Assessment of Respiratory Function In the Intensive Care Unit

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Abstract: Disorders of both the central and peripheral nervous systems are important causes of respiratory insufficiency. However, simple clinical observations and pulmonary function measurements may fail to identify the location and type of disorder. This can often be accomplished by the newly-developed technique of phrenic nerve conduction and needle electromyography of the diaphragm which delineate the various disturbances of central drive, axonal or demyelinating neuropathies of the phrenic nerves and certain myopathies. These studies have been preformed safely and with little discomfort on adults, children or infants, and in out-patient and general ward settings. We have found they are of particular value in the intensive care unit.

Résumé: Évaluation de la fonction respiratoire à l'unité de soins intensifs. Les affections du système nerveux central et périphérique sont des causes importantes d'insuffisance respiratoire. Cependant, la localisation et le type de problème peuvent échapper à l'observation clinique simple et aux mesures de fonction respiratoire. On peut y pallier par la nouvelle technique d'évaluation de la conduction au niveau du nerf phrénique et l'électromyographie du diaphragme qui cernent les différentes perturbations d'origine centrale, les neuropathies axonales ou démyélinisantes des nerfs phréniques et certaines myopathies. Ces études ont été réalisées de façon sécuritaire et sans trop d'inconfort chez les adultes, les enfants et les nourrissons, comme patients externes ou hospitalisés dans une unité de soins généraux. Nous avons constaté qu'ils sont d'une grande utilité à l'unité de soins intensifs.

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This paper deals mainly with investigations of neuromuscular disorders affecting respiration in the critical care unit,[1] but the principles apply equally to patients in other parts of the hospital, to out-patients, and to children, including newborn infants.

Patients in critical care units require assisted ventilation because of primary lung or heart disease, or mechanical problems with airways or the chest wall. In others, there is specific dysfunction of the nervous system, either lack of central drive, due to a wide variety of encephalopathies, or weakness of the muscles of respiration due to diseases of anterior horn cells, peripheral nerves, neuromuscular junction or muscles of the chest wall or diaphragm.[2]

It has long been held that a lack of central drive due to encephalopathy may be determined by observing specific patterns of respiration which are of localizing value[3] (Figure 1). However, these are often absent or are interfered with be 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, hypoxemia and potential apnea prompt assisted ventilation. "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, are overlooked or may be seen in severe chronic obstructive lung disease. Vital capacity, high airway occlusion pressure, the peak negative pressure on maximal inspiration from full expiration, breathing frequency and tidal volume[4] may also provide inconclusive results. Even unilateral damage to the phrenic nerve due to operative trauma[5] remains undiagnosed, despite chest x-rays and fluoroscopy.

Electromyographic techniques can now be applied which are of great value in more precisely pinpointing the nervous system cause, if present, for respiratory insufficiency. The techniques discussed here do not address the investigation of disorders causing upper airway dysfunction; i.e., weakness of the facial and bulbar musculature. Techniques to investigate this are now being developed.[6]

Technical Methods

Central Respiratory Drive

If the patient is on a ventilator, abnormalities of central drive can be determined by discontinuing intermittent mandatory ventilation and allowing the patient to trigger the ventilator, but

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limiting the amount of pressure support from the ventilator with each inspiration, leaving enough to ensure adequate oxygenation. Blood pressure, heart rate, and if possible, oximetry should be regularly observed during this period and full ventilation resumed if necessary. 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 (Figure 1). Magnetic or electrical stimulation[7] 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 conduction[8,9] has, unfortunately, been neglected, perhaps from fear that it was not as accurate as standard conduction studies of limb nerves. By using the technique of Markand and colleagues[10] and observing certain technical considerations[11] we have shown that the repeatability of diaphragm compound action potential measurements is just as good as thenar compound action potential measurements from median nerve stimulation.[12]

Intercostal Nerve Conduction

Intercostal nerve conduction[13] may also be of value, but we have found the marked variability of the compound action potential 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.[14]

The techniques of Goodgold[15] and Saadeh et al.,[16] 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 Koepe,[17,18] which is safe, causes little discomfort and gives excellent recordings of diaphragm activity.[19] It consists of introduction of the recording needle through any interspaces between the anterior auxillary and medial clavicular lines, just above the coastal margin (Figure 2), between the pleural reflection and the lower costal cartilage upon which the diaphragm inserts (Figure 3). 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 (Figure 4), but will do so with coughing or twisting of the trunk. There is regular firing from the diaphragm with each inspiration and the firing pattern and other features of motor unit potentials can be observed (Figure 4). Motor unit potentials in the diaphragm are of shorter duration and smaller amplitude, but more numerous than chest wall muscles, suggesting a relatively low innervation ratio. Abnormal spontaneous activity can be detected in these various muscles, including the diaphragm.

In the critical care unit, in ventilated patients, we temporarily discontinue intermittent mandatory ventilation and reduce pressure support to a level which will just overcome ventilator/airway

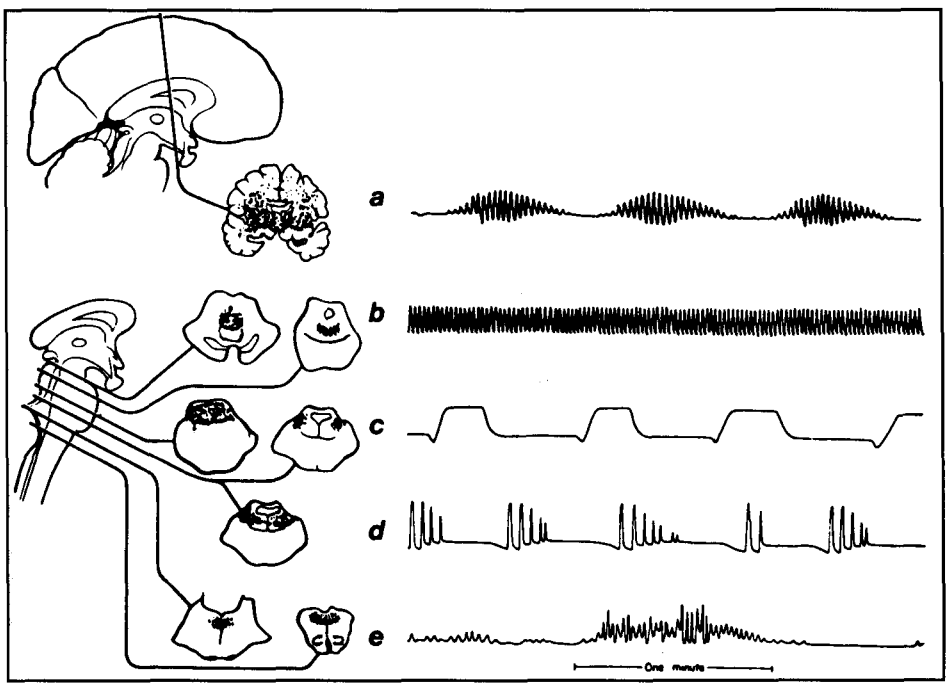

Figure 1: *Abnormal respiratory patterns associated with pathologic lesions (shaded areas) at various locations in the brain.* ***(a)*** *Cheyne-Stokes respiration;* ***(b)*** *central neurogenic hyperventilation;* ***(c)*** *apneusis;* ***(d)*** *cluster breathing; and,* ***(e)*** *ataxic breathing. Here respiratory movements were recorded with a chest-abdomen pneumograph, but we have found such patterns can be detected from needle EMG recordings of the diaphragm. (With permission, Plum and Posner.[3])*

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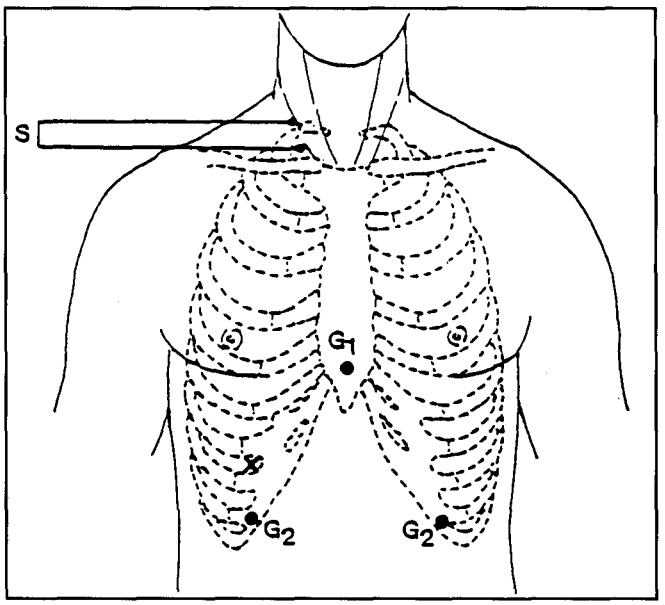

Figure 2: The techniques of phrenic nerve conduction and needle EMG of chest wall and diaphragm. The phrenic nerve is simulated (s) as the posterior border of the sternomastoid muscle. The diaphragm compound action potential is recorded from ipsilateral surface electrodes (G_1 and G_2). Needle EMG of 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.[1])

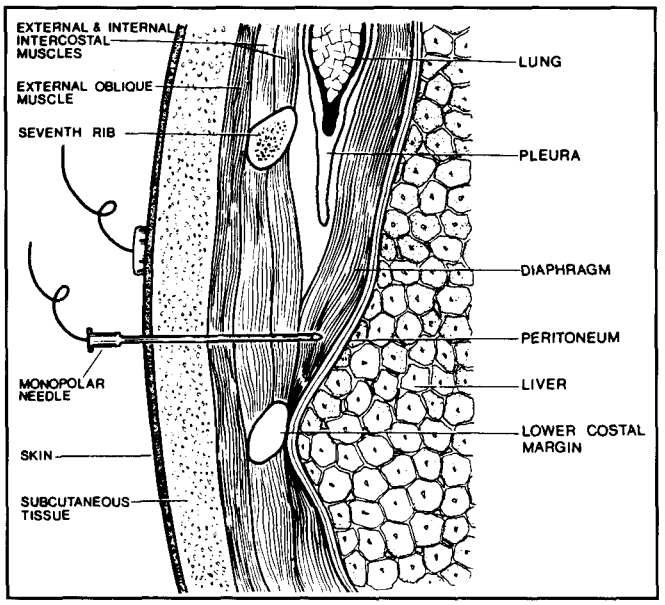

Figure 3: Anatomy of needle electromyography of the diaphragm. The various structures the needle traverses in reaching the diaphragm are shown. The distance between the cartilaginous lower costal margin and the pleural reflection is approximately 1.5 cm. Note in quiet respiration the lung is well removed from the path of needle insertion. With entry into the diaphragm, bursts of motor unit potentials occur with each inspiration. (The reference electrode is shown applied to the skin near the monopolar needle.) (With permission, Bolton.[11])

resistance (approximately 8-10 cm H_2O). While the patient is being closely monitored, recordings from the diaphragm are made during voluntary respirations. This brief respiratory challenge is often enough to increase diaphragmatic drive, thereby allowing an assessment of diaphragmatic activity. If there is total denervation, motor units will not fire in the diaphragm, and an important sign that one is in the diaphragm will be lost. The presence or absence of insertional activity and knowledge of the local anatomy must then be used to define location.

Pneumothorax is a rare complication of the Koepke technique of needle EMG of the diaphragm. Among 1,000 subjects tested to date, only 2 have had this complication.[20] Both were patients with severe, chronic obstructive pulmonary disease and on ventilators who responded promptly to treatment. No instances have occurred in out-patients. Nonetheless, we recommend all persons be observed for a period of one hour following this procedure, to observe pain, shortness of breath, increased heart rate and blood pressure, or decreased breath sounds on ausculation, as early signs of pneumothorax. If present, prompt hospital admission and emergency treatment is indicated.

Results

Encephalopathy

Patterns of respiration may be specific for that area of brain affected (Figure 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 motor unit potentials may disclose these specific patterns. Absent firing suggests a total lack of central drive, as may occur in extremely severe encephalopathies.

Spinal Cord and Nerve Roots

Primary disorders at segmental levels, C3, 4 and 5 may affect the phrenic nerve unilaterally or bilaterally and will typically cause paradoxical respiration; with inspiration, outward movement of the chest occurs but there is an absence of movement or acutal 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. The disorders that affect these structures include trauma, neoplasm, syringomyelia and compression of nerve roots by intervertebral disc.

In high cervical cord or nerve root lesions, compound action potentials 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 motor unit potentials will fire in decreased numbers, or will not fire at all if denervation is complete. Needle EMG of chest wall muscles may show patterns of denervation which will localize levels of anterior horn cell dysfunction within the spinal cord.

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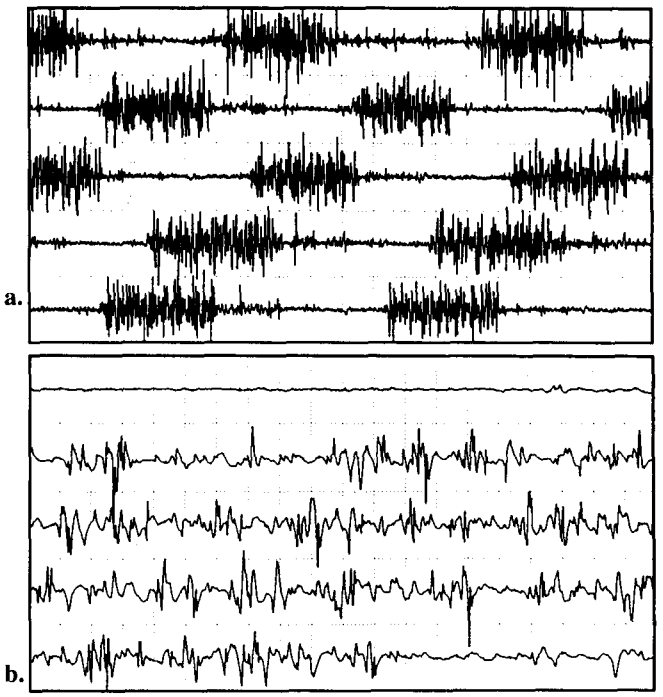

Figure 4: Needle EMG from the diaphragm in a healthy person, deliberately breathing at the rate of 20/s. (a) With a sweep speed of 200 ms/division the pattern of firing of motor unit potentials can be discerned. In this faster mode, continuous sweeps show inspiratory bursts of motor unit potentials, 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.) (With permission, Bolton.[11])

In upper cervical or lower thoracic cord dysfunction, denervation of lumbar, thoracic or cervical paraspinal muscles, or upper limb or chest wall muscles at the same segmental levels, will confirm 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 may disclose low amplitude or absent diaphragm compound action potential amplitudes and needle electromyography will disclose denervation of the diaphragm, cervical paraspinal muscles at C3, 4, 5 levels, and trapezius, levator scapulae and deltoid – which have prominent C4 and 5 innervation.

Amyotrophic Lateral Sclerosis

These patients may present for the first time with respiratory insufficiency. The involvement may be of 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 upper or lower motor neurones. Needle EMG studies are particularly valuable. Thus, needle EMG of the diaphragm may disclose abundant, abnormal spontaneous activity with a decreased number of relatively normal-sized motor unit potentials, suggesting acute denervation and rapidly developing disease. In chronic denervation there may be little abnormal spontaneous activity but a decreased number of motor unit potentials, the remaining ones being quite large as a result of collateral reinnervation. These results are of prognostic value and aid in longterm planning of respiratory management. 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.

Traumatic or Compressive Phrenic Nerve Palsies

Damage to the phrenic nerves may occur anywhere along their course, from the anterior horn cells at C3, 4, 5 to the terminal innervation of the diaphragm. Unilateral lesions are usually due to neoplasm or direct trauma of an accidental or surgical nature. In operative procedures, the phrenic nerve is damaged by retractors or other instruments, or the application of cold during cardiac surgery.[21] In liver transplantation, the phrenic nerve may be traumatized when it is inadvertently clamped, along with the inferior vena cava (personal communication, W.F. Brown). Occasionally, both phrenic nerves are traumatized by operative procedures. Neuralgic amyotrophy is another cause.[22] The phrenic nerve may be unilaterally damaged due to disc herniation[23] or trauma at birth, affecting roots C3 and 4.

Unilateral damage may not be obvious clinically, even after chest x-ray and fluoroscopy and can only be disclosed by EMG techniques. Electrophysiological techniques will show a reduced amplitude of compound action potential from the diaphragm with relatively normal latency. Abnormal spontaneous activity may be present in the affected diaphragm in acute cases and motor unit potentials will be reduced in number with firing during inspiration. In chronic cases, abnormal spontaneous activity may not be present due to collateral reinnervation and motor unit potentials 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

The phrenic nerves are commonly affected in many polyneuropathies. The involvement may be subclinical. In hereditary motor and sensory neuropathy, Type I, despite the absence of symptoms of signs of respiratory insufficiency, phrenic nerve conduction studies reveal considerably prolonged latencies with relative preservation of the diaphragm compound action potential, as would be expected in a diffuse demyelinating polyneuropathy.[24] In porphyric polyneuropathy, the findings are typical of a relatively pure axonal degeneration of phrenic nerve fibers. Phrenic nerve conduction latencies are near normal, but muscle compound action potential amplitudes from the diaphragm are depressed (Figure 5). Positive sharp waves and fibrillation potentials are present in the diaphragm, and few units fire during inspiration (Figure 6). In chronic denervation, there is no abnormal spontaneous activity, simply a reduced number of motor unit potentials which are large and polyphasic. Abnormalities of chest wall muscles confirm muscles are contributing to respiratory insufficiency.

Critical illness polyneuropathy and Guillain-Barré syndrome commonly lead to respiratory insufficiency and the necessity of assisted ventilation in the critical care unit.

Critical Illness Polyneuropathy

This complication occurs in 70% of patients with sepsis and multiple organ failure, which occurs in 50% of patients in major

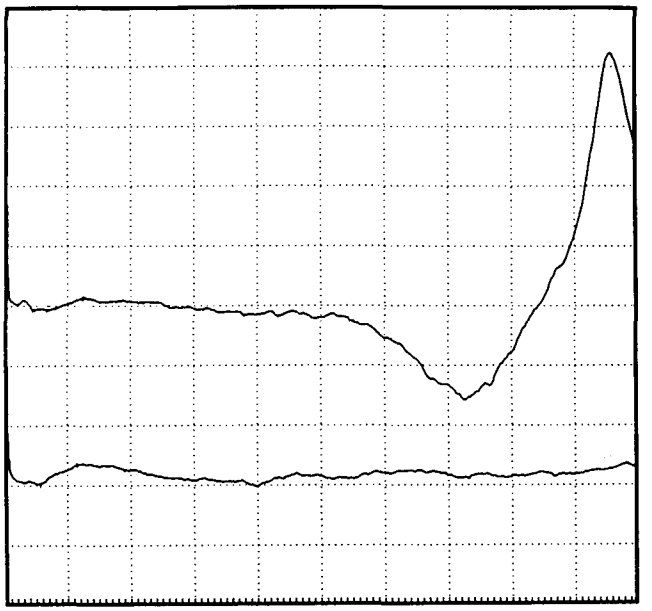

Figure 5. *Phrenic nerve conduction in porphyric neuropathy at 6 weeks following onset. Upper trace is right, and lower trace is left phrenic nerve conduction. Latencies are normal (CAP onset marked by short vertical lines), but amplitudes are markedly reduced, typical of a pure axonal degeneration. (Note large EKG artifact at the end of the upper tract.) (Calibration: 100 ms and 100 μV per division.) (With permission, Bolton.[11])*

medical or surgical critical care units. It often presents with difficulty in weaning from the ventilator,[25-27] just as septic encephalopathy and other manifestations seem to be improving. Clinical signs are often absent and electrophysiological 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.[27,28]

Phrenic nerve conduction studies show near-normal latencies but reduced compound action potential amplitudes from the diaphragms. Such amplitudes have shown a correlation with abnormalities of EMG studies of limb muscles.[28] Needle EMG of the diaphragm may show fibrillation potentials and positive sharp waves as a sign of denervation and motor unit potentials will fire in decreased numbers, either due to the polyneuropathy or the associated septic encephalopathy. No clearcut abnormalities in the morphology of motor unit potentials have yet been defined. Needle EMG of chest wall muscles may also show denervation. 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 longterm prognosis for recovery, and for respiratory management.

Guillain-Barré Syndrome

These techniques are valuable at all stages of this syndrome. In the acute stage, abnormalities may herald the necessity for assisted ventilation. In the later stages, they may predict recovery from respiratory insufficiency.[29] Phrenic nerve conduction

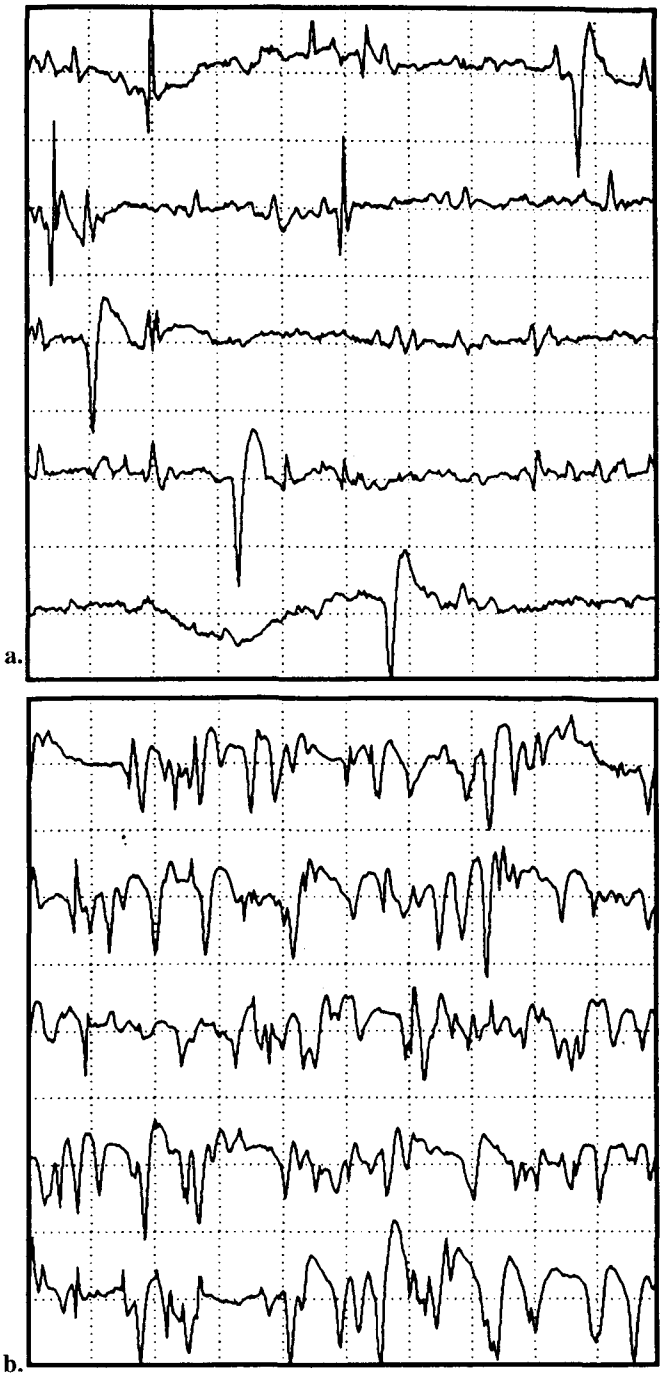

Figure 6: *Needle EMG of the diaphragm in porphyric neuropathy, the same patient as in Figure 5. Axonal degeneration of phrenic nerves occurred rapidly. Thus, there were moderate numbers of fibrillation potentials and positive sharp waves at one week* ***(a)****, but they were abundant at 5 weeks* ***(b)****. No motor unit potentials fired during inspiration at either time. (Calibration: 10 ms and 100 μV per division.) (With permission, Bolton.[11])*

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.[10] Muscle compound action potentials may be reduced in amplitude and dispersed (Figure 7).

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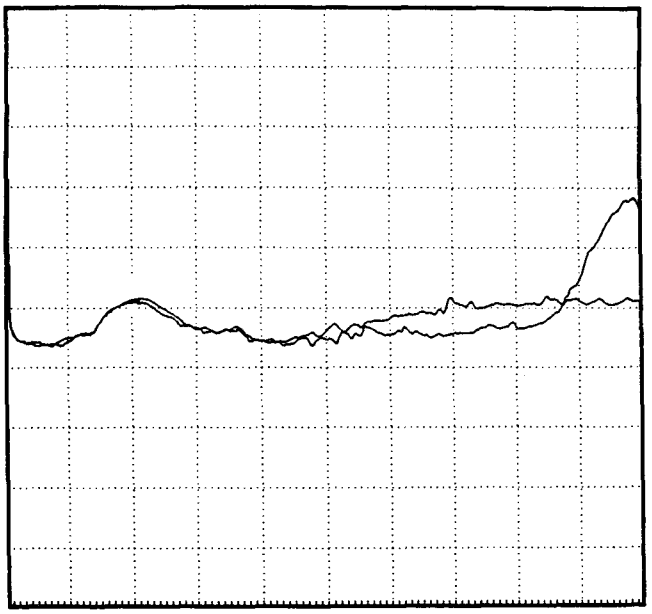

Figure 7: Phrenic nerve conduction in Guillain-Barré syndrome of 3 months' duration. Note the prolonged latency, twice normal, and the reduced amplitude of the diaphragm compound action potential. (Calibration: 10 ms and 200 μV per division.) (With permission, Bolton.[11])

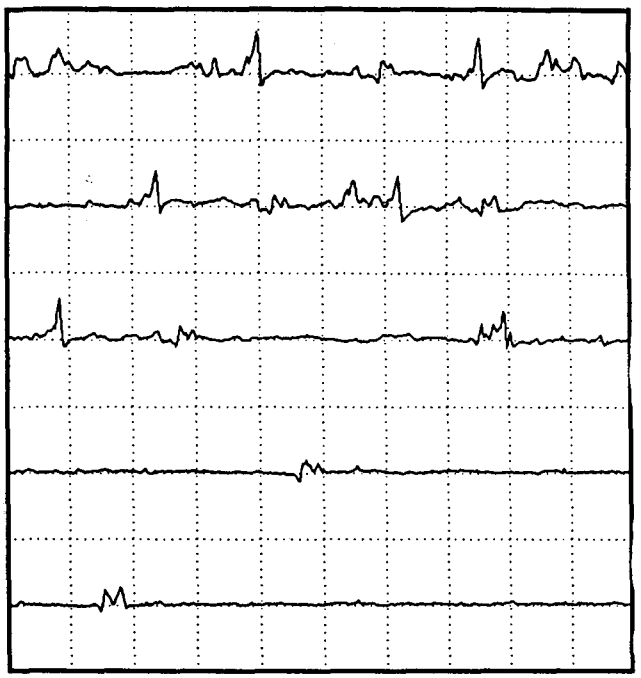

Figure 8: Needle EMG of the diaphragm in Guillain-Barré syndrome, the same patient as in Figure 7. There was no abnormal spontaneous activity, and only a few motor unit potentials fired with each inspiration. These units were low amplitude, suggesting distal nerve block. (Calibration: 10 ms and 200 μV per division.) (With permission, Bolton.[11])

Needle EMG of the diaphragm reveals fibrillation potentials and positive sharp waves if there is axonal degeneration in the phrenic nerve. In relatively pure demyelination, no abnormal spontaneous activity will be found, simply a reduced number of motor unit potentials firing with each inspiration (Figure 8). One phrenic nerve may be more involved 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. These results may aid prognostication, patients with predominantly demyelination of phrenic nerves recovering earlier from ventilator dependence.[30]

Neuromuscular Transmission Disorders

Repetitive phrenic nerve conduction studies may be uncomfortable and difficult to interpret since the diaphragm compound action potential 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. However, repetitive stimulation of limb nerves will usually disclose a defect in neuromuscular transmission and provide indirect evidence that the respiratory neuromuscular system is involved.

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.[31]

Primary Myopathies

In primary myopathies, the latency of phrenic nerve conduction is normal but muscle compound action potential amplitudes from the diaphragm are 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 motor unit potentials is difficult because, normally, these potentials are unusually numerous and of relatively small amplitude and duration (Figure 4). Moreover, the patient is not easily able to control the activation of motor unit potentials with inspiration, which makes the detection of myopathic units, low amplitude, short duration or polyphasic motor unit potentials, particularly difficult. Computer analysis may help solve this problem.

Difficulty in Weaning from the Ventilator

This is often attributed to poorly-defined metabolic and nutritional disturbances[32] or the phenomenon of diaphragmatic fatigue.[13] However, by utilizing the above techniques, we[33] determined that 65% had critical illness polyneuropathy, the commonest cause, a lack of central drive, unilateral or bilateral damage to the phrenic nerves from surgical or other trauma, a neuromuscular transmission defect or a primary myopathy. Patients often had a combination of these disorders. Thus, 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 longterm prognosis and respiratory management.

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REFERENCES

1. Bolton CF. EMG in the critical care unit. *In*: Brown WF and Bolton CF, eds. Clinical Electromyography. Boston: Butterworth Publishers, 1993: 759-773.
2. Kelly BJ, Luce JM. The diagnosis and management of neuromuscular diseases causing respiratory failure. Chest 1991; 99: 1485-1494.
3. Plum F, Posner JB. The pathologic physiology of signs and symptoms of coma. *In*: The Diagnosis of Stupor and Coma, Contemporary Neurology Series. Third Editions, Philadelphia: F.A. Davis Co., 1980; 19: 34.
4. Munro I. Predictors of successful weaning in ventilated patients. The Lancet 1987; 1 (8544): 1245-1246.
5. Abd AG, Braun NMT, Baskin MI, O'Sullivan MM, Alkaitis DA. Diaphragmatic dysfunction after open heart surgery: treatment with a rocking bed. Ann Intern Med 1989; 111: 881-886.
6. Palmer JF, Holloway AM, Tanaka E. Detecting lower motor neuron dysfunction of the pharynx and larynx with electromyography. Arch Phys Med Rehabil 1991; 72: 214-218.
7. Gandevia SC, Rothwell JC. Activation of the human diaphragm from the motor cortex. J Physiol 1987; 384: 109-118.
8. Newsome-Davis J. Phrenic nerve conduction in man. J Neurol Neurosurg Psychiatry 1967; 30: 420-426.
9. Sarnoff SJ, Sarnoff LC, Whittenberger JL. Electrophrenic respiration. VII. The motor point of the phrenic nerve in relation to external stimulation. Surg Gynecol Obstet 1951; 93: 190-196.
10. Markand ON, Kincaid JC, Pourmand RA, et al. Electrophysiologic evaluation of diaphragm by transcutaneous phrenic nerve stimulation. Neurology 1984; 34: 604-614.
11. Bolton CF. Clinical neurophysiology of the respiratory system. AAEM Minimonograph #40. Muscle Nerve 1993; 16: 809-818.
12. Grand'Maison F, Bolton CF, Parkes A, Bernardi L. Phrenic nerve conduction. Can J Neurol Sci 1991; 18: 264.
13. Pradhan S, Taly A. Intercostal nerve conduction study in man. J Neurol Neurosurg Psychiatry 1989; 52: 763-766.
14. Bolton CF. Electrophysiological studies of critically ill patients. Muscle Nerve 1987; 10: 129-135.
15. Goodgold J. Anatomical Correlates of Clinical Electromyography (2nd ed). Baltimore: Williams & Wilkins, 1984: 41.
16. Saadeh PB, Sosner J, Wolf E. Needle EMG of the diaphragm: a new technique. Arch Phys Med Rehabil 1987; 68: 599.
17. Koepke GH. The electromyographic examination of the diaphragm. Bull Am Assoc Electromyogr Electrodiagnosis 1960; 7: 8.
18. Koepke GH, Smith EM, Murphy AJ, Dickinson DG. Sequence of action of the diaphragm and intercostal muscles during respiration: I. Inspiration. Arch Phys Med Rehabil 1958; 39: 426-430.
19. Bolton CF, Grand'Maison F, Parkes A, Shkrum M. Needle electromyography of the diaphragm. Muscle Nerve 1992; 15: 678-681.
20. Bolton CF, Parkes A, Grand'Maison F. Needle EMG of the diaphragm. Muscle Nerve 1993; 16: 324-325.
21. Dunne JW, Reutens DC, Newman M. Phrenic nerve injury in open heart surgery. Muscle Nerve 1991; 14: 883.
22. Lagueny A, Ellie E, Saintarailles J, et al. Unilateral diaphragmatic paralysis: an electrophysiological study. J Neurol Neurosurg Psychiatry 1992; 55: 316-318.
23. Cloward RB. Diaphragm paralysis from cervical disc lesions. Brit J Neurosurg 1988; 2: 395-400.
24. Carter GT, Kilmer DD, Bonekat HW, Liebermann JS, Fowler WM. Evaluation of phrenic nerve and pulmonary function in hereditary motor and sensory neuropathy, type I. Muscle Nerve 1992; 15: 459-462.
25. Bolton CF. Polyneuropathy as a cause of respiratory failure in critical illness. Intensive Crit Care Dig 1988; 7: 7-9.
26. Coronel B, Mercatello A, Couturier J-C, et al. Polyneuropathy: potential cause of difficult weaning. Crit Care Med 1990; 18: 486-489.
27. Zochodne DW, Bolton CF, Wells GA, et al. Critical illness polyneuropathy: a complication of sepsis and multiple organ failure. Brain 1987; 110: 819-841.
28. Witt NJ, Zochodne DW, Bolton CF, et al. Peripheral nerve function in sepsis and multiple organ failure. Chest 1991; 99: 176-184.
29. Ganapathy GR, Bolton CF, Parkes T, et al. Phrenic nerve conduction studies in respiratory insufficiency. Neurology 1991; 41 (Suppl. 1): 416.
30. Crisafulli CM, Saadeh PB, Wolf E. Phrenic nerve conduction studies and needle electromyography of the diaphragm in acute inflammatory demyelinating polyneuropathy. Muscle Nerve 1991; 14: 893.
31. Maher J, Bolton CF, Parkes T. Isolated respiratory muscle denervation in myasthenia gravis. Can J Neurol Sci 1992; 18: 282.
32. Editorial. Critical illness, infection, and the respiratory muscles. Am Rev Respir Dis 1988; 138: 258-259.
33. Maher J, Rutledge F, Remtulla, et al. Neurophysiological assessment of failure to wean from a ventilator. Can J Neurol Sci 1993; 20 (Suppl. 2): S28.