox</td>
<td>Abdominal motion paradox</td>
<td>Rib cage motion paradox</td>
<td></td>
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<tr>
<td>1</td>
<td>PCA</td>
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<td>58</td>
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<td>2</td>
<td>Extradural</td>
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<td>42</td>
<td>2</td>
<td>44</td>
<td>8 48 0</td>
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<tr>
<td>3</td>
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<td>65</td>
<td>24 5 6</td>
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<td>91</td>
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<td>43 57 0</td>
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<td>6</td>
<td>PCA</td>
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<td>98</td>
<td>69</td>
<td>0</td>
<td>9 91 0</td>
</tr>
<tr>
<td>7</td>
<td>Extradural</td>
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<td>0</td>
<td>0</td>
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<td>0</td>
<td>50 50 0</td>
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<tr>
<td>10</td>
<td>Extradural</td>
<td>67</td>
<td>72</td>
<td>8</td>
<td>33</td>
<td>16 51 0</td>
</tr>
</tbody>
</table>
Abdominal movement paradox (fig. 5)

Gastric pressure, oesophageal pressure and abdominal dimension decreased during the first part of inspiration. On the plot of abdominal dimension and gastric pressure, gastric pressure and abdominal dimension decreased in concert at the start of inspiration but then increased in the second half of inspiration. A particular feature of this type of breathing was that during expiration, abdominal dimension decreased as gastric pressure either remained unchanged or increased. This is shown clearly in the example, and was seen in all subjects. As expected, rib cage dimension increased but abdominal dimension decreased at the start of inspiration, but again in late inspiration both dimensions increased together.

In several subjects, rapid and frequent changes between these breathing patterns were noted, whereas in other patients more stable patterns of breathing, particularly that of gastric pressure paradox, were noted. Often airway obstruction became progressively more severe and ended with apparent arousal and restoration of a temporarily normal breathing pattern.

All patients received oxygen 2 litre min⁻¹ from a nasal cannula. Despite airway obstruction, oxygen saturation did not decrease to less than 90% in any patient. Occasionally the oxygen saturation display was less than 90%, but in each of these events, the observer could see both the patient and the plethysmographic waveform displayed by the oximeter, and judged the low saturation value artefactual, usually caused by patient movement.

Discussion

We have described the patterns of respiratory mechanics that occur after major abdominal surgery...
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in association with breathing and breathing disturbances. These patterns proved remarkably consistent, although the relative changes were complex. Statistical analysis of the changes was categorical because several factors prevented a more quantitative approach.

The oesophageal balloon method is not of great value for estimating the absolute value of pleural pressures in supine subjects, particularly when lung volume is small, although pressure changes can be measured satisfactorily. In our subjects, abdominal muscle activity was frequently present to some degree, so the absolute values of gastric pressure would be affected. However, we believe that within-breath changes can be used descriptively, as we have done in the example shown, and that breaths a few minutes apart (as in figs 3–5) can be compared usefully.

Measurement of chest wall motion using inductance bands is a simple and non-invasive method, but is often difficult in recumbent patients or after operation. Our patients were always supervised closely to ensure that the bands remained in about the same place, but changes in position disturbed the signal for some time and was the most common reason for an uninterpretable trace. There are several alternative methods for calibration of the inductance band device, but only the original isovolume manoeuvre is sufficiently robust statistically to properly partition abdominal and rib cage volume con-
butions⁷. Repeated calibration of the bands under the conditions of this study was impossible, and we only used these signals qualitatively. For example, a simultaneous reduction in the changes in oesophageal pressure and rib cage dimension was interpreted as a decrease in respiratory drive. Such changes were noted cyclically in some patients during recovery from an episode of obstruction.

We used nasal pressure signals similarly to provide a breath-by-breath indication of flow. Clear changes were seen in association with obstruction, followed by arousal, with transient increases in flow and breathing movements. EMG signals can be quantified by rectification and integration, but we were unsure of the stability of the recording system over several hours. The signals were used to indicate the phase and pattern of external oblique activity.

The patterns of motion observed are worthy of comment, and each variable studied will be considered in turn. Pressures related to elastic and resistance components can be distinguished by their relationship to flow and volume change. For example, the flow-related component of the oesophageal pressure change is more than doubled after sedation with midazolam¹⁸ because airway resistance is increased. Oesophageal pressure change is greater, for the same degree of neural activation of the inspiratory muscles, if inspiration is impeded. This is partly because the muscles shorten less and more pressure is generated. The extreme example of this is occluded inspiration where the muscles contract but cannot shorten, other than to distort the chest wall, and oesophageal pressure decreases considerably more than during a normal breath. If airway resistance is increased, but flow is not reduced, then the inspiratory muscle activity must have increased. Transpulmonary pressure during inspiration, associated with that flow, is greater because of the increased resistance. We infer that airway obstruction is responsible for the increased change in oesophageal pressure early in inspiration that could be seen when the abdominal wall moved inward during inspiration, and this inference was supported by the statistical association between episodes of airway obstruction and abnormal chest wall mechanics.

In a normal breath, gastric pressure and abdominal dimension increase and decrease together, suggesting that abdominal motion is the result of diaphragm action, and that the compliance of the anterior abdominal wall does not change during the respiratory cycle.

On the other hand, abdominal wall compliance clearly does alter when gastric pressure decreases at the start of inspiration (fig. 4). At the same time, rib cage dimension increases and oesophageal pressure decrease rapidly. Such changes could be caused by the inspiratory action of the rib cage. They could equally well be the result of relaxation of the abdominal muscles, if these muscles have acted to decrease the dimensions of the lower rib cage, or possibly caused by both of these actions. These alternatives can be distinguished partly by considering the abdominal dimension/oesophageal pressure plot, so that the inspiratory action of rib cage muscles and relaxation of the abdominal expiratory muscles generate movement of the plotted values in different directions. The slope of the plot depends on the compliance of the abdomen. Abdominal compliance is small if the abdominal muscles and diaphragm are contracting at the same time, whereas it is greater if the inspiratory rib cage muscles are active alone, as the abdominal contents are drawn passively in a cranial direction as pleural pressure is reduced. In this study, abdominal pressure decreased with little change in abdominal dimension, so the abdomen or diaphragm was relaxing. Previous observations suggest that large changes in gastric pressure, particularly during expiration, can be related directly to abdominal muscle activity¹⁹ ²⁰.

When both gastric pressure and abdominal volume decrease together, there must be a dominant effect of the rib cage inspiratory muscles. Even when the abdomen moves paradoxically on inspiration, transdiaphragmatic pressure still increases (fig. 5), indicating that the diaphragm is active, and that it is contracting against an increased respiratory impedance. Indeed, pressure changes tended to be greater when respiration was impeded. However, this action does not displace the abdominal contents because gastric pressure shows no increase. In such conditions, assessment of “dysfunction” using changes in transdiaphragmatic and gastric pressure is impossible. We had no index of diaphragmatic activation, as could be provided by electromyography. However, the commonly used index of contractility, the ratio of EMG to Pdi, is altered by both posture and the degree of abdominal muscle activity²¹, and therefore this would not have been of great value. Other workers have found that maximum diaphragmatic electromyographic activity is reduced after abdominal surgery but this appears to be in proportion to the reduction in tidal volume²². Contractility appears unaffected²³. Similarly, extradural analgesia was noted to restore both electrical activity and tidal volume by approximately similar amounts²⁴. Paradoxical chest wall movements similar to those we have described have been noted in obese patients with sleep apnoea²⁵ and patients with airway obstruction after surgery²⁶.

We suggest that these abnormal patterns of gastric pressure and abdominal dimension indicate activity of the inspiratory rib cage muscles and abdominal expiratory muscles. Airway resistance probably activates the intercostal muscles because of the stretch reflex. Intercostal muscles have many muscle spindles¹⁰ and have a clear reflex response to imposed loads²⁷ ²⁸. In contrast, the diaphragm has few spindles and no clearly defined reflex response to loads, other than via vagal reflexes²⁹. Rib cage movement is other greater in early inspiration and this feature can be seen to a lesser extent even in “normal breathing” after operation (fig. 3). The decrease in gastric pressure was similar but not identical to that seen in unilateral diaphragm paralysis³⁰ or in the horse³¹ when the abdominal muscles are used to assist expiration. EMG assessment of the relative timing and activation of the diaphragm and intercostal muscle activity would be helpful to examine the possible reflex augmentation of intercostal activity in early inspiration.

Previous workers have used the relationship between gastric and oesophageal pressure changes to assess the relative contribution of the diaphragm¹². This approach is generally valid if only the diaphragm or inspiratory rib cage muscles, or both, are active; in
the present situation, there may be differences in the timing of activity of these muscles, the magnitude of their reflex activation by increased airway resistance may differ and, in addition, the abdominal muscles are active in expiration.

Opioid administration may enhance rib cage stiffness. Patients in this study received only moderate quantities of opioid (table 2). Extradural analgesia had no clear influence on abnormal chest wall mechanics.

Opioid administration may also influence the observed changes by provoking airway obstruction, particularly during sleep. Previous observers found that episodes of airway obstruction were associated with episodes of desaturation. Catley and colleagues found that hypoaxemic episodes occurred during sleep and were associated with obstructive apnoea and opioid analgesia. However, their identification of episodes of airway obstruction depended entirely on the quantitative use of the inductance band method, which is unlikely to remain accurate for long periods of time. Although these workers have illustrated paradoxical changes in chest wall dimensions similar to those in this study, such movements have been incompletely investigated and described. We found that outward rib cage motion was almost completely preserved during airway obstruction and conclude that although this movement does not indicate abnormalities of breathing in patients after operation, it provides a reliable indication of inspiratory effort. Measurement of gas flow at the nose in addition to rib cage movement allows airway obstruction to be recognized. The combined measurement of rib cage movement and gas flow at the nose could provide an indication of airway obstruction with minimal disturbance to the patient. An episode of obstruction would be recognized reliably by outward movement of the rib cage (showing an inspiratory effort) with absence of gas flow at the nose.

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