A non-invasive method for measuring inspiratory muscle fatigue during progressive isocapnic hyperventilation in man

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Summary. Eleven normal adults each performed a ten minute progressive isocapnic hyperventilation (PIHV) test in which ventilatory levels were increased every two minutes. All subjects exhibited mechanical fatigue by failing to maintain the target of 80% of maximum voluntary ventilation (MVV). The mean ventilation at this level was 67.5 ± 1.4 %MVV. This fatigue was accompanied by a fall in transdiaphragmatic pressure. During the test the EMG of the sternomastoid (SM) was monitored by surface electrodes and was analyzed using fast-fourier transform. The centroid frequency (Fc) fell as ventilation increased, and correlated negatively with the inability to achieve target ventilation ($r = -0.99, p < 0.015$). Five subjects performed the test while the diaphragmatic EMG was recorded from an oesophageal electrode (D1es) and from surface electrodes (D1s). The Fc of D1es fell with increasing ventilation levels ($r = -0.95, p < 0.05$) and there was a correlation between the Fc changes of both D1es and the SM ($r = 0.92, p < 0.001$). The Fc of D1s did not correlate with either mechanical performance or the Fc of D1es, because of contamination of surface signals by signals from expiratory muscles. It is concluded that the PIVH along with surface monitoring of EMG activity from the sternomastoid can serve as a non-invasive method for evaluating inspiratory muscle fatigue.

Key words: Sternomastoid — Diaphragm — Electromyography — Centroid frequency

Introduction

It is well established that the respiratory muscles have limited endurance in performing high levels of ventilation and that the critical ventilation which can be sustained for 15 min by normal untrained subjects varies from 39 to 69% maximum voluntary ventilation (MVV) (Freedman 1970; Tenney and Reese 1968; Zocche et al. 1960). The inability to sustain high ventilation levels for long periods is thought to stem from fatigue of the respiratory muscles. This may be due to the inability to adequately increase energy supplies to the diaphragm (Roussos and Aubier 1982; Tenney and Reese 1968) or to an increase in blood lactate concentration as a result of anaerobic metabolism (Freedman et al. 1983; Roussos and Aubier 1982).

Respiratory muscle fatigue has been investigated commonly by resistive loading of the inspiratory muscles and by following changes in transdiaphragmatic pressure (Pdi). Such studies have been undertaken in normal and ill subjects (Bellemare and Grassino 1982a; Belman and Mittman 1980; Cohen et al. 1982; Grassino and Macklem 1984; Gross et al. 1979, 1980). However, tests involving inspiratory loading are invasive in that they require subjects to swallow esophageal balloons. They also require considerable cooperation in reproducing exact Pdi swings (Bellemare and Grassino 1982). A more recent approach which involves the production of muscle fatigue by sustained hyperventilatory effort has been used by a few investigators (Bai et al. 1984). While mechanical fatigue in the earlier method is defined as the inability to achieve requested levels of Pdi, fatigue in the latter method is defined as the inability to achieve target ventilatory levels upon request.
Diaphragm electrical activity during inspiratory resistive loading exhibits changes in the EMG frequency distribution, with a decrease in the ratio of the high to low (H/L) frequency components (Bellemare and Grassino 1982b; Gross et al. 1979). Much of the original work in this field used EMG signals from the diaphragm recorded by surface electrodes, but the reliability of such recordings has been challenged by the same investigators who suggested that it may be contaminated by signals from the abdominal muscles (Bellemare and Grassino 1982b).

The sternomastoid is an important muscle of respiration. Moxham et al. (1980; 1981) in two separate studies investigated the “force-frequency” response of the sternomastoid and diaphragm. They showed that after fatigue less force was generated by either muscle in response to a given stimulation. This technique, however, is not suitable as an everyday clinical technique because of the painful procedure involved. In order to develop a simple non-invasive test that could be applied easily to healthy or sick patients, including children, we decided to investigate the use of progressive isocapnic hyperventilation (PIHV) as a test of respiratory muscle fatigue, and to compare the electrical behaviour of the sternomastoid with that of the diaphragm.

Subjects and methods

Eleven healthy volunteers, aged 25 to 40 years, participated in the study. Forced vital capacity manoeuvres and 15 s MVV were measured in each subject, using a No. 3 Fleisch pneumotachograph (Hewlett-Packard 47804A based pulmonary calculator system). The best of 3 efforts was taken as the baseline. Anthropometric data and lung functions are presented in Table 1. While seated on a chair, each subject was asked to breathe quietly through a one-way valve for two minutes and then to breathe at 20, 40, 60 and 80% of his/her MVV level in a progressive fashion. Each level was maintained for two minutes. The target ventilation level was set by evacuating a balloon in the expiratory line at the desired rate through a rotameter. The subject was asked to breathe hard enough to keep the balloon from collapsing. Expiratory flow was measured with a Fleisch No. 3 pneumotachograph and was electronically integrated (HP 8815A) to give minute ventilation (Ve). The system was calibrated before each test, and all data were corrected to BTPS. End tidal CO₂ was continuously monitored by an infra-red CO₂ analyzer (901-MK2, PK Morgan Ltd, Great Britain), and CO₂ was added to the inspired air as required to keep end-tidal PCO₂ at the resting level.

The EMG of the sternomastoid (SM) was recorded by a pair of bipolar electrodes 2 cm apart, (Silver/Silver Chloride “Pregelled Disposable Premie ECG Electrodes”, Conmed, NY, USA) which were attached above its mid-point on the right side of the neck. Five of the subjects performed the test while the electrical activity of the diaphragm was also recorded by both oesophageal (D1es) and surface (D1s) electrodes. The oesophageal electrode (an adapted pacemaker, “VISI-PEEL 6”, 5651 USCI, Chaston, USA), was passed through the nose into the stomach and then placed just above the gastro-oesophageal junction (Onal et al. 1981) under fluoroscopic control. Once in place, the tube was secured to the nostrils by adhesive tape to prevent displacement during the test. The diaphragmatic surface electrodes were placed on the 6th and 7th intercostal spaces, 1 cm from the costal margin as described by Gross et al. (1979). The subjects were asked to perform 10 Mueller manoeuvres and 10 deep breaths to confirm the correct location of all the electrodes over inspiratory muscles. It can be seen (Fig. 1) that during both manoeuvres all three electrodes recorded inspiratory muscle activity simultaneously.

The EMG signals were amplified to a maximum of ±5 volts by a set of 2 amplifiers. A pre-amplifier (home made) with a frequency range of 10–1000 Hz, amplified the signals roughly 4000 times. Signals were further amplified by a medium-gain amplifier (Hewlett-Packard 8802A) with the same frequency range. Inspiratory flow together with the EMG signals were recorded on a 4-channel tape recorder (Hewlett-Packard 3964A).

The EMG and flow signals were played back, one muscle at a time, from the magnetic tape through an A/D converter and into a computer (Digital PDP 11/23) at a sampling rate of 1000 Hz. The bandwidth chosen for the EMG analysis was limited to 20–250 Hz (Schweitzer et al. 1979). The first stage of the analysis of both oesophageal and surface diaphragmatic electrical activity involved eliminating cardiac electrical activi-