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Reduction of Tonic Ribcage Muscle Activity by Anesthesia with Thiopental

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Surface electromyograms were recorded from the scalene, sternomastoid, and parasternal intercostal muscles in 16 supine premedicated adult patients before, during, and after the induction of anesthesia with thiopental and after the administration of succinylcholine. Muscle activity was measured as the rectified, integrated signal, and expressed as a percentage of the activity detected during a maximal inspiration. Activity was found in the scalene and sternomastoid muscles of all patients, and in the intercostal muscles of 13. This activity was more commonly tonic, and, at end-expiration, it was 7%, 6%, and 5% of the activity that could be generated by a maximal inspiration, for the scalene, sternomastoid, and intercostal muscles, respectively. In all subjects, this activity was markedly reduced or abolished by the administration of thiopental, to 0.7%, 0.6%, and 0.6%, respectively. There was no tonic activity in these muscles during spontaneous respiration after induction of anesthesia. It is suggested that the reduction of tonic inspiratory activity in these muscles may contribute to the reduction in functional residual capacity associated with anesthesia. (Key words: Anesthetics, intravenous: thiopental. Lung: functional residual capacity. Respiration: muscles.)

IN MOST STUDIES in humans, it has been shown that general anesthesia is associated with a decrease in lung volume at end-expiration (FRC),[1] which occurs about 15–45 s after loss of consciousness.[2] This reduction in lung volume could be the result of relaxation of the ribcage muscles, or of the diaphragm, or the accumulation of blood within the thorax. In two volunteers, Froese and Bryan[3] demonstrated that the dome of the diaphragm moved in a cranial direction after induction of anesthesia, and Muller *et al.*[4] showed that tonic activity in the diaphragm was abolished by halothane anesthesia. However, activity in the diaphragm at the end of expiration in supine conscious subjects has not been shown by others,[5] and recent studies with ultrasound suggest that movement of the diaphragm associated with induction of anesthesia is small and does not always occur.[6]

Relaxation of inspiratory muscles inserted into the ribcage could be responsible for part of the decrease in FRC that occurs with anesthesia. Abolition of activity in the intercostal muscles in conscious subjects using small doses of muscle relaxants is associated with a reduction in FRC,[7] and the scalene muscles, which were previously often considered to be only accessory muscles of inspiration, have been recently shown to be active during normal breathing.[5,8]

The present study was designed to measure the activity of some of the upper ribcage inspiratory muscles, in patients about to undergo anesthesia, and assess the change in activity associated with the loss of consciousness, which was the time at which Bergman[2] noted that FRC was reduced.

Materials and Methods

The experiment was approved by the anesthetic section of the Area Ethical Committee. Twenty patients (seven male and 13 female, mean (SD) age 53 ± 11 yr, height 163 ± 10 cm, weight 70 ± 10 kg) were studied, selected from patients scheduled for elective surgery, with no clinical evidence of cardiac, respiratory, or neuromuscular disease, and not at risk from regurgitation. The patients were told that the study was to examine the effects of anesthesia on some of the muscles of the body, but not that there was a particular interest in the activity of these muscles before the induction of anesthesia. Verbal consent was obtained the day before the study.

Each patient received premedication with temazepam, 20 mg by mouth, 1 h before the study. The study was carried out in a quiet, warm room. An intravenous infusion of lactated Ringer's solution was established, and an extension tube was attached so that anesthesia could be started without disturbing the patient. After the electrodes (see below) had been attached, the patient was encouraged to relax while in the supine position, with the head in the midline resting on a soft pillow, and the arms supported at the sides. The recording apparatus was kept out of sight of the patient, so that there was no opportunity for visual feedback from the EMG display.

The patient was asked to rotate the head against restraint, so that the sternomastoid muscle was palpable, and the scalene muscle mass could be felt as a separate mass in the posterior triangle of the neck. The skin was prepared with abrasive electode jelly and acetone. Two disposable pregelled silver/silver chloride electrodes (Medicotest R-OO-S) with a gel diameter of 1 cm, were applied with centers 5 cm apart, over each muscle. For

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the sternomastoid, the lower electrode was placed at the division of the clavicular and sternal heads, and the scalene electrodes were placed over the mass of the scalenus medius at the same level. The intercostal electrodes were placed in the second intercostal space, immediately lateral to the sternum. A single reference connection was used over the clavicle, which was also used as a reference electrode for the EKG. EMG signals were recorded through short 5-cm leads connected to a buffer amplifier (NL101) and amplified with a matching differential amplifier (NL104, Digitimer Ltd.). The signals were passed through a bandpass filter (NL125) set to 10–5000 Hz and recorded, along with the EKG and a commentary channel, on an FM tape recorder (Digitimer D146) for subsequent analysis. After the amplifier gains had been adjusted to obtain satisfactory signal amplitudes, each patient was encouraged to take two maximal inspirations. The amplifier gains were then increased to measure the spontaneous resting activity and the patient was allowed to relax for at least 5 min, before anesthesia was induced with an injection of thiopental, 3.5–4.5 mg/kg given over 25–30 s. After loss of consciousness, the recording was continued for about 30 s, and oxygen was then administered, followed by succinylcholine 1.5 mg/kg iv to facilitate intubation. The recording was continued so that a "zero activity" signal after succinylcholine could be obtained. In five patients, the administration of succinlycholine was delayed for 5 min so that a period of spontaneous respiration during anesthesia could be recorded.

The signals were analysed by replay through a delay unit and gating device triggered by the non-delayed EKG recording, so that the EKG artifact on the EMG signal could be gated out, similar to the device used by Muller *et al.*[4] The signal was then passed through another 5–5,000 Hz bandpass filter, rectified, integrated with a "leaky integrator" (Digitimer NL703) with a time constant of 100 ms, and recorded with a UV galvanometer recorder (Bell and Howell 5-137). A calibration signal of 1 V was also passed through the delay system and integrator for calibration purposes. Zero activity was taken as the signal obtained from integration of the recording obtained after succinylcholine administration. Measurements of activity were made over 20 s immediately before thiopental administration, and between 50 and 70 s after administration had started. In those patients in whom activity increased during induction of anesthesia, an indication of this activity was obtained by taking the mean of the two breaths showing the greatest activity. EMG activity was calculated in terms of averaged root mean square voltage, in microvolts, and also as a percentage of the maximal activity of that muscle, during maximal voluntary inspiration. Since both these values showed a markedly skewed distribution pattern, mean values were calculated using the logarithm of the actual values, but the results are converted back to the original values for presentation. Consequently, the SD values given indicate both a lower and upper value. Statistical analysis was done with the Wilcoxon rank test for paired values,[9] using a probability of <0.05 to indicate significance. To assess the results in terms of obesity, patient weight was expressed as a percentage of the average weight for a subject of medium build of the same sex, age, and height.[10]

Results

In three patients, the integrated electrical activity in the intercostal muscles during maximal inspiration was less than 5 microvolts. In a further patient, 50 Hz (line electrical) interference was present in one channel of the recording. These data were not analysed further. The remaining 16 patients (6 males) had a mean (SD) age of 53 ± 11 yr, height 163 ± 10 cm, and their mean weight, expressed as % expected, was 107 ± 16%. Two patients weighed more than 120% of their expected weight.

The mean values (with range of ± one S.D.) of the maximal electrical activity were scalene, 63 (35 − 113); sternomastoid, 70 (28 − 177); and intercostal 24 (12 − 44) microvolts respectively. The electrical activity measureable in the intercostal muscles was significantly less than in the other two muscles ($P < 0.01$).

Before induction of anesthesia, continuous EMG activity was present in the scalene and sternomastoid muscles in all patients. Intercostal muscle activity was not detected in one patient. In most patients, muscle activity was tonic, but, in five of the patients, respiratory modulation was present (*i.e.*, activity during inspiration was at least 25% greater than activity during expiration). The activity present, relative to the activity that could be elicited during a maximal inspiratory effort, varied considerably. In the scalene muscles, the range of activity in expiration was between 1 and 29%, with a mean value of 8%. The mean values, with the SD range, calculated from the log values, are given in table 1.

A typical response to the induction of anesthesia is shown in figure 1. As the patient lost consciousness, there was a transient period of increased activity associated with clinically obvious increased respiratory movement. Phasic activity became apparent in each muscle. Immediately after this, the tonic activity present in expiration became less. This phasic pattern of respiration was seen in nine of the 16 patients. In all patients, the activity in each muscle group became markedly reduced between 30 to 50 s after the start of the injection of thiopental ($P < 0.01$), so that, at 60 s after the injection started, no activity was measureable

in 12 of the subjects' scalene muscles, and 12 of the sternomastoid muscles. In two subjects, residual activity was detected in both muscle groups. Some activity remained in the intercostal muscles of two patients after onset of anesthesia. The mean values of these activities, assuming that no detectable activity could represent 0.5% of maximal, are given in table 1, and the individual values are shown in figure 2.

The lash reflex was abolished in all patients after induction of anesthesia. Transient apnea was noted in ten, but apnea was only present in four of the patients at the time the measurements were made. In the two patients with muscle activity detectable in both sternomastoid and scalene muscles, responses to the application of the facemask and adjusting the head position were noted, and further thiopental was administered.

Five patients were studied during spontaneous respiration for 5 min, first breathing oxygen, and then enflurane in oxygen, before the administration of succinylcholine. In these patients, phasic respiratory activity gradually developed in both scalene and sternomastoid muscles. In three, no activity was present during expiration. However, as the influence of the thiopental wore off, the activity increased and became more tonic. The administration of enflurane, 1.5–2.5% inhaled concentration, abolished activity in all three muscle groups, over about 3 min.

There was no relationship between the degree of obesity (body weight as % average) and the degree of activity (as % maximal effort) in any of the muscle groups, nor in the change in activity noted with induction of anesthesia.

Discussion

Surface electrodes, rather than needle or implanted wire electrodes, were used for two reasons. First, invasive techniques involve discomfort and the risk of pneumothorax, which was not ethically justifiable in a study of this number of patients. Second, electrode insertion causes increased apprehension in the patient, which could increase tonic activity. Premedication was used to reduce apprehension. However, in other patients studied without premedication, the muscle activity noted was similar to the activity found in the present study. In addition, studies of FRC changes have generally used premedicated patients.

However, surface electrodes are prone to two problems. First, the signal size is much less than with intramuscular electrodes, particularly with small muscles, such as the intercostals. Consequently, fine details of activity in the intercostal muscles were difficult to detect. As a result, some patients in whom satisfactory intercostal signals could not be obtained had to be excluded from further analysis, and it is possible that these patients may have had different responses. However, the changes in sternomastoid and scalene activity in these patients were very similar to those seen in the 16 patients whose responses are presented. The size of the

TABLE 1. Mean Activity at End-expiration in the Muscles, Before, During, and After the Induction of Anesthesia, Expressed as Percentages of the Activity during Maximal Inspiratory Attempt

	Scalene	Sternomastoid	Intercostal
Before	8 2–24	6 2–23	6 1–26
During (n = 11)	8 4–20	8 2–31	6 1–34
After	0.7 0–1	0.6 0–1	0.6 0–1

The ranges represent the mean ± SD calculated from the log values. For the purpose of calculating the log values, zero % activity was assumed to be 0.5%.

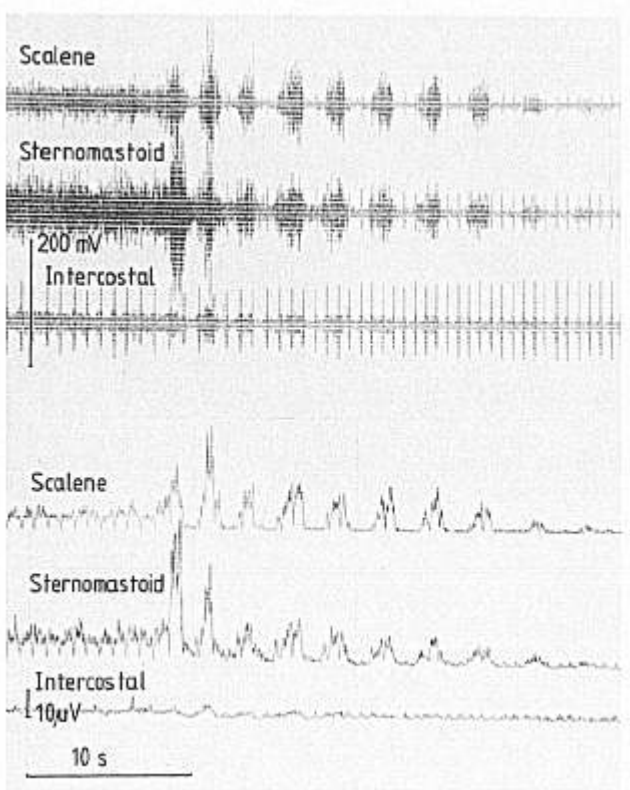

FIG. 1. Direct and integrated EMG recordings showing the response to induction of anesthesia in a typical patient. Thiopental administration commenced at the start of the time marker. There was an increase in the depth of breathing coincident with the loss of consciousness. Tonic activity is followed by a period of phasic activity, which is then lost. Calibration: direct signal, 200 mV, integral, 10 microvolts, time marker 10 s.

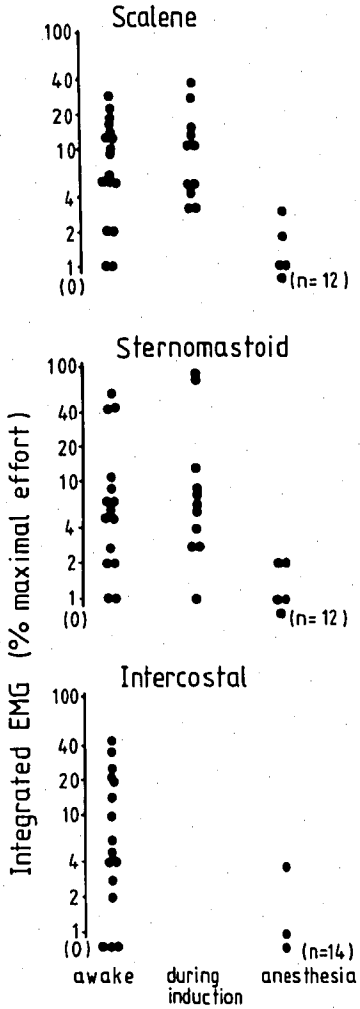

FIG. 2. Mean electrical activity in expiration in the three muscle groups, before, during, and after induction of anesthesia (as % activity during maximal inspiratory effort). The 11 values indicated during induction represent the maximum activity in expiration in those patients in whom this activity increased transiently. The values are plotted on a log scale; no detectable activity is arbitrarily shown as 0.5%. The numbers of these subjects are shown as n.

signal obtained from the sternomastoid muscle, in the relaxed awake state, is similar to, but slightly less than, the signal reported by Benthuysen *et al.*[11] Second, the EMG of interest may be contaminated by signals from nearby muscles. This problem is least with the sternomastoid. Large isolated signals could be obtained by maneuvers such as head lift and rotation against restraint, indicating that the degree of contamination of the scalene signal from the sternomastoid muscles was insignificant, since only small increases in activity in this signal were seen during head lift. The intercostal signal could be contaminated by activity in the pectoralis muscle, which overlies it; however, by supporting the arm, this signal could be abolished.

There are differences, of at least tenfold, between the size of surface EMG potentials obtainable from different individuals. Consequently, the EMG is best compared with a signal obtained in that individual, and, in this study, a maximal inspiration was chosen. This does not necessarily represent the maximal activation of the muscle: for example, greater signals are obtained from the sternomastoid during head lift against restraint. In addition, when inspiration is less than maximal, the degree of activation of different groups of respiratory muscles need not be proportional. However, this maneuver was chosen so that the activities measured would be related to the function of these muscles that was of interest; namely, inspiration. Since the volume inspired was not measured, the effort used by each patient may not have been truly maximal. When the patient was asked to make two successive attempts, it was usually noted that the second attempt was not as good as the first. Consequently, the activity measured in the resting state, when expressed as a percentage of the activity during the maximal effort, may represent an overestimate of the true inspiratory activity present. However, by restricting the analysis of the results to the use of a paired, nonparametric test, valid conclusions may be drawn about activity changes within each patient.

Electrical interference was minimized by careful grounding and screening and the use of a reference electrode. Only one recording could not be analyzed because of interference. The recording after succinylcholine was valuable in allowing an incontrovertible baseline to be established, but interference was rarely a problem, and activity of the order of 1–2% of the reference values could be discriminated. EKG signals can swamp the surface EMG of the intercostal muscle, when this is integrated to allow measurement. For this reason, the EKG was gated out of the signal before integration. This technique leaves "holes" rather than "blips" on the trace (fig. 1), but interpolation by eye is possible.

Since the aim of the study was to investigate the changes caused by anesthesia, over approximately the same timescale as studied by Bergman,[2] a dose of thiopental that could reliably induce anesthesia in the majority of patients was chosen. In two patients, this dose was only just sufficient. In most patients, however, apnea occurred at some time in the period of observation. For this reason, a longer period of spontaneous respiration was studied in five patients so that the EMG pattern could be assessed. In preliminary studies, it was consistently noted that the slow administration of thiopental, in 50-mg increments, resulted in a period of markedly increased EMG activity, before the lash reflex was abolished, and that individual dose requirements were extremely variable. For this reason, the thiopental was given as an undivided dose, arbitrarily adjusted within the chosen range on a clinical basis according to the age and build of the patient.

The principal finding of this study was that, in the awake, relaxed, premedicated patient before anesthesia and surgery, there was tonic, or phasic and tonic activity, in the scalene, sternomastoid, and parasternal inter-

costal muscles during expiration which was abolished by the administration of thiopental, over the same timescale as a change in the resting end-expiratory position has been shown to occur.[2] Previous investigators have shown similar patterns of muscle action in awake subjects.[5] How can this loss of activity be related to our present knowledge of the mechanics of the ribcage, and be related to the possible mechanisms of FRC reduction?

In the sitting human subject, there is good evidence that FRC is influenced by activity of the ribcage muscles. Partial paralysis with nondepolarising muscle relaxants, which abolishes intercostal muscle activity, is associated with a decrease in FRC.[7] Administration of pyridostigmine to patients with myesthenia gravis increases FRC.[12] The compliance of the thorax in the "relaxed" awake subject is greater than after anesthesia and paralysis, suggesting that the ribcage muscles assist passive movements.[13] In quadriplegic patients, those with some intercostal muscle activity have normal lung mechanics and a normal FRC, whereas those who only have diaphragmatic activity have a reduced lung compliance, and their lung volume is about 1 liter less than predicted.[14] This indicates that the upper ribcage muscles have sufficient influence on FRC to be able to account for changes of the order of those seen with the induction of anesthesia. However, the influence of the ribcage muscles on the maintenance of FRC in the supine position has not been investigated.

The scalene and sternomastoid muscles move the upper ribs and sternum in a cranial direction and increase the anteroposterior diameter of the upper rib cage, increasing the ribcage volume.[15,16] Although the action of these muscles cannot be considered in isolation, because the interaction of muscles on the rib cage is complex,[17] it is clear that muscles inserted into the upper ribs can influence the movements of the whole ribcage. In normal subjects, voluntary suppression of scalene muscle action during inspiration not only leads to indrawing of the upper ribcage, but also reduced expansion of the lower ribcage.[8] In patients with high cervical paralysis, use of the cervical accessory muscles increases the anteroposterior diameter of the middle ribcage, whereas electrical stimulation of the diaphragm results in a decrease of this dimension.[16] The degree of shortening of the scalene and sternomastoid muscles is less than that of other respiratory muscles[18] and, hence, even though integrated electrical activity is commonly considered to be proportional to the isometric tension developed by a muscle,[19] it is unwise to estimate the volume contribution from the degree of activation noted in this study. Indeed, volume changes, if they were measured, could not be attributed to any set of muscles unless the activity and mechanical effectiveness of the other respiratory muscles could be proved to be constant. However, despite these provisos, it is of interest that the loss of about 6% of the activity appears to be of the appropriate order of magnitude.[18]

The reduction in FRC has been attributed to loss of tonic diaphragmatic activity.[3,4] However, in a study designed specifically to investigate tonic, as well as phasic, respiratory muscle activity, other workers[5] were unable to demonstrate any activity in the diaphragm at end-expiration in nine awake, supine subjects, despite the use of esophageal electrodes, although there was considerable tonic activity in the ribcage muscles.

In addition, cranial movement of the diaphragm does not necessarily indicate that it has relaxed. Cranial diaphragmatic movement can be the result of loss of ribcage volume, because a major part of the abdominal contents are contained within the lower ribs, enclosed within that part of the diaphragmatic dome that is applied to the inner surface of the lower ribcage. If ribcage volume is reduced, the abdominal contents will be displaced, resulting in displacement of the anterior abdominal wall forwards, or of the diaphragm cranially, without any change in the length of the diaphragm.[20] The relative movement in each direction determines the actual displacement of the dome of the diaphragm. Recent observations[6] suggest that diaphragmatic movement on induction of anesthesia is insufficient to account for the known decrease in FRC. However, in a supine subject with unilateral phrenic nerve palsy, cranial displacement of the diaphragmatic dome on the affected side may be detected radiographically, suggesting that the decrease in FRC in this condition may be because of a difference in activity between the sides, or possibly because the affected side is more liable to passive stretching.

A further factor suggested to reduce FRC is an increase in blood volume within the thorax, but direct evidence for this possibility is lacking.[21] The hypothesis was proposed in studies in which the changes in external dimensions of the ribcage and abdomen did not account for the reduction in FRC that was observed. However, no account was taken of the degree of flexion of the spine, which could increase on induction of anesthesia, and reduce the volume within the lungs, despite constant transverse ribcage and abdominal dimensions.[22]

The findings of the present study, of a loss of inspiratory ribcage tone, are consistent with the results of two recent investigations using computed tomography,[23,24] where induction of anesthesia and paralysis reduced the volume of the ribcage, as would be found after loss of ribcage muscle action. These observations, and those of the present study, are also consistent with the observations that muscle paralysis after induction of anesthesia has no further effect on lung volume.[1,2]

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