RIB CAGE CONTRIBUTION TO RESTING AND CARBON DIOXIDE STIMULATED VENTILATION DURING 1 MAC ISOFLURANE ANAESTHESIA

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SUMMARY

Using respiratory inductive plethysmography, we have measured rib cage and abdominal motion during isoflurane anaesthesia in 16 healthy day-surgery patients. Anaesthesia was induced with propofol and maintained with 1 MAC isoflurane in air-oxygen via a laryngeal mask. Measurements were taken during both resting ventilation and hyperpnoea induced by rebreathing carbon dioxide. For resting ventilation, the rib cage contributed a mean (SD) of 33 (15) % of the total ventilation whilst awake, and 39 (12) % during anaesthesia (ns). With increasing end-tidal carbon dioxide whilst awake, the subjects showed a mean increase in the percentage rib cage contribution of 7.1 (12.5)%/kPa of carbon dioxide. With isoflurane anaesthesia, there was significant depression of this rib cage recruitment with the mean contribution decreasing by 3.6 (7.4) % kPa⁻¹ (P < 0.05). These results indicate that 1 MAC of isoflurane does not selectively depress rib cage motion, except during carbon dioxide stimulated hyperpnoea.

KEY WORDS

A reduction in the motion of the rib cage during general anaesthesia was first described by Snow in 1858, and reported to indicate that “a little more chloroform had been inhaled than was necessary” [1]. Previous authors have described selective depression of intercostal muscle activity as contributing both to depressed ventilation [2] and to the reduction in lung volume [3].

Over the past 20 years, techniques have been developed to measure rib cage and abdominal motion, first by antero-posterior magnetometers, then circumferential strain gauges, and more recently by measurement of cross-sectional area using respiratory inductive plethysmography (RIP). Both magnetometers [2] and strain gauges [3] have been used to measure rib cage motion during halothane anaesthesia with a tracheal tube; in both studies, general anaesthesia reduced the percentage rib cage contribution to ventilation to approximately 50% of the awake value. However, other studies with non-inhalation agents have failed to show any reduction in rib cage contribution [4—7], although only one of these used RIP [4].

The rib cage component of the ventilatory response to carbon dioxide during anaesthesia has also been studied previously. With halothane anaesthesia and magnetometers [2], the overall reduction in carbon dioxide response was ascribed almost entirely to depression of rib cage motion. This is in contrast with a study of barbiturate anaesthesia using RIP, in which there was only a modest reduction in rib cage contribution [4].

There are three possible explanations for this difference between studies of inhalation and i.v. anaesthesia. First, selective depression of the rib cage component of ventilation may be unique to inhalation anaesthetics. Second, the depth of anaesthesia is unlikely to be equivalent between studies of halothane and, for example, methohexitone. Finally, Bickler's recent study of methohexitone anaesthesia [4] is the only study so far to
have used RIP, and so the discrepancy seen between studies may simply reflect differences of methodology.

We have therefore used RIP to study ventilation during inhalation anaesthesia, and so have eliminated methodological differences, enabling a valid comparison with Bickler's study. Also, by the use of a laryngeal mask airway instead of a tracheal tube, we have been able to perform the study at 1 MAC with a reliable clear airway, thus eliminating the deep anaesthesia seen with previous studies of inhalation agents.

PATIENTS AND METHODS

Subjects

After approval by the local Ethics Committee and with informed patient consent, we studied 16 male day-case surgical patients immediately before surgery. All were ASA I or II, non-obese (body mass index < 29), and had no history of respiratory disease. Before the study, forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV₁) were measured; all values were greater than 80% of values predicted for height and age. ECG, heart rate, end-tidal carbon dioxide concentration and oxygen saturation were monitored throughout the study, and no adverse effects were seen.

Apparatus

The apparatus used for the study is shown in figure 1. This consisted of a circle system with a non-rebreathing valve at the mouthpiece, connected to a dry rolling-seal spirometer (AirCo Ohio 840), which was calibrated before each subject using a 1.5-litre gas tight syringe. The spirometer gave an electrical volume signal displayed on a chart recorder, from which tidal volume and ventilatory frequency were calculated. A soda-lime canister could be connected to the expiratory limb of the circle for resting ventilation measurements. A separate circuit sampled gas from the mouthpiece and back to the system through a multiple gas analyser (Datex Capnomac) which was calibrated before each subject using gas mixtures supplied by the manufacturer (48.7 ± 0.5% oxygen, 4.55 ± 0.2% carbon dioxide, 2.8 ± 0.05% enflurane). The gas analyser displayed inspired oxygen concentration, end-tidal carbon dioxide concentration, and inspired/end-tidal isoflurane concentrations. Taps 1 and 2 enabled the subject to breathe air whilst the spirometer was filled or emptied via tap 3 and the gas inlet.

Read's rebreathing method was used to assess the ventilatory response to carbon dioxide [8]. The spirometer was filled to a volume 2 litre more than the subject's vital capacity with 50% oxygen and 7% carbon dioxide in nitrogen. Tap 1 was closed to connect him to the inspiratory side of the circuit, allowing the 2 litre of extra gas in the spirometer to flush the deadspace of the subject and circuit, before tap 2 was switched to complete the circle. Ventilation and $P_{E'CO_2}$ were recorded for 4 min, or until the subject terminated the study because of dyspnoea.

Subjects had their rib cage and abdomen contributions to ventilation measured continuously using respiratory inductive plethysmo-
Many methods of calibrating RIP have been described, most aimed at measuring tidal volume from the RIP signals without any special manoeuvre being required by the subject. The isovolume technique is a simple method, which requires some coaching of the subject, but is as accurate for measuring rib cage and abdomen contributions to ventilation as the other more complex techniques [9]. The calibration was performed at functional residual capacity with a large isovolume manoeuvre, and the subject remained in the same position throughout the study (including during anaesthesia). Calibration during anaesthesia was not attempted as obtaining an isovolume line by obstructing the airway invalidates the manoeuvre by causing distortion of the rib cage and abdomen compartments. The rib cage and abdomen signals, with analogue signals of carbon dioxide concentration and spirometer volume, were charted on a four-channel chart recorder for later analysis.

**Anaesthesia**

Subjects received no premedication. Anaesthesia was induced with propofol 3–4 mg kg\(^{-1}\) via an indwelling cannula on the dorsum of the hand, and maintained with isoflurane in an air–oxygen mixture (\(F_{\text{IO}_2} = 0.5\)) with spontaneous ventilation via a laryngeal mask and Mapleson A breathing system. Overpressure was used to achieve an end-tidal concentration of 1.1–1.2% (1 MAC) as quickly as possible, and this was maintained for 10 min to allow equilibration. Anaesthesia was achieved with the patient connected to the spirometer, by addition of 0.1-ml aliquots of liquid isoflurane to the expiratory limb of the circle. By this means, end-tidal isoflurane concentration was maintained within the limits 1.0–1.2%.

### Procedure

The study was performed in the anaesthetic room with subjects in the supine position with the lumbar spine supported by small pillows to minimize spinal movement. Throughout the awake period of the study, patients listened to music through headphones.

The sequence was as follows:

1. Calibration of the RIP by the isovolume manoeuvre.
2. “Run-in” period with no mouthpiece until regular ventilation returned (as judged by the RIP signal).
3. Recording of RIP signals for 1 min with no mouthpiece, allowing the subject to breathe via his natural airway.
4. Subject connected to spirometer (including soda-lime) with a mouthpiece/noseclip and a
further run-in period allowed with the subject breathing 50% oxygen.
(5) Resting ventilation and end-tidal carbon dioxide concentration recorded.
(6) Read’s rebreathing carbon dioxide response performed.
(7) Subject allowed to recover from the hyperpnoea for 5–10 min.
(8) Anaesthesia induced and stabilized as above.
(9) Rib cage contribution to non-stimulated ventilation recorded with the patient attached to a Mapleson A breathing system.
(10) Non-stimulated ventilation measured with the spirometer circuit connected directly to the laryngeal mask.
(11) Read’s rebreathing carbon dioxide response repeated.

Data processing
The rebreathing runs were divided into six to eight periods (each lasting 30 s), depending on the length of the run. For each period (including the resting ventilation) the ventilatory frequency, tidal volume ($V_t$) at ATPS, minute volume ($V_e$) at ATPS, end-tidal carbon dioxide concentration, and percentage rib cage contribution ($\%RC$) to ventilation was calculated. All volumes were converted to BTPS using measured room temperature and assuming the body temperature of all subjects was 37 °C.

For the carbon dioxide ventilation response, $V_e$ (litre min$^{-1}$) was plotted against $P\bar{e}'_{CO_2}$ (kPa). Using least squares linear regression, straight lines were fitted to all the data for both awake and anaesthetized carbon dioxide responses in each subject. The mean slope for all subjects was determined and a line of this slope drawn through the grand mean point. The line was plotted between points on the $X$-axis representing the mean compartmental minute volume and $P\bar{e}'_{CO_2}$ at the beginning and end of the rebreathing runs. From data provided in Tusiewicz’s study, we have plotted also the mean compartmental ventilation lines for her subjects between arbitrary points of 6.5 and 8 kPa on the $X$-axis.

(2) The tidal volumes were normalized for body weight and expressed as ml kg$^{-1}$, as in the study of Bickler, Dueck and Prutow [4]. These data were converted to rib cage and abdomen components and lines plotted as for (1). Similarly, we have plotted equivalent compartmental response lines for Bickler’s subjects alongside ours, between 6.5 and 8.5 kPa.

Comparisons between awake and anaesthetized data were by paired Student’s $t$ test, except for the ventilatory response slopes. These data have been described previously as differing from a normal distribution [10], and our data did so significantly (by Shapiro–Francis $W'$ test), so were compared using Wilcoxon’s signed rank test.

RESULTS
One subject was excluded from the analysis because of an abnormal RIP trace during anaesthesia, showing a phase difference between the rib cage and abdomen signals. This subject is discussed below, and in all other subjects the peaks of the RIP signals were in phase.

The remaining 15 patients had mean age 28.2 yr, body mass index 23.7, FVC 4.3 litre and FEV$\textsubscript{1}$ 3.9 litre (table I). Mean (sd) resting ventilation awake was 8.1 (2.2) litre min$^{-1}$ at $P\bar{e}'_{CO_2}$ 5.39 (0.36) kPa and, after maintenance of 1 MAC of isoflurane for 10 min, 6.0 (1.1) litre min$^{-1}$ with $P\bar{e}'_{CO_2}$ 6.29 (0.75) kPa (fig. 2). The mean $\%RC$ contribution to quiet breathing awake was 33 (15) % with a large scatter range (range 16–57 %); during anaesthesia the mean was 39 (12) % (range 22–55 %) (ns) (fig. 3).

The ventilatory response to carbon dioxide was significantly depressed during anaesthesia (fig. 2), the mean slope decreasing from 14.2 litre min$^{-1}$ kPa$^{-1}$ awake to 3.6 litre min$^{-1}$ kPa$^{-1}$ during anaesthesia ($P < 0.001$). The intercepts of the response lines were also decreased significantly, from a mean of 5.8 kPa awake to 4.8 kPa anaesthetized ($P < 0.02$).

Despite a wide scatter, the mean (sd) slopes of
**Fig. 2.** Mean (sd) carbon dioxide response lines (● = awake; ▲ = isoflurane 1 MAC) and resting ventilation (○ = awake; △ = isoflurane 1 MAC).

**Fig. 3.** Mean (sd) rib cage contribution (RC%) to resting (open symbols) and carbon dioxide stimulated ventilation. ●, ○ = Awake; ▲, △ = isoflurane 1 MAC.

$\Delta \% \text{RC}/\Delta P_{\text{ETC0}_2}$ (fig. 3) decreased significantly, from 7.1 (12.5)% kPa$^{-1}$ awake to −3.6 (7.4)% kPa$^{-1}$ during anaesthesia ($P < 0.05$). The slopes for awake subjects differed significantly from zero ($P < 0.05$), but slopes from anaesthetized subjects did not.
DISCUSSION

The anaesthetic technique used for this study was designed to reflect modern day-case clinical practice whilst providing 1 MAC of anaesthesia with a single volatile agent. In these patients, it is unlikely that the propofol was still contributing appreciably to anaesthesia at the time of the study (15 min after induction) [11]. For any study of respiratory mechanics during anaesthesia, an unobstructed airway is mandatory and a laryngeal mask provides this at lighter planes of anaesthesia than has been possible previously with tracheal intubation.

The subject who was excluded developed an abnormal RIP pattern of non-stimulated ventilation during anaesthesia, despite a clinically clear airway (fig. 4). The abdomen motion appears normal and predominant, but causes indrawing of the upper rib cage during inspiration, presumably representing abolished intercostal muscle tone. During expiration, the rib cage rapidly returns to normal, and even expands slightly. This may result either from activity of the intercostal/accessory muscles during expiration or, more likely, from active expiration by the abdominal muscles passively expanding the rib cage. This pattern has been described in other studies (see below), but occurred in only one of our 16 subjects.

Non-stimulated ventilation

We have found no significant change in the %RC contribution to non-stimulated ventilation at 1 MAC of isoflurane anaesthesia. Tusiewicz, Bryan and Froese [2] and Jones and colleagues [3] both reported profound depression of the rib cage contribution to spontaneous ventilation during halothane anaesthesia. There are several possible explanations for this conflict with our results:

Methodology. Our study measured cross-sectional areas rather than circumference or antero-posterior diameter. These methods agree well in physiological studies [12], although in the study by Jones and colleagues [3] (using circumference measurements) the resting %RC of 14% is less than 50% that in most other studies of supine subjects. Methodology of rib cage-abdomen partitioning may therefore have contributed to differences in results between these studies, particularly that of Jones’ group.

All body surface measurements of rib cage and abdomen do not necessarily reflect activity of the
intercostal and diaphragmatic muscles. For example, contraction of the diaphragm may cause expansion of the lower rib cage and activity of abdominal wall muscles will alter all dimensions of the abdominal cavity. Studies of the EMG of these muscle groups have been performed [13,14], but we know of no study which has related EMG activity to body surface motion. Without EMG it is impossible to assess the activity of expiratory muscles in our subjects, or its subsequent effect on upper rib cage motion.

Airway. Preliminary studies in awake subjects indicated that the resistance to breathing between laryngeal masks and tracheal tubes is similar [15]. It is possible that differences in airway resistance, despite a clinically adequate airway, may result in various degrees of resistive loading and so alter %RC. This is clearly impossible to elucidate without measuring airway resistance through a laryngeal mask during anaesthesia, but the magnitude of the change in resistance in patients with adequate airways is unlikely to explain our findings. A study of methohexitone anaesthesia used two groups, with either intubation or facemask for airway maintenance. Despite a finding of different functional residual capacity with the differing airways, no comment was made on the %RC between the two groups [4]. It has been suggested that a tracheal tube may stimulate receptors in the airway, causing a reflex change in pulmonary mechanics, for instance bronchoconstriction [16], although it is not known if this reflex alters chest wall motion.

Use of suxamethonium. In the studies by Tusiewicz, Bryan and Froese [2] and Jones and colleagues [3], the majority of subjects were given suxamethonium to facilitate tracheal intubation. Their results may therefore represent a selective reduction in intercostal activity after suxamethonium, although this seems unlikely as 90 min elapsed between induction and study in Tusiewicz’s subjects.

Specific respiratory effects of different anaesthetic agents. Respiratory depression at 1 MAC of isoflurane is well known [17]; our subjects' resting minute volumes decreased by 2 litre min⁻¹ with a consequent increase in $Pr\cdot CO_2$ of 0.9 kPa. The size of $VT$ has been shown to affect %RC. Voluntarily increasing $VT$ in awake supine volunteers increased the %RC in the majority of subjects [18], but we know of no studies of involuntary changes in $VT$ or the effects of decreasing $VT$. As increasing $VT$ results in increased %RC, it is likely that decreasing $VT$ would reduce the %RC, which is the opposite of our findings. Also, taking potency into account, isoflurane does not differ greatly from halothane in its ventilatory effects, causing a similar reduction in $VT$ with less increase in ventilatory frequency [19]. Changes in $VT$ are therefore unlikely to account for the difference in our observations from those with halothane [2,3].

There are other circumstances during anaesthesia in which %RC has been maintained or even increased. It is agreed that paralysis and IPPV cause an increase in rib cage contribution, regardless of the other agents used [3,20], so only spontaneous ventilation is discussed further. Methohexitone bolus followed by infusion caused no change in %RC [4], whilst a similar regimen of ketamine resulted in a significant increase in rib cage contribution [5]. Sedation with either midazolam or diazepam also increased rib cage contribution to ventilation, by 15–20% [6,7]. Pethidine administered during midazolam sedation also caused an increase in %RC, this effect being antagonized by naloxone [6]. The benzodiazepines and volatile agents used in all these studies cause similar degrees of overall respiratory depression, reducing $VT$ to approximately 40% of control so, as discussed above, the varied effects on %RC cannot be ascribed simply to changes in $VT$.

It is clear, therefore, that different agents and doses do have variable effects on the rib cage and abdomen components of the respiratory system. However, it remains unclear as to how this occurs, and where the effect is mediated between the medullary respiratory centre and the muscle fibre.

Depth of anaesthesia. Our study was undertaken at a lighter level of anaesthesia than those of Tusiewicz, Bryan and Froese [2] or Jones and colleagues [3]. Tusiewicz’s study was performed at 1.4 MAC of halothane in oxygen, whilst Jones’ study used between 0.83 and 3.3 MAC halothane in addition to nitrous oxide. Presumably this involved at least 50% nitrous oxide, providing a minimum total of 1.33 MAC. Jones’ group also demonstrated that progressive rib cage depression occurred with increasing anaesthetic concentration. A previous preliminary report of a study of isoflurane and rib cage motion showed no significant change in rib cage contribution up to
1.5 MAC, although the %RC did decrease by 15% at this value [21]. This study also demonstrated that airway obstruction during anaesthesia caused instability of the lower rib cage. We saw no evidence of this in the high rib cage recordings in our study, except in the excluded subject who showed similar rib cage instability at nipple level with a clear airway. It seems very likely that the most important difference between our findings and those of Tusiewicz, Jones and their colleagues is the greater depth of anaesthesia used to allow tolerance of the tracheal tube during inhalation anaesthesia with spontaneous breathing—a problem we have avoided by use of a laryngeal mask.

**Carbon dioxide stimulated ventilation**

Slopes of ventilatory response to carbon dioxide were reduced to about 25% of the awake value (fig. 2), which is similar to results in other studies [17, 19]. We have found a small, but significant, increase of 7.1% kPa⁻¹ in the rib cage contribution to ventilation during carbon dioxide rebreathing whilst awake. This rib cage “recruitment” has been demonstrated previously in conscious subjects, and is particularly marked in subjects who have large increases in tidal volume [22]. In the present study, this rib cage recruitment was abolished by anaesthesia, which at 1 MAC resulted in a negative Δ%RC/ΔPFe CO₂ slope not significantly different from zero (fig. 3). Absolute rib cage ventilation continued to increase with PCO₂ but the slope of the rib cage component of minute volume was greatly reduced (fig. 5). Tusiewicz, Bryan and Froese [2] previously demonstrated profoundly decreased rib cage motion during carbon dioxide rebreathing at 1.4 MAC halothane anaesthesia, and concluded that a major part of the ventilatory depression seen with halothane resulted from suppression of intercostal muscle activity. However, these observations were illustrated (in their fig. 5) by total abolition of rib cage response to carbon dioxide in one subject, who was aged only 9 yr and not necessarily representative of the general adult population. From our figure 5, it can be seen that even when our results are processed in the same way, and using the mean of Tusiewicz's five subjects, there is more pronounced depression of rib cage contribution in those subjects than ours. We believe this again results from deeper anaesthesia in the subjects studied by Tusiewicz's
group. Our results for compartmental tidal volumes are very similar to those of Bickler (methohexitone anaesthesia), our subjects displaying, if anything, greater depression of the rib cage (fig. 6). Equivalence of the depth of anaesthesia between inhalation and i.v. agents is difficult to assess, and it is possible that Bickler did not achieve a depth of anaesthesia equivalent to 1 MAC as in our study.

In conclusion, we have shown that at 1 MAC of isoflurane, selective depression of rib cage motion is undetectable without the severe provocation of carbon dioxide rebreathing. There is at present no evidence that any currently used anaesthetic agent other than halothane can cause significant depression of rib cage motion during ventilation without carbon dioxide stimulation. Thus most modern inhalation anaesthetic agents at 1 MAC should not compromise patients with poor ventilatory muscle function, even when breathing spontaneously. Compared with the awake state, anaesthesia causes a reduction in the relative contribution of the rib cage to carbon dioxide stimulated ventilation, but both rib cage and abdomen compartments continue to contribute, although at a reduced level, and depression of the overall ventilatory response to carbon dioxide cannot be ascribed solely to the rib cage.

REFERENCES
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