Electrophysiologic Evaluation of Phrenic Nerves in Severe Respiratory Insufficiency Requiring Mechanical Ventilation*

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Diaphragmatic paralysis in patients with respiratory insufficiency compounds the problems in the management. In the presence of lower lobe atelectasis, pleural effusion, or a patient's poor respiratory effort, fluoroscopic examination is often not a reliable way to diagnose diaphragmatic paralysis. We observed that transcutaneous phrenic nerve stimulation in the neck and recording the diaphragmatic potentials from electrodes placed on the lower part of the chest is a simple, reliable, and noninvasive technique to diagnose diaphragmatic dysfunction at the bedside in critically ill patients. In 14 postoperative patients and one with cervical spinal cord injury with respiratory failure, we found ten patients who showed phrenic nerve dysfunction. Besides diagnostic utility, the electrophysiologic evaluation of phrenic-diaphragmatic function provides critical information needed for therapy.

Contraction of the diaphragm produces 60 percent of the tidal volume in normal supine individuals at rest. Evaluation of diaphragmatic function is, therefore, important in patients with respiratory insufficiency. Paralysis of the diaphragm is usually diagnosed by an elevated diaphragm on the chest x-ray film and paradoxical movement of the diaphragm observed with inspiration during fluoroscopic examination. The radiologic diagnosis is difficult in the presence of lower lobar atelectasis, pleural effusion, or with poor respiratory effort of the patient. It may also be difficult sometimes to transport a critically ill patient to the radiology unit for fluoroscopic study. We observed that electrophysiologic studies of phrenic nerves provide a useful technique for diagnosing diaphragmatic dysfunction in critically ill patients.

MATERIALS AND METHODS

Because of our interest in the electrophysiologic studies of the diaphragm, we were asked to evaluate 21 patients who had respiratory insufficiency of recent onset requiring mechanical ventilation. Fourteen patients developed respiratory dysfunction in the postoperative period following a surgical procedure, usually a thoracotomy. An elevated diaphragm on the chest x-ray film, persistent lower lobe atelectasis, inability to wean from ventilatory support, and suspicion of phrenic nerve damage during surgery prompted the anesthesiologist and the surgeon to seek a study of phrenic nerve conduction in these patients. One patient had traumatic quadriplegia due to a high cervical cord injury. The remaining six patients had decreased respiratory effort on the basis of severe polyradiculoneuropathy of the Guillain-Barré type (five patients) or myasthenia gravis (one patient).

With the patient lying supine and the head in midline, we stimulated the right and left phrenic nerves transcutaneously behind the sternocleidomastoid muscle at the level of the upper border of thyroid cartilage by conventional bipolar stimulating electrodes (TECA Corp.). The cathode was placed close to the clavicle. A disk electrode over the manubrium sternum acted as a ground electrode. Suprathreshold square-wave pulses of 0.1 to 0.5 m sec at a rate of 1/sec were delivered. The frequency response of the amplifier was set at 1.6 Hz to 32 kHz (−3 dB). By adjusting the position of the stimulating electrodes, the phrenic nerve could be selectively stimulated with minimal brachial plexus stimulation.

The diaphragmatic evoked potentials were recorded from two surface disk electrodes 8 mm in diameter; one was placed at the seventh intercostal space (7ICS) closest to the costochondral junction and the other over the xiphoid process (XP). The electrodes were held to the skin by electrolyte paste (Grass) and further secured with cloth tape (Scotch). The ipsilateral 7ICS electrode was connected to the input terminal 2 (red terminal) and the XP electrode to the input terminal 1 (black terminal) of the electromyograph (TECA-TE42). Using this XP-7ICS derivation, positivity at the 7ICS electrode yielded an upgoing waveform. The details of the technique are reported by Markand et al. The upper limits of the phrenic nerve conduction time in normal adults is less than 9.75 m sec (mean ± 2.5 SD). The amplitude of the diaphragmatic potential is greater than 0.4 mV. In children the latency is shorter (4.3 to 4.9 m sec) and is dependent on the length of the chest. The amplitude ratio of left and right responses are normally over 0.6 and less than 1.6.

RESULTS

All patients with polyradiculoneuropathy (Guillain-Barré syndrome) had abnormal electrophysiologic studies of the diaphragm, the details of which have been previously described. The amplitude of the diaphragmatic response was significantly decreased bilaterally. In two, the phrenic nerves were totally inexcitable during the height of the illness. When the diaphragmatic responses were elicitable, the latencies were usually prolonged. Two patients had serial studies starting less than a week from the onset of Guillain-
Table 1—Phrenic Nerve Conduction in 14 Postoperative and One Post-traumatic Patient with Respiratory Insufficiency

<table>
<thead>
<tr>
<th>Patient, Sex, Age (yr)</th>
<th>Precipitating Cause of Respiratory Failure*</th>
<th>Radiologic Findings†</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, M, 61</td>
<td>CABG</td>
<td>LLL atel; L pl eff; † L hemidiaphragm</td>
<td>0.28</td>
<td>9.0</td>
</tr>
<tr>
<td>2, M, 3 mo</td>
<td>Repair of coarctation of aorta</td>
<td>R lung inf/atel; LUL inf/atel; † L hemidiaphragm</td>
<td>0.90</td>
<td>4.1</td>
</tr>
<tr>
<td>3, M, 73</td>
<td>CABG 5 yr ago; recent cholecystectomy</td>
<td>† L hemidiaphragm</td>
<td>NR</td>
<td>...</td>
</tr>
<tr>
<td>4, F, 9 mo</td>
<td>Repair of congenital MI</td>
<td>RUL atel/inf; bil perihilar inf; paradoxical movement of R diaphragm (?)</td>
<td>0.80</td>
<td>4.5</td>
</tr>
<tr>
<td>5, M, 37</td>
<td>Ca of testes; thoracotomy for metastases</td>
<td>† R hemidiaphragm</td>
<td>NR</td>
<td>0.38</td>
</tr>
<tr>
<td>6, F, 4</td>
<td>Repair of artioventricular canal</td>
<td>RUL atel/inf; bil LL inf</td>
<td>0.55</td>
<td>4.3</td>
</tr>
<tr>
<td>7, F, 4</td>
<td>Repair of congenital VSD</td>
<td>R pl eff; bil pulmonary inf; † L hemidiaphragm</td>
<td>0.55</td>
<td>4.9</td>
</tr>
<tr>
<td>8, F, 71</td>
<td>CABG</td>
<td>LLL atel; L pl eff</td>
<td>0.18</td>
<td>8.5</td>
</tr>
<tr>
<td>9, M, 62</td>
<td>CABG</td>
<td>Bibasilar atel/inf; bil pl eff</td>
<td>0.45</td>
<td>9.5</td>
</tr>
<tr>
<td>10, F, 54</td>
<td>Right upper lobectomy for Ca of lung</td>
<td>RUL inf; R eff; L inf; fluoroparadoxical movement R</td>
<td>NR</td>
<td>1.30</td>
</tr>
<tr>
<td>11, M, 62</td>
<td>Colectomy for Ca and resection of metastases in left lung</td>
<td>LLL inf/atel; L pl eff</td>
<td>1.10</td>
<td>8.5</td>
</tr>
<tr>
<td>12, M, 3 mo</td>
<td>Repair of truncus arteriosus</td>
<td>LLL atel/inf and pl eff</td>
<td>1.25</td>
<td>6.2</td>
</tr>
<tr>
<td>13, M, 12</td>
<td>Crouzon’s disease</td>
<td>LLL atel/inf; RUL inf</td>
<td>0.70</td>
<td>6.0</td>
</tr>
<tr>
<td>14, F, 78</td>
<td>AV and MV replacement</td>
<td>LLL inf/atel; bil pl eff</td>
<td>0.50</td>
<td>8.2</td>
</tr>
<tr>
<td>15, M, 7</td>
<td>Fracture of cervical spine</td>
<td>LUL atel/inf; RUL atel</td>
<td>0.10</td>
<td>6.5</td>
</tr>
</tbody>
</table>

*CABG, Coronary arterial bypass graft; MI, mitral incompetence; Ca, carcinoma; VSD, ventricular septal defect; AV, aortic valve; and MV, mitral valve.
†LLL, Left lower lobe; atel, atelectasis; L, left; pl, pleural; eff, effusion; †, elevated; R, right; inf, infiltrate; LUL, left upper lobe; RUL, right upper lobe; bil, bilateral; and LL, lower lobe.
‡Amp, Amplitude; Lat, latency; and NR, no response.

Barré syndrome. In both, the diaphragmatic potential regained normal amplitude over several weeks parallel with the improvement in the patient’s ventilation; however, the latencies of the diaphragmatic responses remained prolonged. The only patient with myasthenia gravis in this series had his diaphragmatic responses decreased in amplitude bilaterally, but the latencies were normal.

The data on 14 patients who developed respiratory insufficiency after a surgical procedure (usually cardiac or thoracic) are shown in Table 1. Four patients (cases 4, 6, 7, and 9) had normal phrenic studies; their respiratory difficulties were apparently due to atelectasis or infiltrates bilaterally (Fig 1). One patient (case 3) had no recordable diaphragmatic potential on stimulation of either phrenic nerve. He required mechanical ventilation until his death three months later. Unilateral diaphragmatic paresis or paralysis was demonstrated in six patients (four with inexcitable left phrenic nerve, one with inexcitable right phrenic nerve, and one with lower amplitude response with left phrenic stimulation), with the diaphragmatic responses being totally normal on the other side (Fig 2). Three patients had evidence of bilateral but asymmetric involvement. In one, the right phrenic nerve was inexcitable, but the left-sided response was present at a reduced amplitude. The remaining two had paralysis of the left hemidiaphragm, with paresis of the right side.

One patient with traumatic quadriplegia due to high cervical cord injury showed evidence of total paralysis on the left with severe paresis of the right hemidiaphragm. The diaphragmatic-evoked response on stimulation of the right phrenic nerve was 0.1 mV, which was believed to be unsatisfactory for diaphragmatic pacing.

**Discussion**

In patients with severe respiratory insufficiency, diaphragmatic paralysis may constitute an important contributing factor to respiratory failure and difficulty in weaning from mechanical ventilatory support. Diaphragmatic contraction contributes 60 percent of the normal minute ventilation, and its paralysis is associated with reduction of vital capacity, maximum voluntary ventilation, forced expiratory volume in one second, and functional residual capacity, and an increased ventilation-perfusion mismatch. In our study of 21 patients with respiratory failure requiring assisted ventilation, 17 showed electrophysiologic evidence of unilateral or bilateral diaphragmatic palsy.

Detection of phrenic nerve paralysis in the past has been largely by radiologic investigations. An elevated...
hemidiaphragm on the chest x-ray film with atelectasis or infiltrates of the lower lobe on the affected side suggests unilateral phrenic paralysis. Fluoroscopic examination of the chest demonstrating reduced excursion of the hemidiaphragm and paradoxical motion is considered diagnostic of diaphragmatic paralysis. Unfortunately, fluoroscopic study may be delayed due to the difficulties in transportation of the critically ill patient to the radiology unit. Furthermore, the radiologic findings are not pathognomonic of phrenic palsy. Similar findings may also occur with eventration of the diaphragm, local pulmonary conditions (infiltrates, atelectasis, effusion, etc), and subphrenic pathologic abnormalities. The radiologic diagnosis of diaphragmatic weakness is, therefore, difficult and often impossible in the presence of coexisting pulmonary pathologic abnormalities. Such a complicating situation is commonly encountered in patients with respiratory failure in the postoperative period following a surgical procedure on the chest (eg, coronary arterial bypass graft). These patients not only show severe pulmonary infiltrates or atelectasis, but are also at risk to develop phrenic nerve damage secondary to surgical trauma, traction, or hypothermia. In two of our patients, radiologic studies raised the suspicion of diaphragmatic paralysis, but electrophysiologic studies indicated normal phrenic-diaphragmatic function.

Although electrophysiologic techniques for studying phrenic nerves and diaphragmatic function have been available since the original work of Davis in 1967, they have not been widely employed in critically ill patients due to lack of a standardized, noninvasive, and reliable technique of phrenic nerve stimulation and recording. The availability of portable electrophysiologic instruments and experience with transcutaneous phrenic stimulation and recording of diaphragmatic potential make this technique feasible and reliable and simple to perform in the intensive care unit. We found that stimulation of the phrenic nerve by surface electrodes was effective and without discomfort. The invasive technique of stimulation through a needle electrode placed close to the phrenic nerve as proposed by some is not necessary.

Both latency and amplitude of the diaphragmatic

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**FIGURE 1.** Normal and symmetric diaphragmatic potentials evoked by right and left phrenic nerve stimulation in four-year-old child four days after repair of congenital heart disease (case 7).

**FIGURE 2.** Absence of diaphragmatic potential on left phrenic stimulation in 78-year-old patient two weeks following aortic and mitral valvular repairs. Diaphragmatic response is normal to right phrenic stimulation (case 14).
potential are important for the diagnosis of diaphragmatic dysfunction. Prolongation of the latency provides unequivocal evidence of slowed conduction along the phrenic nerve, a common finding in polyradiculoneuropathies and in some patients with intraoperative injuries of the phrenic nerves. The amplitude of the diaphragmatic potential, on the other hand, provides a measure of the number of diaphragmatic muscle fibers activated by phrenic nerve stimulation. Abnormalities of amplitude may be the sole evidence of diaphragmatic paresis in patients who develop respiratory insufficiency on the basis of myasthenia gravis and motor neuron disease and also in patients with intraoperative phrenic nerve injuries in the thorax.

Diagnosis of phrenic palsy is not only helpful in understanding the pathophysiology, but also in planning therapy in patients with respiratory insufficiency. Patients with bilateral phrenic dysfunction are likely to need continued positive-pressure ventilation or continuous positive airway pressure for their respiratory support.\textsuperscript{11-14} Unilateral diaphragmatic paralysis causes respiratory insufficiency only if there are other complicating factors such as intrinsic pulmonary or pleural pathologic abnormalities. Correction of these complications is, therefore, crucial in restoring normal ventilation. In patients with diaphragmatic paralysis, plication of the diaphragm is useful.\textsuperscript{13} In patients with traumatic quadriplegia with respiratory insufficiency due to cervical cord injury, phrenic studies are crucial in deciding if diaphragmatic pacing (electrophrenic respiration) would have a place in the management of the patient.\textsuperscript{10} If phrenic nerve stimulation elicits more or less normal diaphragmatic responses, it can be assumed that the anterior horn cells and their axons innervating the diaphragm are intact. Diaphragmatic pacing would be indicated in such patients. On the other hand, if phrenic stimulation produces no response or a low-amplitude response, diaphragmatic pacing would not be beneficial, as was the case in our patient with high cervical cord injury with quadriplegia.

We conclude that transcutaneous phrenic stimulation with surface recording of the diaphragmatic potential is a noninvasive and reliable technique for diagnosis of diaphragmatic dysfunction. The test can be performed easily at the bedside of a critically ill patient with pulmonary insufficiency. Electrophysiologic evaluation of phrenic nerves must be included in the assessment of all patients who develop respiratory insufficiency. When performed with customary radiologic investigation, such evaluation provides useful diagnostic and therapeutic information in such patients.

REFERENCES