An editorial written 10 years ago (Jones, 1977) postulated that the abnormality of gas exchange associated with anaesthesia was the result of decreases in the tone and movement of the chest wall which, together with an increase in thoracoabdominal blood volume ($V_{TAB}$), contributed to the decrease in functional residual capacity (FRC). The waning of support for the hypothesis of closing volume—as a cause of the gas exchange abnormality during anaesthesia—left considerable uncertainty about the causal relationship between a decrease in FRC and the increased abnormality of gas exchange (Rehder et al., 1977). Despite the fact that a reduction in lung volume during anaesthesia is associated with an increased abnormality of gas exchange, increasing lung volume does not reverse this abnormality (Heneghan, Bergman and Jones, 1984). On this basis, is a reduction in lung volume relevant to impaired gas exchange during anaesthesia, or is it a coincidental finding?

This editorial reviews some of the evidence that changes in the tone and movement of the chest wall following the induction of anaesthesia have an effect on lung volume; that an increase in thoracoabdominal blood volume is an important cause of the reduction in FRC and the increased abnormality of gas exchange (Rehder et al., 1977). Despite the fact that a reduction in lung volume during anaesthesia is associated with an increased abnormality of gas exchange, increasing lung volume does not reverse this abnormality (Heneghan, Bergman and Jones, 1984). On this basis, is a reduction in lung volume relevant to impaired gas exchange during anaesthesia, or is it a coincidental finding?

Little information was available about the effect of anaesthesia on diaphragm tone in man until Muller and colleagues (1979) measured the EMG as an index of tone in the diaphragm. During halothane anaesthesia they showed a significant reduction in diaphragm tone and postulated that this caused the reduction in FRC during anaesthesia. This mechanism supported the widely quoted report of Froese and Bryan (1974) that there was a cephalad shift of the diaphragm during general anaesthesia. The tone of the intercostal muscles is even more sensitive than that of the diaphragm to the depressant effects of volatile anaesthetics (Jones, 1977). This would suggest that anaesthesia, by reducing the tone of the diaphragm and intercostals, causes a reduction of lung volume as a result of an inward movement of the chest wall. However, it would not be expected that the reduction of muscle tone with general anaesthesia would, by itself, be as great as that when combined with neuromuscular blockade. A surprising finding, therefore, has been the similarity, both in terms of a reduction of FRC and worsening of gas exchange, of the effects of general anaesthesia compared with combined anaesthesia with neuromuscular blockade (Hewlett et al., 1974a, b). Is there some other explanation for the similar effects of two quite different anaesthetic techniques on FRC?

An attempt to partition the reduction of FRC during halothane anaesthesia into rib cage and diaphragm components (Jones et al., 1979) led to the surprising finding that no change occurred in end-expiratory position of either rib cage or abdomen, and it was postulated that any reduction of FRC must be caused by an increase in thoracoabdominal blood volume ($V_{TAB}$). Other workers also found little or no change in chest wall volume when blood moved in or out of the thoracoabdominal cavity (Vellody et al., 1978; Hedenstierna, Lofstrom and Lundh, 1981; Kimball et al., 1985). However, Sjostrand (1952) had shown that, following the release of blood trapped in the legs by tourniquets, there was an increase in chest wall volume ($V_{CW}$) and a decrease in lung volume ($V_L$). Theoretically, if there is an increase in $V_{TAB}$ then it would be expected that a reduction in lung volume or an increase in chest wall volume,
or both, might ensue. These changes are summarized as follows:

$$\Delta V_{\text{TA}} = -\Delta V_L + \Delta V_{\text{RC}} + \Delta V_{\text{AB}}$$

where the chest wall volume is the sum of rib cage volume ($V_{\text{RC}}$) and abdominal volume ($V_{\text{AB}}$). From the compliance of each of the three components it would be expected that an increase in $V_{\text{TA}}$ of 1 litre would decrease $V_L$ by 600 ml and increase $V_{\text{CW}}$ by 400 ml. However, Gilroy and co-workers (1985) showed that the actual changes in $V_h$ and $C_w$ were the reverse of those predicted. Kimball, Kelly and Mead (1986) produced an acute increase in $V_{\text{TAB}}$ by inflating leg splints and showed similar changes with protection of lung volume. However, these changes are the opposite of those seen during anaesthesia where lung volume is not well protected. A partial explanation was proposed by Kimball, Kelly and Mead (1986) who noticed that, if the increase in $V_{\text{TA}}$ was maintained, there was a gradual reduction in the volume of the chest wall to control values, as a result of changes in chest wall muscle tone (Freund, Roos and Dodd, 1964; Muller et al., 1979), increase in lung recoil (Gelb, Southorn and Rehder, 1981), or airway closure (Jones, 1982); these may be the factors that explain the different effects of anaesthesia on the maintenance of lung volume.

That muscle tone is probably of particular importance was suggested by Mankikian and associates (1986) who showed that ketamine anaesthesia was associated with an increase in RC movement (as did Morel, Forster and Gemperle (1986)), no change in lung volume, but an increase in chest wall volume—which implies that lung volume was maintained by increasing chest wall volume. Bickler, Dueck and Prutow (1987), using methohexitone anaesthesia, showed no impairment of RC movement and no change in FRC with mask breathing, but a significant reduction in FRC following intubation and coughing.

More sophisticated techniques are now available for determining the relative contribution of $\Delta V_{\text{TA}}$, $\Delta V_{\text{RC}}$ and $\Delta V_{\text{AB}}$ (diaphragm) to changes in FRC during anaesthesia (Kraey et al., 1987). Dynamic spatial reconstruction of the thorax is achieved before and during anaesthesia using high-speed x-ray computed tomography. The internal volume of the thorax is derived and the difference between thoracic volume and FRC gives tissue volume, the latter including blood volume. Anaesthesia with neuromuscular blockade reduced FRC by about 600 ml, but increased thoracic blood volume 300 ml. This contrasts with the findings of Hedenstierna and co-workers (1985) who found a reduction of thoracic blood volume of about 300 ml, but an increase in abdominal blood volume of 400 ml. Krayer and colleagues (1987) showed large individual differences in the effects of anaesthesia on $\Delta V_{\text{RC}}$ and $\Delta V_{\text{AB}}$ in their subjects, although the most consistent change was a reduction of $V_{\text{RC}}$. A cephalad volume shift of the diaphragm occurred in only four of eight subjects, which conflicted with the predictions of Froese and Bryan (1974).

Logan and co-workers (1987) have used stereogrammetric analysis to examine changes in the chest wall before and during thiopentone anaesthesia. Trunk volume increased in four and decreased in three subjects. There was no consistent increase or decrease in the rib cage or abdominal components, although patients with greater than normal weight tended to show a reduction in RC volume and the opposite change occurred in those with less than predicted weight. A larger reduction in lung volume during anaesthesia in obese patients was also reported by Lehane, Jordan and Jones (1980).

These results suggest that anaesthesia with volatile agents, with or without neuromuscular blockade, reduces FRC by increasing $V_{\text{TA}}$ with a variable effect on the rib cage and diaphragm because of reduced ventilatory muscle tone. Anaesthesia with ketamine or methohexitone may preserve the tone of the chest wall musculature and protect against a change in FRC following any change in $V_{\text{TA}}$.

Thus a change in $V_{\text{TA}}$ is a determinant of FRC during anaesthesia and in anaesthetized, paralyzed, ventilated patients the addition of halothane or enflurane caused an increase in lung volume in some subjects (Lehane, Jordan and Jones, 1980). Watney, Jordan and Hall (1987), in this issue, used a similar technique in ponies and showed that different volatile anaesthetics may have different effects on lung volume which may be attributable to effects on vascular capacitance. The major
blood reservoir of the body lies in the abdominal vascular bed more than the limb vessels, and this can take up or release, actively and passively, the major part of any change in circulatory blood volume (Hainsworth, 1984). In contrast, the pulmonary blood volume may change by little more than 300 ml in man, the change occurring passively as a consequence of changes in extrapulmonary vascular beds. Studies of the effects of anaesthetic agents on splanchnic vasculature have been reported (Henriksson et al., 1985), and others reviewed by Arndt (1986), but detailed information on the effects of different anaesthetics on splanchnic capacitance in man is incomplete. The technique described by Lehane, Jordan and Jones (1980) and Watney, Jordan and Hall (1987) provides one approach to this problem which is relevant to the major fluid shifts taking place in anaesthesia, but which are largely uninvestigated.

This leaves for discussion the mechanism whereby a reduction in FRC causes an impairment of gas exchange. The forced airflow oscillation method for measuring airway resistance produces a graphic demonstration of the progressive reduction in airway calibre with diminishing lung volume (Jones et al., 1987) and raises the question of the likelihood of atelectasis during anaesthesia. Atelectasis while breathing at low lung volume was first demonstrated in fighter pilots exposed to high acceleration forces (Green and Burgess, 1962), and the first demonstration of impaired gas exchange when breathing at low lung volume was by Nunn, Bergman and Coleman (1965). Until recently, however, there was no direct evidence that the reduction of FRC during anaesthesia caused atelectasis. It had been assumed that the mechanism causing an abnormal gas exchange was a reduction in lung volume below the closing volume point of dependent airways. Heneghan, Bergman and Jones (1984) were unable to reverse the abnormal gas exchange induced by anaesthesia by increasing the FRC to preoperative values and postulated that collapse of the dependent lung had occurred, but that passive distension of the lung would be insufficient to expand the collapsed units. Subsequently, they showed that, when the same increase in lung volume was achieved in two ways, either by application of PEEP, or by phrenic nerve stimulation, the latter manoeuvre, by producing a greater degree of movement of the dependent part of the diaphragm, produced a significantly greater improvement in gas exchange (Heneghan and Jones, 1985).

However, the definitive proof of atelectasis occurring during anaesthesia was obtained using CT scanning of the lungs of anaesthetized patients and was presented by Hedenstierna and colleagues (Brismar et al., 1985; Hedenstierna et al., 1986; Tokics et al., 1987) in a series of brilliant papers which demonstrated the following:

1. Dependent lung atelectasis developing during anaesthesia.
2. The gas exchange abnormality could be explained entirely by the magnitude of atelectasis.
3. Atelectasis persisted into the postoperative period, in some patients for at least 24 h.
4. Atelectasis was only partially reversed by lung inflation.

A correlation between the magnitude of anatomical shunt and degree of lung collapse has previously been described in spontaneous pneumothorax (Norris, Jones and Bishop, 1968). It might be expected that active pulmonary vasoconstriction would reduce the magnitude of the gas exchange abnormality in man (Norris, Jones and Bishop, 1968); however, this effect may only partially compensate for reduced ventilation or be reduced by the concomitant administration of anaesthetics (Benumoff, 1986).

Atelectasis in the dependent lung during anaesthesia appears to be ubiquitous. Are there techniques which can reduce this effect? It has been shown clearly that adding nitrogen to the inspired gas during anaesthesia reduces the magnitude of atelectasis (Browne et al., 1970), but no apparent benefit on gas exchange has been demonstrated (Webb and Nunn, 1967). However, in the majority of patients the effect of atelectasis on PaO₂ appears to be relatively small and may have a greater significance for postoperative oxygenation rather than during anaesthesia itself (Jones et al., 1985). Nevertheless, it is standard anaesthetic practice, certainly in the U.K., to administer at least 30% oxygen to counter this effect. The development and increasing application of pulsed oxygen saturation monitoring during anaesthesia suggests that the advantages of the routine administration of 30% oxygen may not provide as great a margin of safety as that obtained by breathing 21% oxygen, but with continuous
monitoring of oxygen saturation. In this way impaired oxygen saturation can be immediately detected, underlying causes identified and appropriate treatment instituted. Is it only a matter of cost which prevents such a simple but essential device from being used to monitor every general anaesthetic?

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REFERENCES


