AAEM MINIMONOGRAPH #40:
CLINICAL NEUROPHYSIOLOGY
OF THE RESPIRATORY SYSTEM

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CME STUDY GUIDE

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EDUCATIONAL OBJECTIVES

The educational objectives are to learn the indications, details of technique and complications in performing phrenic nerve conduction and needle electromyography of the diaphragm; and to learn the results of the use of these techniques in a variety of diseases which affect the respiratory neuromuscular system.

INSTRUCTIONS

1. The reader should carefully and thoroughly study this minimonograph. If further clarification is needed, the references should be consulted. Do not neglect illustrative material.

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Many neuromuscular disorders affect respiration. This discussion deals mainly with investigations in the critical care unit, but the principles apply equally to patients in other parts of the hospital, to outpatients, and to children, including newborn infants.

The majority of patients in critical care units have an illness severe enough that they require assisted ventilation, many requiring intubation and support by a mechanical ventilator. The reasons for this are often due to primary lung disease or mechanical problems with airways or the chest wall. In a significant number, however, the reasons involve specific dysfunction of the nervous system, either lack of central drive or weakness of the muscles of respiration. The former are induced by a wide variety of encephalopathies and the latter by diseases of anterior horn cells, peripheral nerves, neuromuscular junction, or muscles of the chest wall or diaphragm. These last “neuromuscular conditions” have been traditionally listed as polio, Guillain–Barré syndrome and myasthenia gravis, but our experience is that the neuromuscular complications of sepsis and traumatic damage to the phrenic nerves are the most common causes of neuromuscular problems in the critical care unit.

A lack of central drive due to encephalopathy can often be discerned by observing specific patterns of respiration which are of localizing value (Fig. 1). These clinical signs, however, are often absent or are interfered with by ventilatory assistance. In many instances, it is not possible to determine whether there is a lack of central drive or a neuromuscular problem. The early clinical signs of neuromuscular respiratory dysfunction are rapid, shallow breathing and a rise in blood carbon dioxide levels. In later stages, hypoventilation, hypoxia, and potential apnea prompt assisted ventilation. The observation of “respiratory alternans” (alternation of rib cage and abdominal movement) or “abdominal paradox” (inward movement of the abdominal wall during inspiration), which may suggest neuromuscular respiratory failure, are often absent or are overlooked. More sophisticated measurements such as vital capacity, high airway occlusion pressure, the peak-negative pressure on maximal inspiration from full expiration, breathing frequency, and tidal volume may also provide inconclusive results. Even unilateral damage to the phrenic nerve due to operative trauma is often undiagnosed, despite chest x-rays and fluoroscopy.

It is, unfortunately, not recognized that electromyographic techniques can now be applied which
are of great value in more precisely pinpointing the nervous system cause, if present, for respiratory insufficiency. This minimonograph will describe these techniques and the results of their use in clinical investigation. Our observations over the last 10 years and pertinent articles in the literature are the basis of these statements. The techniques do not address the investigation of disorders causing upper airway dysfunction, i.e., weakness of the facial and bulbar musculature; EMG techniques to investigate this are now being developed.27

TECHNICAL METHODS

Assessing Central Respiratory Drive. Since clinical examination of cerebral function is difficult in the critical care unit, we have found the electroencephalogram (EEG) to be of great value. The nature and degree of abnormality of the EEG will give a strong indication of the severity of the encephalopathy7,35 and, hence, indirect evidence as to whether one might expect a lack of central drive. If the patient is on a ventilator, abnormalities of central drive can be determined by discontinuing intermittent mandatory ventilation, i.e., allowing the patient to trigger the ventilator, but limiting the amount of pressure support from the ventilator with each inspiration as much as possible, but enough to ensure adequate oxygenation. Blood pressure, heart rate, and if possible, venous oxygen tension, should be regularly observed during this period and full ventilation resumed if the patient gets into any difficulties. It may be possible to keep the patient off the ventilator for a number of minutes and observe the pattern of respiration, which may be quite helpful in determining the degree and nature of central drive or the lack of it (Fig. 1). Magnetic or electric stimulation15 of the brain, with recording of the response from the diaphragm, and comparing the latency with that obtained by direct phrenic nerve stimulation, may provide an accurate measure of central conduction.

Phrenic Nerve Conduction Studies. The technique of phrenic nerve conduction26,32 has, unfortunately, been neglected over the years. One of the reasons may be the fear that it was not as accurate as standard conduction studies of limb nerves. We have used a modification of this technique by Markand and colleagues.24 Recent studies in our labo-
ratory have shown that, using this method, the repeatability of diaphragm compound action potential (CAP) measurements is just as good as thenar CAP measurements from median nerve stimulation. 18

Certain technical points, however, must be observed. G1 and G2 surface electrodes are applied as shown in Figure 2, 16 cm apart. These are kept at standard locations, and readjustment is not necessary, except in young children where a shorter, arbitrary distance is used. The phrenic nerve is stimulated with surface electrodes at the posterior border of the sternomastoid muscle, the cathode (placed inferior to the anode) being approximately 3 cm above the clavicle. Electric stimuli of 0.1 or 0.2 ms duration are utilized. Small adjustments in electrode position and voltage are necessary to ensure a supramaximal response. In patients with large necks, stimuli as long as 0.5 ms may be necessary. The neck should be in a neutral or slightly extended position.

The most important technical problem is inadvertent brachial plexus stimulation. This results in a CAP, volume conducted from chest wall muscles innervated by the brachial plexus, that results in an action potential with an initial positive phase and much shorter latency (Fig. 3). Repositioning the stimulus electrode so that only the phrenic nerve is being stimulated will eliminate this problem. The electrocardiogram will produce a high voltage discharge which will interfere with diaphragm CAP recording. This discharge is always quite obvious and should be ignored. Simply repeat the stimulus to obtain the uncontaminated diaphragm CAP. The diaphragm CAP amplitude also varies with respiration and electrode placement. With our method, the amplitude is greatest during inspiration; however, with Swenson's method, in which the recording electrode is on the costal margin, and the reference electrode below the umbilicus, the amplitude is greatest on expiration. 33 If supramaximal stimulation of the phrenic nerve is repeated several times during quiet respiration and the two highest amplitude potentials obtained, however, amplitude measurements are quite repeatable (Fig. 3) and the phases of respiration are not a factor. Averaging is not necessary. The distance from the point of stimulation on the phrenic nerve to the G1 recording electrode over the xiphoid is routinely measured, although it has been

FIGURE 2. The techniques of phrenic nerve conduction and needle EMG of chest wall and diaphragm. The phrenic nerve is stimulated (s) at the posterior border of the sternomastoid muscle. The diaphragm compound action potential is recorded from ipsilateral surface electrodes (G1 and G2). Needle EMG of the chest wall and diaphragm can be recorded with a monopolar electrode inserted at right angles to the chest wall in one of several interspaces (x) between the anterior axillary and medial clavicular lines. There is at least 1.5 cm between the pleura above and the lower costal margin below, on which the diaphragm inserts. The presence of insertional activity indicates when the needle is in muscle. Bursts of motor unit potentials characteristically occur with each inspiration when the needle is in the diaphragm. (With permission, Bolton, CF.4)

FIGURE 3. Phrenic nerve conduction in a healthy subject. The upper tracings show the result obtained when the phrenic nerve only was supramaximally stimulated and the two best diaphragm compound action potential results recorded. The lower tracings show the results when the nearby brachial plexus was stimulated, not the phrenic nerve; the volume conducted compound action potential, of shorter latency with initial positive wave, comes from chest wall muscles supplied by the brachial plexus; thus, brachial plexus stimulation should be avoided (calibration: 10 ms and 200 µV per division).
found that this measurement does not vary much in adults. In children, there would be a considerable effect and here the measurement becomes more important. Our control values for phrenic nerve conduction have been similar for adults to those of Markand and colleagues (latency, 6.3 ± 0.8 ms; amplitude, 597 ± 139 μV; mean ± SD).

**Intercostal Nerve Conduction.** Intercostal nerve conduction may also be of value, but we have found the marked variability of the CAP from the rectus abdominus muscle requires multiple recording sites. Thus, it is a time-consuming technique we rarely use.

**Needle Electromyography of the Diaphragm.** We had originally believed that needle electromyography of the diaphragm was too risky for fear of inadvertent puncturing of lung, liver, spleen, or colon.

The techniques of Goodgold, Saadeh and colleagues in which the needle is inserted through the abdominal wall and angulated upward under the costal margin, was technically difficult in our hands. Therefore, we recently developed a technique, briefly described many years ago by Koepeke, which is safe, causes little discomfort, and gives excellent recordings of diaphragm activity. It consists of introduction of the recording needle through any interspaces between the anterior axillary and medial clavicular lines (Fig. 2). The needle should be introduced just above the costal margin, where there is an approximately 1.5 cm distance between the pleural reflection and the lower costal cartilage upon which the diaphragm inserts (Fig. 4). Thus, the needle does not traverse either the pleural space or the lung. Recordings can be made as the needle passes through external oblique or rectus abdominus muscles, external and internal intercostal muscles, and finally, diaphragm. With quiet respiration, the chest wall muscles do not fire, or only a few units fire (Fig. 5), but will do so with coughing or twisting of the trunk. There is regular firing from the diaphragm.

![Figure 4: Anatomy of needle electromyography of the diaphragm. The various structures the needle traverses in reaching the diaphragm are shown. The distance between the cartilaginous lower costal margin and the pleural reflection is approximately 1.5 cm. Note in quiet respiration the lung is well removed from the path of needle insertion. Entry of the needle into the peritoneum is signaled by pain and loss of muscle insertional activity (the reference electrode is shown applied to the skin near the monopolar needle).](image-url)
Koepke technique of needle EMG of the diaphragm. A telephone survey of those currently using the technique indicates that among 1000 subjects, only 2 have had this complication. Both were patients with severe, chronic obstructive pulmonary disease and on ventilators who responded promptly to treatment. No instances have occurred in outpatients. Nonetheless, we recommend all persons be observed for a period of 1 h following this procedure to observe pain, shortness of breath, increased heart rate and blood pressure, or decreased breath sounds on auscultation, as early signs of pneumothorax. If present, prompt hospital admission and emergency treatment is indicated.

RESULTS IN CLINICAL INVESTIGATION

Electromyographic Results in Specific Disorders of the Neuromuscular Respiratory System. Encephalopathy. Dysfunction of the various parts of the brain may induce patterns of respiration that are specific for that area (Fig. 1). These patterns may be difficult to detect clinically, particularly if the patient is on a ventilator. In encephalopathy, electromyographic techniques will disclose normal phrenic nerve conduction and no abnormal spontaneous activity in chest wall or diaphragm. However, with the patient briefly off intermittent mandatory ventilation but kept on pressure support to provide adequate oxygenation, the pattern of firing of MUPs may disclose these specific patterns. Absent firing suggests a total lack of central drive, as may occur in extremely severe encephalopathies or in developmental anomalies, such as anencephaly.

Spinal Cord and Nerve Roots. Primary disorders at segmental levels, C-3, -4, and -5 may affect the phrenic nerve unilaterally or bilaterally and will typically cause paradoxic respiration; with inspiration, outward movement of the chest occurs but there is an absence of movement or actual inward movement of the abdomen. With dysfunction at thoracic levels, which causes dysfunction of nerves to chest wall muscles, the opposite may occur; that is, with inspiration there will be a failure of chest wall movement or even an inward movement, but there will be an outward movement of the abdomen. If there is dysfunction at both high cervical and thoracic levels, all respiratory movements will be weak and the breathing will tend to be rapid and shallow. These specific clinical signs may be absent, however, particularly if the patient is on a ventilator. The types of disorders that primarily affect these structures are quite varied and include

FIGURE 5. Needle EMG from the diaphragm in a healthy person deliberately breathing at the rate of 20 per minute. (a) With a sweep speed of 200 ms/division the pattern of firing of motor unit potentials can be discerned. In this raster mode, continuous sweeps show inspiratory bursts of motor unit potentials, alternating with expiratory intervals relatively free of activity. (b) At a 10 ms/division sweep, the morphology of motor unit potentials is evident. Again, there is little activity during expiration. Diaphragm motor unit potentials are normally smaller, and more numerous, than units in chest wall or limb muscles (calibration: 200 μV/division).
trauma, neoplasm, syringomyelia, and compression of nerve roots by intervertebral disc or trauma.

EMG techniques will show in high cervical cord or nerve root lesions that on phrenic nerve conduction, CAPs from the diaphragm will be reduced or absent, unilaterally or bilaterally, but the latencies will be normal or near normal. Fibrillation potentials and positive sharp waves will appear as signs of denervation from one or both hemidiaphragms and MUPs will fire in decreased numbers, or will not fire at all if denervation is complete. In instances of damage to the thoracic spinal cord or nerve roots, we have not found intercostal nerve conduction studies to be of value because they are time-consuming and technically difficult. However, needle EMG of chest wall muscles may show patterns of denervation which will localize levels of anterior horn cell dysfunction within the spinal cord.

If there is denervation of lumbar, thoracic, or cervical paraspinal muscles, or upper limb or chest wall muscles at the same segmental levels in cases of upper cervical or lower thoracic cord dysfunction there will be confirmation that the lesion is intraspinal, although the lack of denervation does not entirely exclude an intraspinal lesion. Thus, in a patient who is quadriplegic due to a high-cervical cord lesion and in respiratory failure, phrenic nerve conduction will disclose low amplitude or absent diaphragm CAP amplitudes with denervation of the diaphragm on needle electromyography, but denervation will also be present in cervical paraspinal muscles at C-3, -4 and -5 levels. Denervation may also be found in the upper limb muscles—trapezius, levator scapulae, and deltoid—which have prominent C-4 and -5 innervation.

Amyotrophic Lateral Sclerosis. These techniques are valuable in the investigation of this large group of patients who may present for the first time with respiratory insufficiency. The involvement may be either upper or lower motor neurons and at high cervical or thoracic levels. The same principles apply as with other primary disorders of the spinal cord affecting the anterior horn cells. We have found needle EMG studies to be particularly valuable. For example, needle EMG of the diaphragm may disclose abundant, abnormal spontaneous activity with a decreased number of relatively normal-sized MUPs suggesting acute denervation and rapidly developing disease. On the other hand, in more chronic denervation there may be little abnormal spontaneous activity but a decreased number of MUPs, the remaining ones being quite large as a result of collateral reinnervation. Thus, EMG techniques may be of some prognostic value and aid in long-term planning of respiratory management in patients with amyotrophic lateral sclerosis. Moreover, when combined with EMG studies of limb nerve and muscle, it will usually be possible to determine that amyotrophic lateral sclerosis is, in fact, the cause of the respiratory insufficiency and it is not due to a variety of other neuromuscular conditions which may present primarily with respiratory failure.

Traumatic or Compressive Phrenic Nerve Palsies. Lower motor neuron damage to the phrenic nerves may occur anywhere along their course, from the anterior horn cells at C-3, -4, and -5 levels to the terminal innervation of the diaphragm. Strictly unilateral lesions are almost always of traumatic or compressive origin, due to neoplasm or direct trauma of an accidental or surgical nature. Operative procedures are relatively common causes of the phrenic nerve being damaged, either directly by compression by retractor or other instruments or the application of cold during cardiac surgery.¹² In the procedure of liver transplantation, the phrenic nerve may be traumatized when it is inadvertently clamped, along with the clamping of the inferior vena cava (personal communication, W.F. Brown). Occasionally, both phrenic nerves are traumatized by operative procedures. Laguenuy and colleagues²² report 4 cases of presumed neuralgic amyotrophy in which phrenic nerve conduction and needle EMG of the diaphragm disclosed that the phrenic, as well as limb, nerves had been affected.

The phrenic nerve may be unilaterally damaged due to disc herniation⁸ or trauma at birth, affecting roots C-3 and -4. Such unilateral damage may not be obvious clinically, even after chest x-ray and fluoroscopy and can only be disclosed by EMG techniques.

Electrophysiologic techniques will show a reduced amplitude of CAP from the diaphragm with relatively normal latency. Abnormal spontaneous activity may be present in the affected diaphragm in acute cases and MUPs will be reduced in number with firing during inspiration. In chronic cases, abnormal spontaneous activity may not be present due to collateral reinnervation and MUPs firing during inspiration will be decreased in number but unusually large. The site of damage along the course of the phrenic nerve can be localized by performing needle electromyography of the levator scapulae and cervical paraspinal muscles.

Polyneuropathy. Experience in doing phrenic nerve conduction studies and needle electromyography on outpatients with a variety of neuromus-
cular disorders indicates that the phrenic nerves are commonly affected in many polyneuropathies. In some instances, this involvement is subclinical. For example, in the familial polyneuropathy of hereditary motor and sensory neuropathy, type 1, there may be no symptoms or signs of respiratory insufficiency, yet phrenic nerve conduction studies reveal considerably prolonged latencies with relative preservation of the diaphragm CAP, as would be expected in a diffuse demyelinating polyneuropathy. More severe involvement will occur with more severe polyneuropathies and, if they affect the phrenic nerves severely, respiratory insufficiency occurs and, in such instances, there will be abnormalities consistent with the type of neuropathy. For example, in porphyric polyneuropathy, the findings are typical of a relatively pure axonal degeneration of phrenic nerve fibers. Phrenic nerve conduction latencies will be near normal but muscle CAP amplitudes from the diaphragm will be depressed (Fig. 6). There will be positive sharp waves and fibrillation potentials on needle electromyography of the diaphragm, and few, if any, units, may fire during inspiration (Fig. 7). In chronic denervation, there will be no abnormal spontaneous activity, simply a reduced number of MUPs which are large and polyphasic. Such abnormalities of chest wall muscles will confirm these as a contributing cause to respiratory insufficiency.

The following is a brief discussion of critical illness polyneuropathy and Guillian–Barré syndrome, polyneuropathies that commonly lead to respiratory insufficiency and the necessity of assisted ventilation in the critical care unit.

**Critical Illness Polyneuropathy.** This is a complication of sepsis and multiple organ failure which occurs in up to 50% of patients in major medical or surgical critical care units. It often presents with difficulty in weaning from the ventilator, just as septic encephalopathy and other manifestations seem to be improving. Clinical signs are often absent and electrophysiologic studies are necessary for diagnosis. These show the presence of a primary, predominantly motor, axonal polyneuropathy. If sepsis and multiple organ failure can be brought under control, as occurs in 50% of patients, recovery from the polyneuropathy will occur unless it is unusually severe. The cause is thought to be a fundamental defect in the septic syndrome.

Electrophysiologic studies have been invaluable in establishing this polyneuropathy as the cause of difficulty in weaning from mechanical ventilation.

**FIGURE 6.** Phrenic nerve conduction in porphyric neuropathy at 6 weeks following onset. Upper trace is right, and lower trace is left phrenic nerve conduction. Latencies are normal (CAP onset marked by short vertical lines), but amplitudes are markedly reduced, typical of a pure axonal degeneration. (Note large EKG artifact at the end of the upper trace) (calibration: 10 ms and 100 μV per division).

**FIGURE 7.** Needle EMG of the diaphragm in porphyric neuropathy, the same patient as in Figure 6. Axonal degeneration of phrenic nerves occurred rapidly. Thus, there were moderate numbers of fibrillation potentials and positive sharp waves at 1 week (a), but they were abundant at 5 weeks (b). No motor unit potentials fired during inspiration at either time (calibration: 10 ms and 100 μV per division).
Phrenic nerve conduction studies show near-normal latencies but reduced CAP amplitudes from the diaphragms. Such amplitudes have shown a correlation with abnormalities of EMG studies of limb muscles. Needle EMG of the diaphragm may show fibrillation potentials and positive sharp waves as a sign of denervation and MUPs will fire in decreased numbers, either due to the polyneuropathy or the associated septic encephalopathy. No clearcut abnormalities in the morphology of MUPs have yet been defined. Needle EMG of chest wall muscles may show denervation, as well as the diaphragm. In some instances, critical illness polyneuropathy may show predominant involvement of limb, phrenic, or intercostal nerves, so that all three areas should be studied to gain a complete picture of the severity and distribution of the polyneuropathy. This will provide valuable information on long-term prognosis for recovery and for respiratory management.

**Guillain–Barré Syndrome.** We have found these techniques valuable at all stages of this syndrome. In the acute stage, abnormalities may herald the necessity for assisted ventilation. In the later stages, they give some idea as to the prognosis for recovery from the neuromuscular respiratory insufficiency. Phrenic nerve conduction latencies may be near-normal in the early stages of Guillain–Barré syndrome and tend to become more prolonged, sometimes severely prolonged, in the later stages. Muscle CAP may be reduced in amplitude and dispersed (Fig. 8).

Needle EMG of the diaphragm may reveal fibrillation potentials and positive sharp waves if there is a significant amount of axonal degeneration in the phrenic nerve. If it is relatively pure demyelination, however, no abnormal spontaneous activity will be found and there will simply be a reduced number of MUPs firing with each inspiration (Fig. 9). With some patients, there has been much more severe involvement of one phrenic nerve than the other, or the intercostal nerves may be predominantly affected. Needle EMG of chest wall muscles may show abnormalities similar to that of the diaphragm. Crisafulli and colleagues, in a preliminary report, have found these results may aid prognostication, patients with predominantly demyelination of phrenic nerves recovering earlier from ventilator dependence.

**Neuromuscular Transmission Disorders.** These techniques may be usefully applied to this category of disease. However, repetitive phrenic nerve conduction studies may be somewhat uncomfortable and difficult to interpret since the diaphragm CAP amplitude normally varies with the phase of respiration, or may be interfered with by the higher voltage and longer duration electrocardiographic potential. Thus, we have not used the technique in our patients, nor, to our knowledge, have others. Repetitive stimulation of limb nerves, however, will usually disclose a defect in neuromuscular transmission and provide indirect evidence that the respiratory neuromuscular system is also involved; such studies should be performed in all suspected

**FIGURE 8.** Phrenic nerve conduction in Guillain–Barré syndrome of 3 months' duration. Note the prolonged latency, twice normal, and the reduced amplitude of the diaphragm compound action potential (calibration: 10 ms and 200 μV per division).

**FIGURE 9.** Needle EMG of the diaphragm in Guillain–Barré syndrome, the same patient as in Figure 8. There was no abnormal spontaneous activity, and only a few motor unit potentials fired with each inspiration. These units were low amplitude, suggesting distal nerve block (calibration: 10 ms and 200 μV per division).
cases of neuromuscular transmission defect: myasthenia gravis, Lambert–Eaton syndrome, botulism, the effects of neuromuscular blocking agents, etc.

Phrenic nerve conduction and needle EMG of the diaphragm may provide other useful information, as illustrated by a case of myasthenia gravis which had denervation of the diaphragm and chest wall muscles.25

Primary Myopathies. Our experience with this group of neuromuscular diseases has been limited but the results, in the main, are what would be expected from previous knowledge of electrophysiologic studies of limb nerve and muscle. In primary myopathies, the latency of phrenic nerve conduction is normal but muscle CAP amplitudes from the diaphragm may be normal or reduced. Needle electromyography of the diaphragm may show abnormal spontaneous activity such as myotonic discharges in patients with myotonic dystrophy; however, analysis of morphology of MUPs is difficult because, normally, these potentials in the diaphragm are unusually numerous and of relatively small amplitude and duration (Fig. 5). This, coupled with the fact that the patient is not easily able to control the activation of MUPs with inspiration makes the detection of so-called myopathic units, low-amplitude, short-duration, or polyphasic MUPs, particularly difficult. Certain types of computer analysis may help solve this problem.

Difficulty in Weaning from the Ventilator. This has traditionally been attributed to still poorly defined metabolic and nutritional disturbances30 or the phenomenon of diaphragmatic fatigue.30 By utilizing the techniques of phrenic nerve conduction and needle electromyography of diaphragm and chest wall muscles, however, we have determined that clearly identifiable causes were critical illness polyneuropathy, the most common cause, unilateral or bilateral damage to the phrenic nerves from surgical or other trauma, a primary myopathy, or a lack of central drive (Maher, Bolton, and colleagues, unpublished data). Since difficulty in weaning from the ventilator remains a serious problem in critical care units and greatly adds to the expense of patient care, there is a strong indication to utilize these EMG techniques to aid long-term prognosis and respiratory management.

FUTURE DIRECTIONS
As interest grows in studying the respiratory system by electrophysiologic techniques, there will be, undoubtedly, modifications of the above techniques and development of newer approaches. It is possible that many cases of acute respiratory insufficiency have an underlying central or peripheral nervous system cause, and it would be worthwhile to know that at the onset of management so that decisions regarding intubation, type of ventilation, and so forth, could be made more effectively. Further information may also be gained by needling other chest wall muscles, such as higher intercostal muscles and pectoralis muscles, to gain a better idea of the pattern of activation of the respiratory muscles in different disease states. Finally, methods of testing central conduction, particularly magnetic stimulation of the brain, spinal cord, and possibly peripheral nervous systems, may add further important information regarding abnormalities of central versus peripheral conduction.

REFERENCES


