New techniques

Assessment of respiratory muscle function and strength

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Respiratory muscle weakness is present in many patients and may represent a significant problem for several reasons. It is a common feature of several neuromuscular diseases (box 1) and is associated with a higher morbidity and mortality. Recently, there has been increasing awareness that respiratory muscle weakness can be a compounding factor in many diseases, such as malnutrition, chronic obstructive pulmonary disease, congestive heart failure, and sepsis. Furthermore, respiratory muscle weakness is a complication of many metabolic diseases (box 2), endocrine disorders, and steroid therapy. Measurement of respiratory muscle strength is useful in order to detect respiratory muscle weakness and to quantify its severity, and to determine optimal long-term management. Respiratory muscle testing is useful in establishing the causes of unexplained dyspnoea, bulbar problems, and impaired cough, and is also important for the follow-up of patients with respiratory muscle weakness or those on respiratory muscle training programmes, and it is currently being used in ventilator weaning protocols. Indications for evaluation of respiratory muscle strength are shown in box 3.

History and physical examination

Mild weakness of respiratory muscles or hemidiaphragmatic paralysis may be asymptomatic, or may present with dyspnoea in the presence of coexisting respiratory disease. Isolated diaphragm weakness and even paralysis is without life-threatening sequelae, which underlines the fact that, for ventilatory failure to occur, load must exceed capacity. Global, severe respiratory muscle weakness causes dyspnoea, initially during exertion and eventually at rest. Severe isolated weakness of the diaphragm or bilateral diaphragm paralysis causes orthopnoea when the patient is in the supine position. Thus, patients with bilateral weakness of the diaphragm are unable to lie flat. Severe orthopnoea and obvious abdominal paradox occur only when the strength of the diaphragm is reduced to approximately 25% of normal. Severe respiratory muscle weakness may of itself cause ventilatory failure (eg, amyotrophic lateral sclerosis), but most commonly weakness is one of several factors contributing to ventilatory failure (eg, patients in an intensive care unit with weaning difficulties). Ventilatory failure due to respiratory muscle weakness usually develops gradually and hypventilation first develops during sleep. Such patients present with characteristic problems of disordered sleep, including daytime tiredness, somnolence, headache, and cognitive and intellectual impairment. Eventually ventilatory failure, pulmonary hypertension and cor pulmonale develop.

Some patients with neuromuscular diseases (box 1) have bulbar dysfunction which predisposes to aspiration. Abdominal muscles are also weak and this impairs cough and clearance of secretions. These factors predispose to recurrent respiratory infections. If neuromuscular disease spares the diaphragm, ventilatory failure is unlikely to occur. However, in generalised neuromuscular disorders it is unusual for the respiratory muscles to be spared. History and examination may confirm features of the many conditions, both neurogenic and myopathic, that can cause respiratory muscle weakness. Wasting and fasciculation of the limb and accessory muscles can be striking, such as, for example, in motor neurone disease. An important specific symptom of severe diaphragm weakness is dyspnoea when sitting or supine, or when standing in water. The characteristic finding of profound bilateral diaphragm weakness or paralysis is paradoxical movements of the rib cage and abdomen (abdominal paradox), and respiratory alternans, which are more obvious with the patient supine. There is active contraction of accessory muscles when ventilation is increased. In the upright position, the abdominal muscles may be visibly recruited during expiration, serving to elevate the diaphragm and allowing...
Assessment of respiratory muscle function

Neuromuscular diseases associated with respiratory muscles

**Neurogenic**
- tetraplegia and paraplegia (trauma)
- hemiplegia (stroke)
- motor neurone disease
- polynuropathy
- Guillain-Barré syndrome
- Werdnig-Hoffman syndrome
- amyotrophic lateral sclerosis
- poliomyelitis
- phrenic nerve paralysis (surgical trauma, tumour infiltration)
- ‘frostbitten phrenic’ (cardiac surgery, cardioplegia)
- transverse myelitis
- multiple sclerosis
- neuralgic amyotrophy
- Parkinson’s disease
- Charcot-Marie-Tooth disease

**Muscular**
- muscular atrophy
- muscular dystrophy (Duchenne)
- myotonic dystrophy (dystrophia myotonica)
- chronic obstructive pulmonary disease
- congestive heart failure
- inflammatory myopathies
- systemic lupus erythematosus
- rheumatoid arthritis
- dermatomyositis
- polymyositis
- kyphoscoliosis

**Neuromuscular function**
- myasthenia gravis
- Eaton-Lambert myasthenic syndrome
- cholinergic crisis
- botulism
- tetanus
- sepsis
- snake envenomation
- tick paralysis

**Drug-induced**
- aminoglycosides
- barbiturates
- anaesthetics
- chloroquine
- quinidine
- dilantin
- tricyclic antidepressants

**Poisoning**
- organophosphates
- lead
- arsenic

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subsequent gravitational-assisted descent during inspiration.\textsuperscript{27} There may be inward movements of the lower rib cage during inspiration (Hoover’s sign). These clinical features are seldom present until diaphragm strength is reduced to a quarter of normal, thus substantial diaphragm weakness can be overlooked on clinical examination.\textsuperscript{20}

**Radiology and imaging**

The radiological assessment of diaphragmatic function has traditionally relied upon plain radiography and erect fluoroscopy.\textsuperscript{28} In hemidiaphragm paralysis, an elevated hemidiaphragm may be visible on plain chest X-ray. Similarly, in bilateral diaphragm paralysis, chest X-ray may show elevation of both domes of the diaphragm. Unilateral or bilateral diaphragmatic paralysis may also be detected by radiological fluoroscopic screening, with the patient in a supine position and taking short sharp sniffs.\textsuperscript{1} Normally, the left diaphragm moves more than the right, with a cranio-caudal excursion of 3 and 6 cm, respectively. Paradoxical movement occurs when the pressure exerted by the abdomen exceeds that of the diaphragm so that movement is upward on inspiration or sniffing. This can occur in diaphragm paralysis due to injury or tumour involvement of the phrenic nerves.

Determining whether diaphragm elevation is due to paralysis or to an abdominal mass may be difficult. Ultrasound is definitive diagnostic investigation. Axial movements of the right hemidiaphragm during tidal breathing can also be recorded using real-time ultrasonography.\textsuperscript{26, 29} This investigation may be of clinical value in the assessment of diaphragm paralysis,\textsuperscript{29} diaphragm rupture,\textsuperscript{31} rib cage abnormalities, and abdominal defects in newborns.\textsuperscript{29}

**Volitional tests**

**PULMONARY FUNCTION TESTS**

Evaluation of pulmonary function would be incomplete without assessment of the respiratory muscles.\textsuperscript{26} Weakness of both inspiratory and expiratory muscles reduces vital capacity (VC). However, a fall in VC occurs only when weakness is moderate to severe, therefore, a normal VC excludes clinically significant respiratory muscle weakness.\textsuperscript{26} The accuracy of VC measurement is dependent on effort and co-operation by the patient. A major limitation of VC is that it is reduced by factors other than weakness, therefore, a low VC can be non-specific and non-diagnostic.\textsuperscript{27} Furthermore, VC is reduced by many pulmonary, cardiovascular and systemic diseases. Thus, it can be a rather insensitive, inaccurate and non-specific measurement of respiratory muscle weakness.\textsuperscript{1}

In patients with substantial diaphragm weakness, VC is decreased in the supine position; the fall in supine VC is more prominent in patients who have orthopnoea and abdominal paradox.\textsuperscript{21} In patients with bilateral diaphragm paralysis, the decline in VC is approximately 50%.\textsuperscript{19} In a clinical setting, VC is of great value in monitoring patients at risk of rapidly progressive muscle weakness or paralysis, for example, Guillain-Barré syndrome.\textsuperscript{32}

Other pulmonary function tests for abnormalities due to respiratory muscle weakness include: reduced total lung capacity and functional residual capacity, usually with a normal residual volume. The restrictive ventilatory defect caused by respiratory muscle weakness reduces overall gas exchange. The transfer factor for carbon monoxide (TLCO) is reduced but it is normal when adjusted for volume. This is important in distinguishing respiratory muscle weakness from lung parenchymal disorders, such as pulmonary fibrosis and pulmonary oedema. Pulmonary function test abnormalities in patients with severe respiratory muscle weakness are shown in box 4.

**ARTERIAL BLOOD GASES**

Isolated bilateral diaphragm paralysis rarely causes ventilatory failure.\textsuperscript{19} Low arterial partial pressure of oxygen (PaO\textsubscript{2}) and/or high arterial partial pressure of carbon dioxide (PaCO\textsubscript{2}) will not normally develop until respiratory muscle strength is reduced to less than 30% of normal.\textsuperscript{2} Initially, hypoxaemia and hypercapnia occur only during sleep, and sleep studies are therefore a part of assessment. From a clinical standpoint, it is important to note that the presence of hypercapnia implies severe weakness, and hypercapnia when awake is usually associated with a poor prognosis.

**POLYSOMNOGRAPHY**

Sleep studies including oximetry may be useful in detecting nocturnal desaturation due to respiratory muscle weakness. A normal nocturnal saturation obviously excludes severe respiratory muscle weakness or paralysis. Although nocturnal desaturation does not occur until global respiratory muscle strength is...
Metabolic and endocrine disorders which predispose to respiratory muscle weakness

Malnutrition
- beriberi
- alcoholic myopathy
- anorexia nervosa

Metabolic
- hypercapnia
- hypoxaemia
- chronic renal insufficiency
- hypocalcaemia
- hypokalaemia
- hypomagnesaemia
- hypophosphataemia
- periodic paralysis
- acute intermittent porphyria

Endocrine
- diabetes mellitus
- steroid myopathy
- hypothyroidism
- thyrotoxicosis
- hyperparathyroidism
- adrenal insufficiency
- acid maltase deficiency

Box 2

Indications for measurements of respiratory muscle strength

- investigation of patients with unexplained dyspnoea and cough
- evaluation of respiratory muscle strength in patients with neuromuscular diseases
- evaluation of respiratory muscle power in patients with cardiopulmonary diseases
- ventilator weaning protocols
- evaluation of therapeutic interventions (follow-up of patients with neuromuscular and cardiopulmonary diseases, patients on corticosteroid therapy, and patients on inspiratory muscle training)

Box 3

Reduced to below one third of normal in patients with neuromuscular disease alone, it may occur earlier if there is coexisting respiratory disease, such as chronic obstructive pulmonary disease and interstitial lung disease.

In some patients, severe isolated diaphragm paralysis is associated with nocturnal desaturation, particularly during rapid eye movement sleep, and the importance of this has yet to be elucidated.

Collapse of the upper airway during sleep may aggravate the situation and is relatively more common in patients with generalised neuromuscular disease.

LIMB MUSCLES

When assessing patients with respiratory muscle weakness it is useful to assess the strength of the peripheral skeletal muscles, since weakness of these muscles is frequently evident in neuromuscular and systemic diseases.

For example, the handgrip force is reduced in patients with neuromuscular disease, and in patients with cardiac cachexia due to congestive heart failure.

MOUTH PRESSURES

The most widely used test of global inspiratory and expiratory muscle strength are the static maximum pressures measured at the mouth (PImax and PEmax). These tests have the advantage that they are non-invasive and normal values have been established in both adults and children. A high PImax (80 cmH2O) or a PEmax (90 cmH2O) excludes clinically important inspiratory or expiratory weakness. PImax and PEmax are influenced by age, gender, posture, lung volume and the type of mouthpiece. They are preferably measured in the sitting position using a standard flanged mouth piece.

Conventionally, PImax is measured from residual volume, and PEmax from total lung capacity, with a noseclip. The highest recorded pressure maintained for 1 s represents PImax or PEmax. Portable mouth pressure metres allow immediate measurement of PImax and PEmax at the bedside or in the clinic. PImax can be easily measured during routine follow-up of ambulatory patients with chronic obstructive pulmonary disease. It is also useful in assessing respiratory muscle strength in patients with respiratory failure who cannot undergo an invasive assessment of inspiratory muscle due to respiratory distress.

There are a few drawbacks to measurements of PImax and PEmax. One problem with static pressures is that they are volitional, thus, for the result to be a true measure of strength, the subject must make maximal contraction. Furthermore, there is wide inter-subject variability in PImax and PEmax in normal individuals, and significant variation in reported normal values for PImax. This may be due to subject motivation, number of pre-measurement runs (learning effect), and peri-oral leaks, especially at higher pressures and in older subjects. The PImax manoeuvre is both demanding and unpleasant. Low PImax may also be due to lack of motivation or co-ordination.

Clinically, PImax is the most widely applied technique in assessment of inspiratory muscle strength. PEmax is the only test generally available for measurement of expiratory muscle power.

SNIFF NASAL PRESSURE

There is a close correlation between nasal, nasopharyngeal, mouth, and oesophageal pressure during a sniff in normal subjects, and in patients without severe nasal disease. A useful alternative method for measuring inspiratory muscle strength consists of asking the subject to perform short sniffs of maximal intensity. The sniff manoeuvre is natural and probably easier to perform than the PImax manoeuvre for most subjects.

Thus, inspiratory muscle strength is often better reflected by maximal sniff pressure (SNIP) than by the PImax manoeuvre. SNIP is measured through a plug occluding one nostril during sniffs performed through the other nostril. Nasal leaks are eliminated by using waxed ear plugs hand-fastened around the tip of a polyethylene catheter. SNIP is measured from functional residual capacity rather than from residual volume, because inspiratory muscle strength is overestimated at levels below functional residual capacity due to the elastic recoil pressure of the thorax.

In clinical practice, SNIP is easier to measure than sniff nasopharyngeal pressure (sniff Pn). SNIP can be predicted from age by a first degree equation for both sexes. Normal values of SNIP have been established in one large study of 160 subjects (men > 70 cmH2O, women > 60 cmH2O).

Nonetheless, the sniff manoeuvre is volition-dependent, and submaximal efforts are most likely to be recorded in patients who are ill or breathless. Other limitations of SNIP include anatomical abnormalities such as nasal polyps, nasal septal defects, and septal deviation, that may impair pressure transmission from the rhinopharynx. Nasal mucosal congestion in patients with rhinitis predisposes to the same kind of limitation.

In the described conditions, there is underestimation...
of sniff oesophageal pressure by SNIP. Furthermore, SNIP measurements are unreliable in the presence of airway obstruction or pulmonary fibrosis that prevent accurate transmission of pleural pressure to the upper airways.46 In patients with chronic obstructive pulmonary disease the sniff inspiratory pressure underestimates inspiratory muscle strength,47 because the sniff is a short manoeuvre.48 However, SNIP is very useful in the evaluation and follow-up of patients with neuromuscular or skeletal disorders.

Maximal SNIP is similar in the sitting and supine position and is significantly higher than PImax in healthy subjects50 for several reasons:

- the sniff measurement is easier and less unpleasant than the PImax manoeuvre and may thereby allow maximal muscular recruitment to be achieved more often51
- dynamic changes in human diaphragm length during maximal inspiratory effort against occlusion using sequential radiography, indicate that this manoeuvre does not represent an isometric contraction
- the level of recruitment of inspiratory muscle group is different during sniff and during PImax manoeuvre.

SNIP and PImax are not interchangeable but rather compliment one another in the assessment of inspiratory muscle strength. In patients suspected of respiratory muscle weakness, a SNIP may be required to confirm respiratory muscle weakness if PImax is low in order to rule out lack of motivation or co-ordination during the PImax manoeuvre. The development of the SNIP test represents a further step towards accurate, easy, and noninvasive assessment of inspiratory muscle strength.28

**OESOPHAGEAL, GASTRIC, AND TRANSDIAPHRAGMATIC PRESSURE**

In some patients, simple noninvasive tests will fail to confirm or refute the possibility of respiratory muscle weakness. Similarly, for patients with known weakness it is important to make a precise estimation of the weakness in order to make decisions about management. Placement of oesophageal and gastric balloons using topical anaesthesia allow measurement of oesophageal, gastric, and transdiaphragmatic pressure, and also dynamic compliance. Oesophageal and gastric pressure are measured by two balloons, one placed in the mid-oesophagus (Poes) and the other in the stomach (Pga) during a PImax manoeuvre. The balloons should be positioned and tested in the standard manner.49 Oesophageal pressure reflects pleural pressure, and Pga reflects abdominal pressure. Transdiaphragmatic pressure (Pdi) is the pressure difference between Pga and Poes. Pdi reflects the tension developed by the diaphragm, and is viewed as a reflection of diaphragmatic inspiratory action. These measurements depend on lung volumes and should be standardised for the lung volumes at which they are measured.

The measurement of Pdi using two balloon catheters is not an easy or short procedure, and in some patients it may not be well-tolerated. Although the two-balloon technique is an invasive technique, PImax measured during voluntary manoeuvres is not a reliable index of diaphragm function. Furthermore, PImax does not permit discrimination between various inspiratory muscle groups. Therefore, measurement of Pdi may be required to assess diaphragmatic function specifically.

**SNIFF OESOPHAGEAL AND TRANSDIAPHRAGMATIC PRESSURES**

In the search for a reliable and practicable test of inspiratory muscle strength, considerable interest has recently focused on the sniff manoeuvre.42 50 51 Inspiratory muscle strength is often better reflected by oesophageal pressure during a maximal sniff (sniff Poes).42 Similar to SNIP, sniff Poes is performed from functional residual capacity without a noseclip. When thus performed the volume increase is about 500 ml,42 and diaphragm contraction is therefore relatively isometric. The normal mean sniff Poes is 93 ± 20 cmH₂O, range 74–135 cmH₂O. The mean value for the ratio SNIP/sniff Poes is 0.91 (0.82–0.99).44 In normal subjects sniff Poes and sniff Pdi have a narrower normal range and a better between-day coefficient variation than the PImax manoeuvre.44 Occasionally, there is overestimation of sniff Poes because of agonist activation of the orofacial muscles in some normal subjects.53

Sniff Poes also provides a reliable estimation of inspiratory muscle strength in patients with neuromuscular and skeletal disorders.54 However, in patients with altered lung mechanics, sniff Poes is usually lower than SNIP. Recently, Uldry and colleagues55 have shown that the mean SNIP/sniff Poes ratio in patients with chronic obstructive pulmonary disease is 0.80, compared with 0.90 in normal subjects.46 SNIP underestimates inspiratory muscle strength due to impaired transmission of pleural pressure in the presence of airway obstruction.46 54 This difference could be expected in view of the short and dynamic character of the
sniff.\textsuperscript{34} Other limitations of sniff Poes and sniff Pdi measurements include anatomical nasal abnormalities, eg, nasal polyps, septal deviation, septal defects, and nasal mucosal congestion due to rhinitis, which may impair pressure transmission from the rhinopharynx.\textsuperscript{41}

The validity of sniff testing also depends on the subject making a maximal effort. It is increasingly recognised that submaximal activation is common during volitional muscle testing, particularly in patients who are ill or breathless.\textsuperscript{46} Nevertheless, in clinical practice, sniff Poes and sniff Pdi are the most accurate and reproducible volitional tests currently available to assess global inspiratory, and diaphragmatic strength.\textsuperscript{25}

Non-volitional tests

PHRENIC NERVE ELECTRICAL STIMULATION

The twitch occlusion technique was endorsed by the 1989 National Heart, Lung and Blood Institute workshop on respiratory muscle fatigue as a potentially major diagnostic tool.\textsuperscript{77} Currently, twitch oesophageal pressure (TwPoes) has been used to assess respiratory muscle function in normal subjects,\textsuperscript{31} and in patients with respiratory muscle weakness.\textsuperscript{3, 15, 55} Bilateral electrical phrenic nerve stimulation (EPS) in the neck and the measurement of twitch Pdi (TwPdi) has the advantage of eliminating variation due to patient motivation. It also allows measurement of electromyographic signals and nerve conduction (normal (SD) range 7.7 (0.8) ms).\textsuperscript{59, 60} In addition, hemidiaphragm function may be successfully assessed by unilateral electrical stimulation of the phrenic nerves.\textsuperscript{18} Twitch potentiation is a potential problem during assessment of diaphragm strength, irrespective of the method of stimulation.\textsuperscript{61, 62} After a maximal voluntary contraction of the diaphragm, the TwPdi at functional residual capacity can increase by 70%.\textsuperscript{61} To minimise potentiation, subjects should rest, breathing quietly for 10–20 minutes before testing.\textsuperscript{63} The normal range for TwPdi is 8.8–33.0 cmH\(_2\)O. This broad range makes it diagnostically useful only when there is severe weakness or when the result is equivocally normal.\textsuperscript{58}

A major drawback to EPS is that it is not an easy technique to perform, and the operator must be highly trained for the results to be relied upon.\textsuperscript{58} Current intensities required to achieve supramaximal activation can exceed 4–50 mA, and can be perceived as too painful by some subjects. Patients may find it difficult to relax during EPS, thereby causing an increase in twitch tension — the phenomenon of twitch potentiation.\textsuperscript{61, 62} Furthermore, EPS is technically difficult in obese patients and Cushingoid patients with “buffalo-hump”.\textsuperscript{63} Indeed the phrenic nerve may be impossible to locate. EPS is potentially dangerous in infants and small children,\textsuperscript{44} and should be avoided in patients with epileptic seizures. Despite some promising findings, this method has yet to gain widespread clinical acceptance because of technical concerns relating to patient discomfort, stimulation of the muscle groups, and concerns about the variability of phrenic nerve stimulation.

EPS Twitch mouth pressure

It is also possible to measure twitch Pmo (TwPmo) during EPS.\textsuperscript{56} TwPmo correlates well with TwPoes, at relaxed functional residual capacity and relaxed volumes above functional residual capacity in normal subjects. However, in patients with chronic obstructive airways disease, pressure equilibration between alveoli and mouth is incomplete due to the airways acting as a resistance-capacitance filter between the two. Because the airway time is long and diaphragmatic contraction time short, TwPmo appears damped and time-lagged with respect to TwPoes.\textsuperscript{3}

MAGNETIC PHRENIC NERVE STIMULATION

By discharging a magnetic coil, it is possible to create a pulsed magnetic field that causes current to flow in nervous tissue, which in turn causes muscle to contract.\textsuperscript{65} Cervical magnetic stimulation (CMS) is performed using a magnetic stimulator (Magstim 200, The Magstim Company Limited, Whitland, Carmarthenshire, Wales). Supramaximal bilateral phrenic nerve stimulation may be achieved using a 9–10 cm circular coil placed over the cervical phrenic nerve roots at the spinous processes of the 5th to 7th cervical vertebrae.\textsuperscript{66, 67} CMS should be performed at functional residual capacity, after airway occlusion, with the diaphragm relaxed. Mean (SD) TwPdi for normal subjects is 31 (6) cmH\(_2\)O.\textsuperscript{66} The magnetically elicited TwPdi is slightly greater than the electrical, the difference being due to a larger TwPoes.\textsuperscript{67, 68} Magnetic stimulation has a higher and better defined lower limit of normal than EPS, and a better correlation with sniff Pdi, which increases its diagnostic sensitivity in patients with moderate diaphragm weakness,\textsuperscript{69} and allows sequential studies in patients.\textsuperscript{70}
During CMS, great care should be taken to locate the optimal position for the magnetic coil on the neck, and the neck should be flexed to obtain maximal contact. Failure to achieve this may result in significantly submaximal stimulation. This can occur if the coil cannot be placed close enough to the phrenic nerves, eg, in patients with ankylosing spondylitis, systemic lupus erythematosus, or Cushing syndrome with "buffalo-hump". Stimulation of the phrenic nerves anteriorly with a figure of eight coil allows easy and accurate quantification of diaphragm function in supine subjects, for example, patients in the intensive care unit, or undergoing coronary artery surgery.

CMS is easier to perform than EPS, is painless and perhaps "closer to life," because it stabilises the rib cage. It is possible to achieve stimulation without the problem of twitch potentiation. Reproducibility is slightly better than for EPS.

In clinical practice, the TwPdi invoked by this method is a valid measure of diaphragm strength that is acceptable to patients. The major advantage of magnetic stimulation is that the test is independent of patient aptitude and motivation. Currently, CMS is beginning to be used in a clinical setting, including evaluating difficult weaning from mechanical ventilation after cardiac surgery. It is likely to be of value in investigation of low frequency fatigue in clinical studies. Because patients find it so acceptable, it may be a useful technique for evaluating strategies aimed at altering respiratory muscle strength, ie, inspiratory muscle training.

CMS Twitch mouth pressure
Measuring pressure at the mouth during magnetic stimulation of the phrenic nerves can be used to assess diaphragm contractility. In normal subjects, mean TwPmo is 13.7 cmH₂O (range 11.3–16.1) and TwPdi (range 10.4–15.9). In normal subjects, the relationship between TwPmo and TwPdi is close to unity with a mean (SD) difference of 0.1 (1.13) cmH₂O. Preliminary studies have shown that TwPmo measured by CMS reflects TwPoes in some patients with lung disease. An advantage of this method is that it avoids the need for oesophageal and gastric balloons, which are usually considered unpleasant by stable patients.

ELECTRICAL VS MAGNETIC STIMULATION
Electrical stimulation is often uncomfortable, particularly when attempts are made to achieve bilateral supramaximal stimulation, while localising the phrenic nerves can be difficult in some subjects with short necks, 'buffalo-hump', and kyphoscoliosis. Because maintaining a constant symmetrical maximal stimulus can be difficult, repeated twitch stimulations are often performed, which in itself can increase twitch pressure, the so-called staircase phenomenon. It has been shown that maximal voluntary contraction can increase TwPdi by up to 70%. With CMS, locating the phrenic nerve roots is not a much of a technical problem, and intact nerves are stimulated invariably. This technique is considerably less painful than EPS, and the risk of the staircase phenomenon is low because locating the area of maximal stimulation usually requires only a few stimulations. In normal subjects at relaxed functional residual capacity, TwPdi elicited by CMS is greater than that elicited by EPS. The reason for the discrepancy in TwPdi values with the two techniques is not completely understood. Magnetic stimulation is considered to act on both the cervical roots and phrenic nerve trunks, and thus should not be affected by the presence of ectopic branches of the phrenic nerve. In addition, contractions of the sternocleidomastoid, trapezius, parasternal and pectoralis muscles are known to decrease rib cage compliance, which should result in a larger TwPoes for a given diaphragmatic contraction. Recruitment of the extradiaphragmatic musculature by magnetic stimulation is possibly responsible for the difference in regional motion of the rib cage between the two stimulation techniques.

In EPS the stimulation is relatively confined to the diaphragm, while CMS stimulates the diaphragm and muscles of the upper rib cage, which may cause a somewhat higher magnetic than electrical TwPdi. However, the relative non-specificity of cervical magnetic stimulation does not undermine its value in the assessment of diaphragmatic function. CMS is as effective as EPS in the detection of diaphragm weakness or fatigue.

MAGNETIC STIMULATION OF THE CORTEX
Recently, it has been shown that respiratory muscle weakness can be assessed using cortical stimulation. Magnetic stimulation of the cortex (CxMS) is performed with a Magstim 200. The coil is positioned over the vertex, 1 to 3 cm behind the midauricular plane, at maximal stimulation intensity. In order to minimise twitch potentiation, the subject should breathe quietly and remain relaxed for 20 min. CMS and CxMS are performed at functional residual
**Step-wise assessment of patients with respiratory muscle weakness**

- history and clinical examination
- chest imaging
- pulmonary function tests: sitting and supine vital capacity, transfer factor, arterial blood gases
- maximal inspiratory and expiratory pressures (PImax and PEmax)
- measurement of Poes, Pga, and Pdi during PImax manoeuvre
- sniff inspiratory pressures (sniff Pmo, sniff Ppas, sniff Poes, sniff Pdi)
- cervical magnetic stimulation of phrenic nerves (TwPmo, TwPoes, TwPdi)
- electrical stimulation of the phrenic nerves (TwPmo, TwPoes, TwPdi)
- cervical magnetic stimulation of phrenic nerves (phonomyography)
- cortical magnetic stimulation (TwPoes, TwPdi)

**Abbreviations**

- CMS and EPS: electromyography
- PImax: maximal inspiratory pressure
- PEmax: maximal expiratory pressure
- Pgas: gastric pressure
- Poes: oesophageal pressure
- Ppas: airway pressure
- TwPmo: twitch phrenic nerve action potential

**Box 5**

**Conclusions**

A scheme for the step-wise assessment of respiratory muscles weakness is shown in box 5. However, the most attractive and innovative assessment of diaphragm function include recording the phonomyogram during magnetic stimulation. The combination of an easy and painless magnetic stimulation and phonomyography may be the standard method for assessment of respiratory muscle function in the next millennium.
Assessment of respiratory muscle function


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