The abdominal muscles in anaesthesia and after surgery

G. B. Drummond

University Department of Anaesthesia, Critical Care, and Pain Medicine, 51 Little France Crescent, Edinburgh EH16 4SA, UK
E-mail: g.b.drummond@ed.ac.uk


Keywords: anaesthesia; muscle skeletal; surgery, abdominal

‘It is generally agreed that in normal man lying supine the act of expiration is passive’. These are the opening words of the classic paper on abdominal muscle activity during anaesthesia, by Freund, Roos, and Dodd in 1963. They had noted abdominal activity in clinical practice, so they studied 24 normal male volunteer subjects. They gave no premedicants and took care to relax the abdomen: they found no activity in conscious subjects. During anaesthesia with halothane, they found that the abdominal muscles became active (Fig. 1). Their findings have been amply corroborated in later studies, although often overlooked by investigators who have concentrated on inspiratory muscle actions, and perhaps come to incorrect conclusions when expiratory activity was a more logical explanation of their findings. For example, many writers have clung to the attractive early theory that the respiratory muscles are depressed by anaesthesia in a form of ‘ascending paralysis’, leaving the diaphragm working alone.

In his Hunterian Lecture to the Royal College of Surgeons of England in 1947, Howkins noted that respiratory complications after abdominal surgery were related to reduced diaphragm movement, and that this in turn could be related to reduced abdominal movement. He suggested a number of remedies that bear consideration today including early mobilization and physiotherapy, although others have vanished with medical progress: to prevent depression of breathing, analgesia was restricted to aspirin and bromide and an example of rapid recovery and good morale was to get a rear gunner back in a bomber on day 17 after surgery! Nevertheless, abdominal movement remains poorly understood in patients after major surgery. Expiratory activity can be easily demonstrated in the lower ribcage and upper abdomen, and causes large changes in abdominal pressure. Simultaneous measurements of several variables must be made to correctly infer the mechanical behaviour of the respiratory system, and previous theories based on limited measurements of movement or pressure have been re-considered and recently reviewed. Put simply, the relaxation of abdominal muscles which have been acting during expiration, will result in pressure changes in the pleural space similar to those caused by ribcage inspiratory activity. These changes can be mistaken for inadequate diaphragm activity. The factors that activate abdominal muscles after surgery appear to be opioid analgesia, airway obstruction, and perhaps wakefulness. Even after limited abdominal surgery, pressure increases during expiration can be considerable (Fig. 2) and would be expected to reduce FRC by about 20%. At present, we have no effective means of reducing the activity, apart from perhaps epidural analgesia.

Abdominal wall: anatomy and actions

There are three flat layers of muscle in the abdominal wall. From the inside out, these are the transversus, the internal oblique, and the external oblique. Together, they form the anterior shell that completes the container of the abdominal contents. Their fibres and aponeuroses are a geodetic continuum with the other sheet-like muscles of the trunk, arranged so that the wall of the abdomen can generate tension in all directions. However, as the radius of curvature is least in the transverse plane, it is the fibres of the transversus, that describe this curve, that are the most active and important in respiration. Despite the assertions of anatomy books that it ‘acts only on the abdominal contents’, these anterior muscles are balanced by the quadratus lumborum at the back: at the sides, the lower rib margin and the iliac crest are almost opposed. The quadratus lumborum acts in concert with the posterior, vertebral part of the diaphragm, especially as its extension, and thus has a distinctly different action from the anterior abdominal muscles. Inserted between these layers, the rectus abdominis appears to have a more postural role, and is often silent during breathing manoeuvres.
The anterior abdominal muscles have two distinct respiratory effects (Fig. 3). First, they pull on the rib margins. This pull is in a downward and inward direction, and if unopposed will reduce the volume of the rib cage, and press on the abdominal contents, displacing them and the diaphragm cranially. This action can be directly opposed by the costal fibres of the diaphragm, which can act to elevate the rib margins, if the central tendon is prevented from descending. Secondly, the abdominal muscles can increase intra-abdominal pressure. If not opposed, this pressure acts to increase the volume of the lower rib cage, by forcing it out by pressure across the pleural sulcus: the area of apposition of the diaphragm to the inside of the rib cage. The exact effects on the ribcage therefore depend on the relative degree of activation of the different muscles of the abdominal wall, and clinical inspection of the anaesthetized patient often reveals differential activation. Activation of both abdominal and lower rib cage expiratory muscles during halothane anaesthesia causes rib cage constriction, presumably reflecting the downward pull on the rib margins, rather than expansion which would result if the main action were through the increase in intra-abdominal pressure. In addition, the results of animal studies may not apply in man, because of the different shape of the ribcage.

As a whole, the abdominal muscles act on the hydraulic core of the abdominal contents, altering its shape as the different muscles generate tension in the abdominal wall, which may be considerably anisotropic. Changing curvature of the spine adds a further factor in the relationship between ribcage and abdominal dimensions and the volume of the lungs. Much remains to be understood about the more subtle shape changes, and many early reports may be only partly correct, in particular because some widely used sensors of abdominal motion do not detect this distortion very well.

Much has been written of the importance of adjusting the diaphragm to its optimal length and shape to generate a maximal inspiratory force when it contracts. There is no doubt that the force that the diaphragm can generate depends on its length, but it is not clear how important this theory is in practical terms. Even in patients with severe chronic obstructive lung disease, with diaphragms shortened by 20%, the motion and change of length during tidal breathing is the same as in normal subjects. Perhaps in acute circumstances such as exercise, in addition to aiding rapid expiration, abdominal muscle contraction can assist the diaphragm contract more strongly, although evidence suggests that it is already at its optimal length. In addition, the stretching effect does not usually persist in inspiration, as abdominal pressure decreases promptly at the onset of inspiration (see for example Fig. 2).

**Control of the expiratory muscles**

In a recent exhaustive review, Iscoe pointed out that in contrast to the inspiratory muscles, the muscles of expiration are relatively little known: yet this review has 15 pages of references, and a more complete account is not to be found. Some of the more pertinent aspects will be considered here. However, even his review does not mention, for example, any aspects of the effect of opioids on abdominal muscles.

The expiratory muscles are controlled by neurones in the ventral respiratory group in the medulla, and project, mainly though polysynaptic pathways, to the lower thoracic and
lumbar spinal cord. In the supine subject, the abdominal muscles are usually inactive in quiet breathing. In the sitting or standing position, they are active, and can have phasic activity during expiration. The transversus abdominis is the most easily activated, the obliques are less readily brought into action, and the rectus abdominis has the least prominent respiratory role. They become much more active when breathing is stimulated, by exercise, chemical stimuli, positive pressure on the airway, or voluntary hyperventilation (Fig. 5).

The expiratory muscles of the rib cage are activated during normal deep sleep, but the response to increases in airway pressure are reduced. Positive airway pressure is a stimulus for abdominal muscle activation. This is mediated by vagal afferents, transmitted via myelinated fibres, which probably represent activity from the classic lung stretch receptors. Activation of C fibres by lung irritation such as i.v. capsaicin inhibits the effects of positive airway pressure. This inhibition of the reflex response to positive airway pressure may explain why patients with lung disease, who may have lung inflammation, allow their lung volume to increase when constant positive airway pressure is applied, rather than reflex contracting their abdominal muscles to offset the increase. However, some of the control of the abdominal muscles must also be from muscle afferents, as dorsal root section, which will interrupt stretch receptor activity also inhibits the response to positive airway pressure.

The inspiratory and expiratory motoneurones are reciprocally activated during breathing, and the spinal cord pathway for breathing control is separate from pathways from the cortex that can activate the expiratory muscles or pathways that control other functions such as cough. For postural activity, the abdominal muscles have separate distinguishable patterns of activity, whereas their activity is coordinated during respiratory activity.

The activity of the muscles is achieved by an increase both in the number of active motor units and the frequency of firing of motor units, in almost equal amounts. The stimulation by hypoxia is reduced by hypocapnia.

The effects of abdominal muscle contraction

Effects on the lung

If abdominal muscle contraction decreases thoracic volume and transpulmonary pressure, then lung volume is reduced. The exact effects of a decrease are not clear. If this decrease was sudden, then airway resistance would increase as the airway dimensions decrease. More importantly, small airways could close as lung volume is reduced below closing capacity. In circumstances where FRC was changed acutely and passively, gas exchange is impaired with a decrease in arterial oxygenation. Active expiration also impairs oxygenation acutely. However, persistent expiratory activity may not have such an effect. If the chest wall is restricted by binding, or even if subjects voluntarily activate the expiratory muscles to reduce lung volume, the recoil pressure of the lung increases, and the airways dilate by a cholinergic mechanism, which might be expected to offset the propensity of airways to narrow and close. In addition, there are intrapulmonary reflexes to redistribute...
blood flow that would offset any persistent impairment in gas exchange. In patients after abdominal surgery, there is no doubt that FRC is reduced and that gas exchange is impaired, and the changes are correlated. However, measurement of airway closure after abdominal surgery is technically difficult and the explanation of hypoxaemia solely in terms of increased airway closure is not well supported.

**Effects on the chest wall**

In exercise, or when the abdominal muscles are activated by loading the inspiratory muscles, the action of the abdominal muscles is to reduce the end-expiratory volume below FRC. In this way, the abdominal muscles can contribute to ventilation. In dogs, anaesthetized in the prone position, about 40% of tidal volume may be generated by the relaxation of the abdominal muscles, as this is a favourable position for gravity to assist recoil of the abdominal wall. In man, abdominal activity may contribute about 20% of the work of breathing.

**Effects of anaesthesia**

In dogs, during stable isocapnic anaesthesia with i.v. agents such as thiopentone or propofol, FRC decreases by between 200 and 300 ml at a rate consistent with the rate of decrease of tonic inspiratory activity in ribcage muscles such as the sternomastoid and scalene, which act to elevate and fix the upper ribcage. After neuromuscular block, there is no further change in FRC. However, in patients anaesthetized with opioids, and breathing spontaneously, neuromuscular block causes an increase in the end-expiratory lung volume of about 400 ml. This is equivalent to removal of an expiratory force on the respiratory system of about 10 cm H$_2$O. The contrasting effects of the loss of inspiratory ribcage activity, and expiratory abdominal activity, in relation to anaesthesia, are shown in Figure 4. When a small dose of fentanyl is given to anaesthetized patients breathing spontaneously, intra-abdominal pressure rapidly increases by about 7 cm H$_2$O and the pattern of abdominal pressure change indicates contraction of the abdominal muscles during expiration. This activity is enough to contribute approximately 20% of the tidal volume.

Opioids increase skeletal muscle tone by a complex pathway involving the locus coeruleus and several transmitters, including glutamine agonism and alpha 2 adrenergic inhibition. After giving opioids, the neural pathway associated with rhythmic activation of the abdominal muscles is the usual pathway associated with respiratory activation of these muscles. In a neonatal rat preparation, pre-inspiratory neurones near the nucleus retrofacialis synapse with bulbospinal neurones in the nucleus retroambigualis. This nucleus is the main location of expiratory motoneurones. Activity passes from here to the first lumbar nerve root to generate abdominal muscle contraction in a respiratory pattern, which persists after opioid administration, even when inspiratory muscle activity is suppressed. Inspiratory and expiratory activity, at the spinal level, appear to be reciprocally controlled by a glycinergic inhibitory system. The action of opioids in this preparation is to selectively depress inspiratory motoneurones by both pre- and post-synaptic actions. However, others have found that opioids act on the rhythm generator, and certainly other depression by other neuromodulators affects pre-inspiratory neurones. Studies in less elemental animal preparations show that opioids activate thoracic motoneurons, but this activity may be entirely caused by the hypercapnia after opioid administration. In man, abdominal muscle activation during anaesthesia is stimulated by hypercapnia, but giving an opioid will increase the activity further. In the presence of an opioid, hypercapnia seems to have little further effect.
Specific circumstances in anaesthesia

In normal breathing, there is remarkable coordination of the activity of the respiratory muscles, so that the entire chest wall moves synchronously and proportionally. Generally, respiratory depression is attributed to a reduction in the central drive to breathe, but it is certainly possible that during anaesthesia, loss of coordination of the chest wall muscles, or changes in the mechanical properties of the respiratory system, may reduce the volume of breathing for a given ‘effort’ or central drive.

Derenne and co-workers favoured this possibility in a study that compared subjects before and during anaesthesia. They assessed central ‘effort’ using occlusion pressure, which was measured as the decrease in mouth pressure when the airway was transiently occluded at the start of inspiration. This provides an index of the force of activation of the inspiratory muscles. It quantifies the integrated inspiratory effort of the respiratory system, with the muscles contracting isometrically (although there may be shortening of some muscles and lengthening of others if the system distorts during this transiently occluded effort). The resultant measure of respiratory system activity can be combined with the volume change during a normal breath to calculate the ‘effective elastance’ of the system. This value can predict the response of the respiratory system when inspiration is impeded by adding a load to the inspiration, and can be thought of as a measure of the mechanical properties of the respiratory system when respiration is active (spontaneous) rather than passive (during mechanical ventilation). It is a measure of ‘stiffness’ of the respiratory system, and attributed predominantly to the length/tension relationship of actively contracting muscle: if a muscle’s shortening is impeded as it contracts, then the tension it generates is increased—an intrinsic load compensation.

Derenne and co-workers noted that during anaesthesia the ventilatory response was markedly reduced, indicated by a decrease in the response of the mean inspiratory flow rate (VT/TI) to increased carbon dioxide. However, the mean occlusion pressure values were not reduced during anaesthesia, although the slope of the response was less. In consequence, the relationship between occlusion pressure and mean inspiratory flow (elastance) was substantially different between the conscious and anaesthetized subjects, suggesting that their inspiratory muscles had to generate far more pressure to produce the same flow during anaesthesia. Their novel conclusion was that at least part of the depression of ventilation was caused by increased stiffness, or changed mechanics, of the respiratory system.

Is this conclusion justified, and is there an alternative explanation? There is no doubt that the occlusion pressure is increased during anaesthesia, but at that time these workers did not consider any contribution to this measure from the abdominal muscles. During stimulation with carbon dioxide, they could be contracting during expiration, and the...
early part of inspiration would be assisted by their relaxation. Indeed inspection of the occlusion pressures illustrated (their fig. 3) show a similar substantial effect early in inspiration, and they did show subsequently that occlusion pressure was generated at least in part by abdominal relaxation. Such a contribution, present during anaesthesia, but not in the control measurements, can explain their observations simply, without the hypothesis that the active elastance of the respiratory system has been affected. This explanation is also consistent with similar observations of occlusion pressure and ventilation. However, in other studies using the same agent (methoxyflurane) the same workers argued that expiratory activity was not present. As this agent is no longer used, it would be helpful to study other current agents to establish the contribution of abdominal muscle action to ‘occlusion pressure’, as there is no doubt that this is increased during anaesthesia.

Others have considered the change in pattern of respiratory movements to explain reduced ribcage responses to stimulation. In a study of adolescents, ribcage responses to stimulation of breathing with carbon dioxide were much more reduced than the abdominal movements during halothane anaesthesia. They attributed these changes to a loss of the contribution of the intercostal muscles, and present recordings that show the change with loss and recovery of consciousness. However, these records also show obvious paradoxical motion of the abdomen developing during anaesthesia, in contrast to the synchronous movement present in the conscious subject. Abdominal muscle action during expiration can expand the ribcage, by generating outward pressure on the lower ribs: at the onset of inspiration, as the abdominal muscles relax, the abdomen changes shape suddenly, and an indrawing is noted, causing a pattern of motion exactly the same as those illustrated in the report by Tusiwickz and colleagues. This explanation is perhaps more valid than attributing the changes to loss of intercostal activity as these muscles act more to stabilise the ribcage than to expand it, and are unlikely to be important agonists. Activation of abdominal muscles during anaesthesia was demonstrated in a later study, which was unable to replicate the large change in ribcage movements found in the study of Tusiwickz. Stimulation of respiration by re-breathing accentuated the activation of abdominal expiratory activity. In most subjects, the ribcage expanded in early expiration. One difficulty with interpreting the results of such studies is that the dimensions are from single circumferences of the ribcage and abdomen, and there is no doubt that the shape changes that occur are complex and poorly expressed by a single summative dimension. For example, the ribcage above and below the zone of apposition to the abdominal contents is exposed to different forces and can move in different ways, and the lateral and central parts of the abdomen can also move paradoxically in circumstances such as loaded breathing (Fig. 5).

Conclusions
Anaesthesia disturbs the coordinated motion of the chest wall. Although there is no doubt that central respiratory drive is reduced by anaesthetic agents, abnormal movements may possibly contribute to reduced ventilation and affect the distribution of ventilation and impair gas exchange. To obtain more exact information about the extent and impact of these changes, better methods for assessment of chest wall movement are required, preferably not involving big expensive apparatus or ionizing radiation. In addition, with a model to investigate features such as how opioids increase abdominal contraction, we might develop treatments to modify these effects, and perhaps reduce respiratory complications after surgery, the goal of Howkins in 1947.

References
6 Bergman NA. Reduction in resting end-expiratory position of the respiratory system with induction of anesthesia and neuromuscular paralysis. Anesthesiology 1982; 57: 14–7
16 Derenne J-P, Couture J, Isoe S, Whitelaw WA, Milic-Emili J.
21 Drummond GB. Reduction of tonic ribcage muscle activity by anesthesia with thiopental. Anesthesiology 1987; 67: 695–700
37 Goldman JM, Lehr RP, Millar AB, Silver JR. An electromyographic study of the abdominal muscles during postural and respiratory maneuvers. J Neurol Neurosurg Psychiatry 1987; 50: 866–9
43 Howkins J. Movement of the diaphragm after operation. Lancet 1948: 85–8
44 Issoc S. Control of abdominal muscles. Prog Neurobiol 1998; 56: 433–506
53 Manco JC, Hyatt RE. Relationship of air trapping to increased lung recoil pressure induced by rib cage restriction. Am Rev Respir Dis 1975; 111: 21–6
54 Martin JG, DeTroyer A. The behaviour of the abdominal muscles during inspiratory mechanical loading. Respir Physiol 1982; 50: 63–73
60 Miller AH. Ascending respiratory paralysis under general anesthesia. J Am Med Assoc 1925; 34: 201–2
63 Newsom-Davis J, Plum F. Separation of descending spinal pathways to respiratory motoneurons. Exp Neurol 1972; 34: 78–94
77 Warner DO. Diaphragm function during anesthesia: still crazy after all these years. Anesthesiology 2002; 97: 295–7
82 Warner DO, Warner MA, Ritman EL. Atelectasis and chest wall shape during halothane anaesthesia. Anesthesiology 1996; 85: 49–59