SUMMARY

We have measured force-frequency curves of the sternocleidomastoid muscle in six patients at three different levels of isoflurane anaesthesia (1.0, 1.4 and 1.8 MAC). Spontaneous ventilation was suppressed by mild hypocapnia induced by mechanical ventilation. An anterior force vector of the sternocleidomastoid muscle was measured during isometric contraction induced by supramaximal electrical stimulation at 20, 50 and 100 Hz to the i.m. accessory nerves of the muscle. The force response at 20 Hz and 50 Hz did not change with an increase in isoflurane concentration, but it decreased at 100 Hz as isoflurane concentration increased. The reduction in the force at 100 Hz may be caused mainly by impaired neuromuscular transmission.

KEY WORDS

It has been shown that volatile anaesthetics depress diaphragmatic muscle function [1-3], but their effects on the sternocleidomastoid muscle, an accessory muscle of ventilation, have not been fully explored. The aim of this study was to examine the effects of isoflurane on the contractile response of sternocleidomastoid muscle to indirect stimulation of increasing frequencies at different levels of anaesthesia.

PATIENTS AND METHODS

We studied six male patients aged 17-49 yr, of average height 167.3 (sd 3.4) cm and weight 66.0 (7.6) kg, respectively. All were undergoing elective orthopaedic surgery under general anaesthesia. None had clinical evidence of any respiratory, cardiovascular or neuromuscular disorder. The study was approved by the Ethics Committee of the University, and informed consent was obtained from all patients.

The patients received atropine 0.5 mg and hydroxyzine 50 mg i.m. 30 min before induction of anaesthesia. Anaesthesia was induced with thiopental 3 mg kg⁻¹ i.v. followed by nitrous oxide and isoflurane in oxygen. No neuromuscular blocker was administered during maintenance of anaesthesia. Immediately after tracheal intubation, anaesthesia was maintained with isoflurane and nitrous oxide in oxygen and the lungs were ventilated mechanically. End-tidal concentrations of carbon dioxide (PETCO₂), isoflurane, nitrous oxide and oxygen were monitored continuously by an infra-red gas analyser (Datex Normac Ultima), which had been calibrated against a standard gas (Datex Quick Cal Can Calibration Gas). Ventilation was adjusted to keep PETCO₂ at 4.0-4.7 kPa at three levels of anaesthesia of 1.0, 1.4, and 1.8 MAC in random sequence. An end-tidal concentration of 1.15% was taken as 1 MAC for isoflurane. All subjects were in the supine position.

The sternocleidomastoid muscle of the left side was stimulated via a surface electrode placed over its midpoint where the muscle receives its motor innervation from the spinal accessory nerve, using an electric stimulator (San-ei Instrument Type 3F-36) (fig. 1). One electrode (anode) was secured to the skin over the upper sternum and the precise position of the stimulating electrode (cathode) on the midpoint of the left sternocleidomastoid muscle was determined by observing the contractions produced by a series of single stimuli. Maximal contraction was achieved by stimulating its midpoint precisely at 80-100 V. On the basis of this observation, we used square-wave impulses of 50 μs duration at 100-120 V for obtaining supramaximal stimulation.

An anterior force vector of the sternocleidomastoid muscle was measured by a method described by Edwards’ group [4, 5] with a probe applied to the tendon of the sternal head of the sternocleidomastoid muscle at 2 cm from the manubrium (fig. 1). The patient’s head was maintained on a firm pillow. The probe attached to an isometric force displacement transducer (MEC Model ME-4021) was applied at right angles to the tendon so as to displace the tendon posteriorly. Further displacement did not then affect twitch height. If the pressure on the tendon was not sufficient, it was noted that the force response was both reduced and variable. A clamp was used to fix the probe and the patient’s head was held firmly in the correct position by an assistant in order to avoid...
ISOFLURANE AND STERNOCLEIDOMASTOID MUSCLE

**Fig. 1.** Method of recording contraction force from the sternal tendon of the sternocleidomastoid muscle. The patient's head is held firmly during the contraction. The head of the transducer probe is applied to the tendon to displace it backwards. When the muscle contracts and attempts to shorten, an anterior force vector is recorded.

Recorder: Rectigraph; transducer = ME-4021; sampling tube is for carbon dioxide and anaesthetic gas.

Artefacts during contraction. Furthermore, to ensure the same initial length of the sternocleidomastoid muscle and the diaphragm, the airway was occluded at functional residual capacity (FRC) before stimulation. Minor movements of the patient's head and slight variations in positioning from one contraction to the next did not significantly influence the results.

The contraction force of the sternocleidomastoid muscle was measured at each level of isoflurane anaesthesia. All measurements were made at least 15 min after establishing a constant end-tidal isoflurane concentration. When ventilation was suspended at FRC, the sternocleidomastoid muscle was stimulated at 20, 50 and 100 Hz for 2–3 s at each frequency, thereby allowing time for plateau forces to be attained. The amplified output from the force transducer was recorded on a four-channel recorder (Nihondenki San-ei Rectigraph). The force transducer response time was rapid, with a linear response over the range of force measurements. Two stimulations were performed at each frequency at 1-min intervals, and the average value of the two was used in the data analysis.

Throughout the study, arterial pressure was monitored continuously and arterial blood-gas tensions were measured at the end of each set of data. All values are given as mean (SD). Statistical analysis was performed using repeated-measures analysis of variance and Tukey's multiple test. Differences were considered significant when \( P < 0.05 \).

**RESULTS**

Arterial blood-gas tensions and acid–base status were maintained within the normal range throughout the study. Spontaneous ventilation never occurred. There was a tendency for arterial pressure to decrease in proportion to the increase in isoflurane concentration, but systolic arterial pressure never decreased to less than 80 mm Hg.

A typical record of the contraction force of the sternocleidomastoid muscle in one subject is shown in figure 2. The contraction force when the sternocleidomastoid muscle was stimulated at 20 Hz increased gradually for 2–3 s after stimulation and did not reach a maximal value during stimulation. The contraction force at 50 Hz reached a peak value immediately after the stimulation was started and then showed a plateau. The contraction force at 100 Hz also showed a peak value immediately after the start of the stimulation, but decreased subsequently for 2–3 s. The peak values at 50 Hz and 100 Hz were almost the same at each level of isoflurane anaesthesia in each patient. These findings were observed at 1.0, 1.4 and 1.8 MAC of isoflurane, but the fade in contraction force at 100 Hz was more prominent in proportion to the increase in isoflurane concentrations.

**Fig. 2.** Typical records of the contraction force of the sternocleidomastoid muscle of one patient with stimulation frequencies of 20, 50 and 100 Hz. a, b and c indicate 1.0, 1.4 and 1.8 MAC of isoflurane, respectively.
The value of the contraction force 2 s after the start of stimulation was used for making the force–frequency curves. Figure 3 shows the average force–frequency curves at 1.0, 1.4 and 1.8 MAC of isoflurane, 100% being the maximal force obtained in each patient. The contraction force at 20 Hz and 50 Hz remained unchanged at all three levels of isoflurane anaesthesia. The contraction force at 100 Hz was reduced significantly in the groups at 1.4 MAC (75.4 (11.4)%) and 1.8 MAC (51.5 (9.5)%) compared with the group at 1.0 MAC (88.0 (10.8)%) (P < 0.01). The contraction force at 100 Hz was inversely proportional to isoflurane concentration.

**DISCUSSION**

The effect of volatile anaesthesia on respiratory muscle function, especially the diaphragm, has been studied in vivo in dogs and rats [1–3], but has not been investigated fully in humans. The diaphragm is the most important muscle of inspiration, but it is sometimes difficult to examine its contractility clinically [6, 7]. In contrast, the sternocleidomastoid muscle may be studied easily with Edwards' technique [4, 5].

In patients with airway obstruction and hyperinflation, the sternocleidomastoid muscle plays an important role for ventilation, serving to raise the sternum and expand the upper thorax, but the sternocleidomastoid muscle in normal subjects is active only at increased frequencies of ventilation. Thus the sternocleidomastoid muscle might have only a minor role in ventilation of all the subjects in this study. However, there is a classical report that a young patient with paralysis of virtually all other respiratory muscles survived for several weeks on his sternocleidomastoid muscles alone [8]. The sternocleidomastoid muscle may therefore be a potentially important muscle of ventilation.

In the present method, the midpoint of the sternocleidomastoid muscle was stimulated percutaneously where it receives its motor innervation, and it is possible that the sternocleidomastoid muscle may be contracted by direct stimulation of the muscle. However, the 50-μs impulse is too short in duration to produce direct muscle contraction [9].

In our study, fixation of the head prevented head movement during maximal tetanic stimulation. Wilson and colleagues have demonstrated that, with this arrangement, overall muscle shortening amounted to only 3–4% of the total muscle length during stimulation [5]. Thus the contraction of the sternocleidomastoid muscle in the present study was considered to be essentially isometric.

We stimulated the intramuscular nerves of the sternocleidomastoid muscle supramaximally. Thus it is possible, by analysis of the force–frequency characteristics of the muscle, to discriminate between the contractile mechanism and the neuromuscular junction as the impaired site in the periphery, if the innervating nerve can be supramaximally stimulated.

The present study showed that the force response at 20 Hz and 50 Hz remained unchanged at 1.0, 1.4 and 1.8 MAC of isoflurane, while the force response at 100 Hz was depressed in proportion to the level of isoflurane anaesthesia. As isoflurane concentration increased, a decrease in the force response at 100 Hz became dominant. These results are almost consistent with those obtained by Veber and colleagues in rat diaphragm in vivo [3].

The physical range of intrinsic neural firing rates has been considered to be 5–30 Hz [10]. Edwards and his co-workers [11, 12] have suggested that the loss of force at these low frequencies of electrical stimulation may be attributed to the impairment of excitation–contraction coupling. On the other hand, the loss of force during greater frequencies of electrical stimulation may result from neuromuscular block and a reduced excitability of the muscle fibre membrane [11, 12]. Thus the impairment of the sternocleidomastoid muscle function at 100 Hz may result from impairment of neuromuscular transmission, reduced membrane excitability, or both, and not from excitation–contraction coupling.

It has been said that fast twitch fatigability is related closely to the impairment of neuromuscular transmission [13]. As the sternocleidomastoid muscle contains more fast twitch fibres than the diaphragm [14, 15], the sternocleidomastoid muscle may be more susceptible to impaired neuromuscular transmission than the diaphragm.

As shown in figure 2, the contraction force stimulated at 100 Hz reached a peak value immediately after stimulation and then faded for 2–3 s. Bowman, Marshall and Gibb have suggested that fade during greater frequency stimulation results mainly from prejunctional block of the neuromuscular junction [16]. In contrast, Waud and Waud showed that isoflurane blocks not only the capacity of carbachol to depolarize the nerve membrane, but also the affinity of acetylcholine receptors for tubocurarine, suggesting a prejunctional action of this drug [17]. Therefore, reduced force of contraction of the sternocleidomastoid muscle at greater frequencies during isoflurane anaesthesia may probably be attributed to impairment of neuromuscular transmission.
A reduction in force at 100 Hz also could be caused by decrease in energy substrate supply [11, 12]. From an anatomical point of view, the sternocleidomastoid muscle receives less blood supply than the diaphragm when arterial pressure decreases [18]. In this study, however, this effect was considered minimal, as systemic arterial pressure did not decrease to less than 80 mmHg throughout the study. Respiratory and metabolic acidosis are also known to modify diaphragmatic contractility [19,20], but none of our patients had such changes, and these effects may be excluded.

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REFERENCES


Hämodynamische Parameter zur Abschätzung
des myokardialen Sauerstoffverbrauchs in der Anaesthesie

P. Saar, D. Kettler und H. Sonntag
Zentrum Anaesthesiologie der Universität Göttingen

Hämodynamische Parameter betrifft rechnet myocardial oxygen
consumption in anesthesia.

Abstract. Direct measurements of myocardial oxygen consumption (MV'O\textsubscript{2}) and of a number of hemodynamic
variables were performed in 60 patients with coronary
heart disease undergoing three-vessel coronary
artery bypass surgery. Anesthetic procedures included halothane,
cyclopropane, isoflurane, propofol, fentanyl, and morphine
anesthesia. The following hemodynamic variables were correlated with MV'O\textsubscript{2}:
maximal systolic pressure (P\textsubscript{smp}), mean arterial pressure
(P\textsubscript{MAP}), heart rate (HR), cardiac index (CI), stroke volume index
(SVI), and the following modified variables:
MV'O\textsubscript{2} \times (HR \times CI)
MV'O\textsubscript{2} \times (HR \times SVI)
MV'O\textsubscript{2} \times (HR \times P\textsubscript{MAP})
MV'O\textsubscript{2} \times (HR \times P\textsubscript{MAP} \times CI)
MV'O\textsubscript{2} \times (HR \times P\textsubscript{MAP} \times SVI)

The product of P\textsubscript{smp} \times (HR \times CI) is equivalent to the
tension-time-index as modified by Breuchner and the
product P\textsubscript{MAP} \times (HR \times CI) to tension-time-index as
modified by Robinson. Production and uptake of lactate as metabolic equivalents of myo-
cardial oxygen consumption were also correlated with myo-
cardial oxygen consumption. Measurements were performed
before induction of anesthesia and after induction of
anaesthesia without surgical stimulation. During anesthesia
and after the operation no obtained 205 evaluable mea-
surements (MV'O\textsubscript{2} \times hemodynamic variables). The cor-
relation of MV'O\textsubscript{2} with systolic blood pressure correlated with heart rate (r=0.72). Stroke
volume was not correlated with myocardial oxygen consumption. Our results indicate that MV'O\textsubscript{2}
cannot be estimated from systolic blood pressure, heart rate and heart rate product

There are no safe limits for mean pressure, heart rate and rate pressure
product that include myocardial ischemia. Because of
regional differences in the myocardial oxygen consump-
tion ischemia cannot be assessed solely from absolute
lactate differences. It is not advisable to assume
"safe limits" of hemodynamic variables, individual
values must be defined. Adequate close monitoring of blood pressure,
heart rate and ECG may help to disclose myocardial ischemia.

Zusammenfassung. An 60 Patienten mit koronarem Herz-
defekt, die sich einer coronaren Bypass-Operation unterzogen, wurden direkte myokardiale Sauerstoffver-
brauchsmessungen durchgeführt. Die Messungen wur-
den unter den Einfluß folgender Anästhesieverfahren durchgeführt: Halothan, Enfuran, Enfuran, Propofol, Fentanyl, Methadon, Fentanyl und Morphin. Folgende hämodynamische Parameter wurden mit dem myokar-
dialen Sauerstoffverbrauch korreliert: maximaler systo-
lischer Druck (P\textsubscript{smp}), mittlerer arterieller Blutdruck
(P\textsubscript{MAP}), Herzfrequenz (Frequenz) und Druck
index (CI). Einige dieser Parameter wurden durch Multiplikation folgender Kombinationsparameter
Sauerstoffverbrauch korreliert wurden: P\textsubscript{smp} \times (HR \times CI), P\textsubscript{MAP} \times (HR \times CI), P\textsubscript{MAP} \times (HR \times SVI), P\textsubscript{MAP} \times (HR \times CI) \times (SVI)

Das Produkt P\textsubscript{smp} \times (HR \times CI) entspricht dem modifi-
zierten "Tension-Time-Index" nach Breuchner und das
Produkt P\textsubscript{MAP} \times (HR \times CI) \times (SVI)

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