Short report

Electromyographic investigation of the diaphragm and intercostal muscles in tetraplegics

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SUMMARY Local electromyographic activity has been demonstrated in the intercostal muscles of tetraplegic patients by using a combination of surface, fine-wire and intra-oesophageal diaphragmatic electrodes. This activity is first present and most evident in the lower chest, the point of maximum deformation in the tetraplegic's thorax. In patients with long standing injury the activity is present and prominent in the more superior intercostal muscles. We believe this activity to be reflex in character and that it develops and facilitates over time, so improving the ventilatory capacity of tetraplegics.

There is a high mortality from respiratory failure among tetraplegic patients. Among 46 acute deaths in spinal patients who died within six weeks of injury, 12 tetraplegics died of respiratory failure. The patient with a high cervical injury maintains ventilation almost entirely with his diaphragm, although a little assistance is obtained from his auxiliary muscles of respiration. The vital capacity is consequently greatly reduced and, when the diaphragm is involved, vital capacities as low as half a litre have been recorded. The investigation of respiratory muscle function is thus of importance from both a practical and a theoretical point of view. Our basic knowledge of the activity of the diaphragm and intercostal muscle interaction, although extensively studied, is limited in the normal subject and almost non-existent in the tetraplegic.¹⁻³ Previous investigation⁴⁻⁶ of respiratory muscle relationships and correlative chest movements have shown the presence of intercostal EMG activity, using a combination of surface and needle electrodes.

The tetraplegic patient is particularly suitable for investigation of these muscles, since in normal subjects the pectoralis major muscle as well as the serratus anterior and abdominal oblique muscles makes it difficult to record surface intercostal

electromyographic activity.⁷⁻⁹ In a patient with a cervical spinal cord lesion these muscles atrophy and are no longer under voluntary control so that more precise electromyographic recording can be made of the underlying intercostal muscles. It is then possible to differentiate the diaphragm from the intercostal electromyographic activity if the diaphragm is monitored by an intra-oesophageal electrode.¹⁰⁻¹⁶

The current investigation was undertaken to correlate the EMGs of the diaphragm and intercostals in order to demonstrate this local activity in the intercostal muscles, and to show that it was not transmitted from the diaphragm.

Materials and methods

Five male tetraplegics were studied. The essential information for each patient is presented in table 1. Only patients with complete lesions of the cervical cord as a result of trauma were investigated. Completeness of the lesion was determined by neurological examination, which revealed complete loss of sensation and motor function below the level of trauma. The patients performed a series of

Table 1 Patient information

Patient	Age (yr)	Level of lesion	Time since lesion
B	19	C5	5 days
F	26	C5, 6	1 month
A	23	C5	9 months
E	30	C5	6 years, 4 months
W	49	C6	25 years

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activities which included quiet and deep breathing, coughing and straining. Each patient completed several series for each recording site.

An intra-oesophageal electrode was utilised in three subjects for the diaphragm electromyographic recordings.¹⁰⁻¹⁷ The placement of the intra-oesophageal electrode was accomplished through the nasal orifice into the pharynx and subsequent swallowing by the patient, in accordance with Schweitzer *et al.*¹⁷ The use of balloons was not considered necessary. The recording apparatus was monitored as the patient alternatively swallowed and took a deep breath until the maximum electromyographic activity was obtained, at which point the lead to the electrode was held in place by securing it to the face with tape. Surface electrodes were placed on the left side of the supine patient from the third to the eighth intercostal spaces sequentially at distances of 9 and 18 cm from the midsternal line. In a previous investigation by Taylor¹⁸ the parasternal region was shown to be the site of optimum inspiratory activity, electromyographic activity being present even during quiet breathing. In three subjects (E, A and W) fine wire electrodes were placed in the intercostal muscles directly beneath the surface electrodes to correlate the activity isolated within the surface electrodes. It has been demonstrated that the fine-wire electrode has a range limited to no more than 1.5 mm, similar to the restricted area of detection of the bipolar needle electrode.^{7 18 19} The combination of intra-oesophageal and bipolar fine wire electrodes allows the differentiation between diaphragmatic and intercostal EMG.¹⁰⁻¹² The amplifier-recording system consisted of four high-gain preamplifiers (Medelac A9) and a jet spray minograph (Siemens Medical). The preamplifiers had bandpass filters of 10-1000 Hz and the gain was

adjusted to 250 V/cm. The galvanometers of the recorder had a resonance frequency of approximately 500 Hz. No attempt was made to quantitate the EMG activity but instead close attention was given to the presence or absence of activity and the onset of activity in each of the EMG channels. The EMG activity was related to the simultaneously recorded spirometry tracing to correlate with the phases of respiration.

Results

The results on all the patients are presented in table 2. Additional information will be provided at appropriate points in the discussion in order to prevent repetition. The first aim was to demonstrate that the activity detected with the surface electrodes over the intercostal muscles arose locally from these muscles. The electromyographic evidence of the activity of the intercostal muscle during a deep breath is shown in fig 1a. There was synchronous onset of activity during inspiration in both the fine-wire and surface electrodes of the eighth intercostal muscle. This onset contrasts with the asynchronous initiation of activity in the diaphragm. The continuation of the activity beyond the expected inspiration into the expiratory period should be noted in the intercostal muscles. Additional continuous simultaneous after-discharge spikes may be observed in both channels beyond the major activity but were not present in the diaphragm recording. This was evidence of local activity of the

Table 2 Results

Activity	Subject	Intraoesophageal electrode	EMG activity					
			Surface intercostal electrodes 3 (9 cm)	4 (9 cm)	5 (9 cm)	6 (9 cm)	7 (18 cm)	8 (18 cm)
Quiet breathing	B	no	0	0	—	—	0	0
	F	yes	0	0	0	0	+	+
	A	yes	0	0	0	0	0*	0*
	E	yes	0	+	0	0*	+	0*
	W	yes	0	+	—	—	+	+
Deep breathing	B	no	0	0	—	—	0	0
	F	yes	0	0	0	+	+	+
	A	yes	0	0	0	0	+	+
	E	yes	0	0*	+	0*	+	+
	W	yes	+	+	—	—	+	+
Cough	B	no	0	0	—	—	0	0
	F	yes	+	0	0	0	+	+
	A	yes	0	0	0	0	+	+
	E	yes	+	+	—	—*	0*	0*
	W	yes	+	+	—	—	+	+
Strain	B	no	0	0	—	—	0	0
	F	yes	+	0	0	0	+	+
	A	yes	0	0	0	0	+	+
	E	yes	0	0*	0	0	0*	0*
	W	yes	+	+	—	—	+	+

+ = activity present

0 = activity absent

— = not recorded

*bipolar fine wire electrodes were used in conjunction with surface electrodes.

intercostals and not of the diaphragm radiating through and being recorded by the intercostal electrodes. While the continuous spike activity seen in the intercostal muscles was not present in every recording it was seen so frequently in both the acute and chronic patients to be a prominent feature.

Figure 1b shows two breaths in patient E. There

is well marked diaphragmatic activity at the initiation of each breath. The intercostal activity is more marked in the third than in the fourth space and is present when the diaphragm is silent. We have seen another patient (G) not included in the following analysis because he had minimal sensory sparing, who had severe spasms of his intercostal muscles

Fig (a) Channel 1—S, spirometer trace; Channel 2—8ICS-S, eighth intercostal space, surface electrode; Channel 3—8ICS-W, eighth intercostal space, wire electrode; Channel 4—D, diaphragm. (b) Channel 1—S, spirometer trace; Channel 2—3ICS—third intercostal space, wire electrode; Channel 3—Dia—diaphragm, intra-oesophageal electrode; Channel 4—4ICS—fourth intercostal space, wire electrode. Activity detected with wire in the third and fourth spaces is well removed from the diaphragm. (c) Channel 1—S, spirometer trace; Channel 2—D—diaphragm; Channel 3—7LICS—seventh left intercostal space, surface electrode; Channel 4—7RICS—seventh right intercostal space, surface electrode. Sp—spasm. The trace is continuous in Patient G. In the early part of the trace (a) the activity of the diaphragm accompanies a small inspiration, however, when there is a generalised spasm of the intercostal muscles the patient is unable to breathe and there is no activity of the diaphragm.

which suppressed the activity of the diaphragm, thus demonstrating conclusively that activity can arise in the paralysed intercostal muscles which is not radiating through from the diaphragm (fig 1c).

Patient B who was in spinal shock was studied by surface electrodes five days after injury. No intra-oesophageal electrode was used but a surface electrode was placed on the belly of the right sternocleidomastoid so that respiration could be correlated with the spirometry trace. No intercostal electromyographic activity was detected nor was any diaphragmatic activity detected through the intercostal spaces.

Table 2 shows a distinct increase in the activity of the intercostal muscles in the more superior intercostal spaces with the length of time since trauma. The acute patient (F) did not have as much EMG activity in the third and fourth intercostal space as does the chronic patient.

Discussion

The results confirm previous findings⁵ that the activity detected in the intercostal muscles is arising within these muscles since it was detected with wires, and wires only detect activity 1.5 mm from their tip. The activity was different in timing from that of the diaphragm, being present when activity in the diaphragm was absent. It was found well removed from the diaphragm in the upper spaces of the chest; it was absent during spinal shock. It was present in the intercostal muscles when the diaphragm was silent during generalised spasm. The nature of the activity showing after-spike discharges was different from normal interference patterns. This activity thus arises locally in paralysed muscles, and we believe it to be reflex, the afferent limb being in the intercostal nerve, the stimulus being deformation of the inferior thoracic wall causing stretching of the muscle spindles of the intercostal muscles resulting in reflex contraction of these muscles.

Both acute (F) and chronic (W) patients demonstrated the reflex activity in the seventh and eighth intercostal spaces. These have been reported to be the points of maximum deformation in early recovery of tetraplegics.²⁰ In a duplication of this experiment in patient (E) the maximum deformation was confirmed by use of calipers at the seventh intercostal space during deep breathing. Additional support for the idea of reflex activity of the intercostals is demonstrated by the fact that even in the acute patient (F) strenuous activity such as the cough or strain consistently produced EMG activity in the superior intercostal muscles (table 2). The

development of this reflex activity is possible because of the presence of a rich supply of proprioceptors to the intercostal muscles.²¹ Since the diaphragm lacks "autogenetic" facilitation due to its diminished number of muscle spindles, the presence of "autogenetic" inhibition from a relative excess of tendon organs creates a potentially dangerous situation. The reflex activity of the intercostals becomes of prime importance in the stimulation of the phrenic motor-neurone pool for respiration.²²

A similar conclusion has been drawn from other lines of research. A non-electromyographic investigation of tetraplegics reached the same conclusion concerning the spastic reflex activity of the intercostal muscles.²³ Goldman and George's study was based on the differences in blood gas analysis during postural changes in tetraplegics before and after one year following injury. They concluded that the decreased activity of the intercostal muscles early in the course of recovery results in hypoventilation of the lungs. The positional hypoxemia is decreased as the spastic reflex activity of the intercostal muscles develops.

These findings are of great functional significance since in the initial stages the vital capacity in a tetraplegic patient can be as little as 300 ml, but as time passes it can rise to two-thirds of the predicted normal. The presence of EMG activity in the intercostal muscles may well contribute to the improvement in the ventilatory function since the rib cage will be stabilised to provide a better fulcrum for the diaphragm to act upon. Conversely when generalised spasms develop in the whole body that involve the intercostal muscles they can inhibit the diaphragm, producing dyspnoea.²⁴

Our findings of differential electromyographic activity in the intercostal muscles and the diaphragm warrant caution in the use of surface electrodes to pick up electrical activity of the diaphragm. Previous investigators have issued this caution,⁷⁻⁹ and we fully support their contention. It may be possible to speak of the total fatigue of both intercostal and diaphragm based on surface electromyographic recordings but one would be hard pressed to attribute diaphragm fatigue to changes in surface electrode electromyograms alone. The difference in slope of the fatigue patterns noted in a previous investigation² may be accounted for by the additive effect of the intercostal reflex activity rather than recording from different motor units, different distances, or differences in dielectric properties.

Our investigation leads us to conclude that there is a distinct reflex activity of the intercostal muscles in the tetraplegic patient which cannot be due to radiation from the diaphragm.

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