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Motion of the rib cage and the abdomen in tetraplegic patients

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Summary

1. We have studied the motion of the abdomen and the rib cage in patients with a transection of the lower cervical spinal cord during normal breathing both in the supine and sitting posture, and compared it with that of normal subjects.

2. In the supine posture the rib cage of a patient moves paradoxically inward, therefore his chest wall is deformed, which explains the high work of breathing.

3. During expiration, beside the recoil of the respiratory system, there is also the recoil of the deformed chest wall, toward its passive configuration, with an expansion of the rib cage above its resting position during the first part of expiration and an alteration of the expiratory flow profile.

4. In a sitting ‘relaxed’ posture the paradoxical inward motion disappears in the lower rib cage, and it is reduced but still present in the higher rib cage.

5. We conclude that contraction of the diaphragm constricts the ‘passive rib cage’, either directly through its insertions or indirectly through the reduction of intrathoracic pressure. In seated subjects the diaphragm causes some expansion of the rib cage at its lower level. Therefore the motion of the rib cage is not only related to the balance between the forces developed by the diaphragm and the intercostal muscles, but also to the diaphragm dome configuration, the geometry of the rib cage and the lung volume.

Key words: abdomen, chest wall, diaphragm, lung rib cage, spinal injuries, tetraplegia.
Abbreviations: A–P, anterior–posterior diameter; FRC, functional residual capacity; L–L, lateral–lateral diameter; TLC, total lung capacity.

Introduction

The diaphragm is the main respiratory muscle still in activity in tetraplegic patients. The motion of the rib cage in these patients can be almost completely attributed either to the direct action of the diaphragm through its insertions on the lower ribs or to its indirect action through the reduction of intrathoracic pressure. The rib cage is essentially passive (‘passive rib cage’ is used to refer to this state). Furthermore, the increasing abdominal pressure caused by the descent of the diaphragm could also have a direct effect on the motion of the lower rib cage (Goldman & Mead, 1973). A paradoxical inward movement of the rib cage during inspiration in subjects with low cervical cord transection has been observed by many authors (Duchenne, 1867; Cameron, Scott, Jousse & Botterell, 1955; Bergofsky, 1964; Moulton & Silver, 1970; Fugl-Meyer & Grimby, 1971). On the contrary, an expanding action of the diaphragm on the lower lateral rib cage has been demonstrated by Lemon (1928a, b) and Douady & Michel (1934).

We have measured the motion of the rib cage and the abdomen in patients having a complete transection of the lower cervical spinal cord during normal breathing in the supine and sitting posture, and by comparison with the corresponding motion in normal subjects we could separate the direct and
indirect mechanical action of the diaphragm on the passive rib cage.

**Methods**

We have measured the motion of the rib cage and the abdomen in eight patients with a complete transection of the lower cervical spinal cord. Six of these patients have been studied only in the supine posture and two both in the supine and sitting posture. Two patients have been studied while breathing through a tracheostomy and after returning to normal breathing through their upper airways (see Table 1).

The motion of the rib cage and the abdomen has been recorded with the magnetometer described by Mead, Peterson, Grimby & Mead (1967), which can measure four different diameters. Each pair of coils of the magnetometer gives a signal linearly related to the change in diameter. Even if the coils of each pair do not exactly face each other the sensitivity and the linearity of the signal are not affected up to at least 4 cm of displacement (Fig. 1). Therefore a shift in their relative lateral position as it might occur during breathing would not alter the signal. No interference ('cross talk') has been detected among the signals originating from the four pairs of coils, even between the coils placed orthogonally in the same plane.

We have measured the anterior-posterior diameter of the abdomen at the level of the umbilicus, two rib-cage anterior-posterior (A-P) diameters at the level of the 7th and of the 3rd rib (7th A-P and 3rd A-P) and one lateral-lateral (L-L) diameter at the level of either the 7th rib (7th L-L) or the 3rd rib (3rd L-L). The respiratory airflow has also been measured by a Fleisch pneumotachograph and a differential pressure transducer connected either to a face mask or to a tracheal cannula. All these signals have been registered on an eight-channel Brush recorder.

The patient was moved from supine to sitting posture by lifting the head part of the bed by 90°. Owing to paralysis of their postural muscles, the patients were in a sitting 'relaxed' posture: their backs were completely supported by the bed-lift and their rib cages were passively supported by the abdomen. The same variables have been measured in seven normal subjects in both postures. These

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**Table 1. Details of the tetraplegic patients**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Lesion</th>
<th>Interval between injury and the start and the end of our study (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.H.</td>
<td>M</td>
<td>29</td>
<td>C5–C6</td>
<td>16</td>
</tr>
<tr>
<td>R.G.*</td>
<td>M</td>
<td>23</td>
<td>C6</td>
<td>6–45</td>
</tr>
<tr>
<td>A.T.*</td>
<td>M</td>
<td>32</td>
<td>C5</td>
<td>36–43</td>
</tr>
<tr>
<td>J.W.</td>
<td>F</td>
<td>17</td>
<td>C6</td>
<td>7</td>
</tr>
<tr>
<td>G.J.</td>
<td>M</td>
<td>34</td>
<td>C5–C6</td>
<td>24</td>
</tr>
<tr>
<td>M.L.</td>
<td>M</td>
<td>20</td>
<td>C5–C7</td>
<td>6</td>
</tr>
<tr>
<td>M.C.+</td>
<td>M</td>
<td>41</td>
<td>C6–C7</td>
<td>31–72</td>
</tr>
<tr>
<td>E.McI.+</td>
<td>M</td>
<td>36</td>
<td>C5–C6</td>
<td>6–20</td>
</tr>
</tbody>
</table>

* Studied in both supine and seated postures.
† Studied while breathing either through a tracheostomy or their upper airways.
Breathing in tetraplegia

Subjects were asked to sit in the same 'relaxed' posture as that obligatory for the tetraplegic patients.

Results

Similar results have been obtained in the eight patients considered.

In Fig. 2 typical tracings of the rib-cage and abdominal diameters recorded with respiratory airflow in a patient and in a normal subject during quiet breathing in supine and sitting postures are shown.

Fig. 3 shows the relationships between the rib-cage (7th A–P and 7th L–L) and abdominal diameters in the two postures derived from the records shown in Fig. 2. The diameters are represented as per cent of the maximal abdominal excursion in the supine position.

As shown in the left panel at the bottom of Fig. 2 and Fig. 3, which refers to a tetraplegic patient in the supine posture, during inspiration there is an expansion of the abdomen and a reduction of both the rib-cage A–P diameters. The 3rd L–L diameter, not shown in these records, behaves like the A–P diameters, and the 7th L–L increases slightly at the start of inspiration.

On the contrary, in a normal subject (Fig. 2 and Fig. 3) the rib cage expands in phase with the abdomen, without any difference between its two levels.

Just before the end of inspiration the rib-cage diameters of the patient terminate their inward movement and start to return to their resting position.

During expiration, there is a progressive reduction of the abdominal diameter and the rib-cage diameters at first increase, crossing over their functional residual capacity (FRC) position, and then decrease, returning to their resting position (Fig. 2 and Fig. 3). The 3rd L–L diameter (not shown in these Figures) during expiration behaves as the other rib-cage diameters.

In the normal subject (Fig. 2 and Fig. 3) during expiration there is a concurrent reduction of all diameters.

Essentially similar patterns for the motion of the rib-cage and abdominal diameters have been recorded in the two tetraplegic patients studied while breathing either through a tracheostomy or through their upper airways.

In the sitting posture the paradoxical inward motion of the lower rib cage seen in the supine posture (Fig. 2 and Fig. 3) disappears and both diameters expand approximately in phase with the abdomen, with a behaviour similar to that of normal subjects (Fig. 2 and Fig. 3). The 3rd A–P diameter (Fig. 2) and the 3rd L–L diameter (not shown) still move inward in the patient, though less than in the supine posture; the tidal motion of the abdomen, relative to rib-cage expansion, is lower than in the supine posture in both patients and normal subjects (Fig. 2 and Fig. 3).

Fig. 4 represents the relationship between the diameters of the rib cage and abdomen of a supine tetraplegic patient plotted in absolute values: the broken line from FRC to total lung capacity (TLC) shows the passive relationship between the rib cage and abdomen (Konno & Mead, 1967) obtained by having the patient relax against closed airways after having inspired to different lung volumes. This relaxation manoeuvre proved to be far easier and very reproducible in these patients compared with normal subjects, presumably because most of their respiratory muscles are paralysed. This relaxation curve represents the passive configuration of the rib cage and abdomen within the inspiratory capacity, i.e. the position of each of the two compartments at different volumes when the respiratory muscles are not active. During inspiration, because of the paradoxical inward movement of the rib cage the chest wall is deformed, hence this line lies outside the relaxation curve. When, at the end of inspiration, the patient relaxes against closed airways, the chest wall recoils, reaching its passive point for that volume (Fig. 4: broken line). In this situation the rib-cage diameter exceeds its end-expiratory position (FRC).

In Fig. 5 the inspiratory and expiratory airflows have been plotted against the abdominal motion for tidal volumes about 20% of the vital capacity, for seven normal subjects and four tetraplegic patients. During inspiration this relationship is essentially equal in the two groups, whereas during expiration the peak flow is attained at a lower abdominal expansion in tetraplegic subjects.

Discussion

The most conspicuous feature of the respiratory mechanics of a supine tetraplegic patient is the paradoxical inward movement of the rib cage during inspiration (Figs. 2, 3 and 4), due to lack of intercostal muscle activity (Duchenne, 1867; Cameron et al., 1955; Bergofsky, 1964; Moulton & Silver, 1970; Fugl-Meyer & Grimby, 1971).

During the first part of expiration the rib cage
expands, while the abdomen returns towards its resting position; therefore there is a shift of volume between abdomen and rib cage and again the rib cage shows a paradoxical motion (Figs. 2, 3 and 4). This 'redistribution' cannot be due to a closure of the glottis, because similar results have been recorded in patients with a tracheostomy, but it could be attributed to the mechanical situation of both rib cage and abdomen at end inspiration. In a normal subject the chest wall is a system where rib
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Fig. 3. Rib-cage and abdomen relationships of the same normal subject (top) and tetraplegic patient (bottom) as recorded in Fig. 2 in supine (left) and sitting (right) posture. Each diameter has been plotted as per cent of the maximal abdominal excursion in the supine posture. O, 7th A–P; ●, 7th L–L (anterior–posterior and lateral–lateral diameters at the level of the seventh rib respectively). The arrows indicate inspiration (−) and expiration (+).

cage and abdomen–diaphragm (which can be considered its two components) operate approximately in parallel (Agostoni & Mead, 1964); they contribute different volumes but undergo almost the same pressure. When the lung volume increases the chest wall also has to increase to the same volume
FIG. 4. Rib-cage and abdomen relationship of a supine tetraplegic patient (E.McI.): 3rd A-P = anterior–posterior diameter at the level of the third rib; ABD = anterior–posterior diameter at the level of the umbilicus. The broken line (left) represents the passive rib cage–abdomen relationship between the end-expiratory volume (FRC) and the total lung capacity (TLC). The continuous line shows the same relationship along the inspiratory (INS) and expiratory (EXP) phase of the vital capacity. The dotted line (right) represents the behaviour of the two diameters when the patient relaxes against closed airways at TLC (see the text).

FIG. 5. Inspiratory and expiratory flow (INS $\dot{V}$ and EXP $\dot{V}$) and abdominal diameter relationship (% of maximum values) of seven normal subjects (○○○) and four tetraplegic patients (●●●●). The horizontal bars represent the maximal variabilities.

(cHEST wall and lungs constitute a system in series), part of this volume being supplied by the expansion of the rib cage and part by the expansion of the abdomen–diaphragm. If the rib cage instead of expanding is constricted, as in tetraplegic patients, the abdomen expands to compensate the negative contribution of the rib cage. In these patients at end inspiration, besides recoil of the respiratory system as a whole, there is a recoil of the two components of the deformed chest wall in an opposite direction: expansion of the rib cage and return of the abdomen–diaphragm toward their resting position.

If the ‘redistribution’ overcame completely the pressure generated by the recoil of the respiratory system responsible for the expiratory flow, the first part of the expiration (EXP in Fig. 4) would be only a redistribution of air between the two compartments, and the expiratory flow would start only when rib cage and abdomen had reached their resting position for that volume. On the contrary, if the recoil of the deformed chest wall were negligible relative to the recoil of the respiratory system, the rib cage would return to its FRC position without at first expanding above it. The fact that in a supine tetraplegic patient the rib-cage diameters, expanding during expiration, cross over their resting position indicates that the ‘redistribution’ of air due to the distortion of the chest wall plays a substantial role in the first part of expiration. The recoil of the deformed chest wall can interfere with the expiratory flow profile, which indeed appears to show a delay of its peak value (Fig. 5).

In tetraplegic patients the work of deformation represents a substantial fraction of the whole respiratory work of breathing. Indeed Bergofsky (1964) found that the respiratory work in these patients during quiet breathing is more than double that in a normal subject.

In the sitting posture the paradoxical inward motion of the upper rib cage is markedly reduced and that of the lower rib cage is reversed to an outward movement. Therefore the chest wall of a seated patient is less deformed than it is in the supine posture, and the respiratory work should be accordingly lower.

The diaphragm when the intercostal muscles do
not work as in tetraplegic patients exerts a constrictor action on the upper rib cage (Fig. 2). Similar results have been observed during tetanic stimulation of the diaphragm in rabbits and dogs, either with open or closed airways (D’Angelo & Sant’Ambrogio, 1974), suggesting that the upper rib cage when the intercostal muscles are inactive is mainly driven by the intrathoracic pressure. Therefore the expanding action of inspiratory intercostal muscles on the upper rib cage should be considered to be greater than that measured by the expansion of the rib cage. In other words the intercostal muscles and the diaphragm, even if both are inspiratory muscles, would exert an antagonistic action on the upper rib cage.

The diaphragm exerts also a direct action on the lower rib cage through its insertions (D’Angelo & Sant’Ambrogio, 1974), which seems to be slightly different for its two orthogonal diameters. The diaphragm exerts a constrictor action on the A–P diameter, as for the upper rib cage, and this is antagonized in normal subjects by the expanding effect of the inspiratory intercostal muscles. On the other hand, the diaphragm appears to have a double action on the L–L diameter in tetraplegic patients: at first expanding and then constricting it.

An expanding action of the diaphragm on the lower lateral sides of the rib cage had already been described (Lemon, 1928a, b; Douady & Michel, 1934). This biphasic action could be understood considering that in the supine posture the lateral sides of the diaphragm dome at FRC have an almost parallel orientation with the lateral rib cage (Campbell, 1958). In this situation the diaphragmatic contraction leads to an expanding action, until its dome becomes flatter and therefore its action is constrictor. These findings suggest that in normal subjects either an outward or inward movement of the L–L diameter depends on the balance between the intercostal muscles and the diaphragm and, in some cases, especially at the end of a deep breath when the diaphragm dome is flatter and the intercostal inspiratory fibres are shorter and develop a lower force, the diaphragm could exceed the intercostal muscles and pull the rib cage inward. Indeed, Agostoni & Mognoni (1966) found a reduction of the L–L diameter in the lower part of the rib cage in two subjects when their respiratory volumes were increased to 70–80% of vital capacity.

In the sitting ‘relaxed’ (see the Methods section) posture, even if the FRC is increased and the diaphragm dome is lowered, the tidal motion of the diaphragm is reduced, as shown radiologically by Wade & Gilson (1951) and Wade (1953), and by the relatively smaller motion of the abdomen (Fig. 2 and Fig. 3). In fact in this posture the abdominal content does not allow the diaphragm to descend as much as in the supine posture and therefore to exert its constrictor action on the rib cage. Similarly D’Angelo & Sant’Ambrogio (1974) have shown a direct constrictor action of the diaphragm on the lower rib cage of a supine rabbit and have demonstrated that this action could be reversed into an expanding one by a mechanical fixation of its dome.

In the sitting posture the diaphragm exerts a direct expanding action on the lower rib cage. In fact, in seated tetraplegic patients the paradoxical inward motion of the lower rib cage, as seen in the supine position, disappears, and in normal subjects there is a larger expansion of the rib cage in sitting than in supine posture, relative to the abdominal expansion.

On the other hand, the diaphragm should still exert a constrictor action on the upper rib cage because at this level its action is uniquely mediated through the reduction of intrathoracic pressure. Nevertheless we would expect the expanding direct action of the diaphragm on the lower rib cage to reduce the inward motion of the upper rib cage. On this basis we understand the smaller paradoxical inward movement of the upper rib cage of our patients in sitting than in supine posture (Fig. 2).
and even the expansion of the rib cage observed in some of the chronic tetraplegic patients of Fugl-Meyer & Grimby (1971).

In the schema of Fig. 6 the presumed effects of the diaphragm (unbroken arrows) and the intercostal inspiratory muscles (broken arrows) on the lateral upper and lower rib cage in supine and sitting posture, at FRC and near TLC, as inferred from our data, have been summarized. Although in normal subjects the resultant motion of the rib cage is the vectorial sum of both forces (Fig. 6: bold arrows), in a tetraplegic patient the motion of the passive rib cage is related only to the diaphragmatic action (unbroken arrows).

In conclusion, the motion of the rib cage is related not only to the balance between the actions of the intercostal inspiratory muscles and the diaphragm but also to configuration of the diaphragmatic dome, geometry of the rib cage and lung volume.

Acknowledgment

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