Assessment of respiratory muscle strength
in the Intensive Care Unit

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ABSTRACT: The measurement of respiratory muscle strength in the intensive care unit (ICU) is potentially useful for the prediction of weaning outcome. An easy and accurate measure would also allow investigation of respiratory muscle weakness in critically ill patients.

At present, there is no satisfactory method of strength measurement in the intensive care unit. Vital capacity is a nonspecific, volitional and relatively insensitive measure of strength. True maximum respiratory pressures are difficult to achieve and, in stable patients, results vary greatly both over time and between observers. For these reasons, there are few good data of respiratory muscle strength in the intensive care unit.

Of the new techniques being developed, magnetic stimulation of the phrenic nerves, combined with the measurement of transdiaphragmatic, oesophageal, or endotracheal tube pressure, offers the greatest promise.

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Respiratory muscle weakness is likely to be a relatively common problem in intensive care unit (ICU) patients, particularly in those requiring ventilatory support for prolonged periods [1]. Some patients have neuromuscular disorders prior to ICU admission, but in most of them weakness is a consequence of the illness requiring ventilation and of the time spent in the ICU. Muscle wasting, metabolic disorders, drug therapy and neuromyopathic changes can all cause weakness. Most patients who have sepsis and organ failure develop neuromuscular abnormalities [2], and acidosis may be a potent cause of weakness [3].

If patients develop respiratory muscle weakness, weaning is likely to be difficult and prolonged. In the ICU, it is frequently clinically relevant to ask the questions: are the respiratory muscles weak; if so, how weak; and, is weakness a significant clinical problem? To answer these important questions, we require tests of respiratory muscle strength that are both sensitive and practical in the complex ICU environment.

Conventionally, the two most frequently used measurements of respiratory muscle strength in the ICU are vital capacity and maximum respiratory pressures.

Vital capacity

Weakness of the inspiratory muscles reduces inspiratory capacity; weakness of expiratory muscles reduces expiratory capacity; and generalized respiratory muscle weakness, therefore, reduces vital capacity (VC). In the pulmonary function laboratory, VC can be a useful and simple measure of weakness [4]. The pressure-volume relationship for the normal lung is such that only modest pressures are required to fully inflate the lung, and strength must be substantially reduced before VC falls. Vital capacity is, therefore, not a very sensitive test for the detection of acute mild to moderate weakness. For patients with chronic respiratory muscle weakness, for example those with muscular dystrophy or neurological disorders, secondary changes in the mechanical properties of the lung and chest wall result in a more linear relationship between VC and respiratory muscle strength. In general, a reduction in VC to less than 30% of predicted normal is usually necessary before patients develop a raised arterial carbon dioxide tension (PaCO₂) [5]. In patients with substantial diaphragmatic weakness, VC is further reduced in the supine posture [6], clearly relevant in the ICU. The data obtained from patients with diaphragmatic weakness studied in the pulmonary function laboratory indicate that a fall in supine VC is not usually substantial until diaphragmatic strength is reduced to one third of normal.

The great virtue of VC as an index of respiratory muscle strength is that it is easy to perform, but its disadvantage is that it is nonspecific. Vital capacity is reduced by both obstructive and restrictive pulmonary disease, as well as by extrapulmonary factors, such as obesity or ascites. Thus, although a normal supine VC excludes moderate to severe respiratory muscle weakness, a reduced VC can be due to weakness, or respiratory disease, or both. Since so many patients in the ICU have pulmonary
disease, such as chronic obstructive pulmonary disease (COPD), pneumonia or adult respiratory distress syndrome (ARDS), the nonspecificity of VC is an important limitation. The vital capacity manoeuvre requires effort and co-operation from the patient, and in the ICU it is frequently difficult to be certain whether or not patients have made truly maximal inspiratory and expiratory efforts. Again, whilst a high VC is reassuring, a reduced VC is too often difficult to interpret, and commonly most difficult in those patients for whom accurate information would be particularly useful. Clearly, VC measurements are difficult in confused patients.

The components of the vital capacity manoeuvre can be measured separately in poorly co-operative patients by their breathing through a one-way valve, arranged such that inspiratory (or expiratory) reserve volume is cumulatively estimated until no further breath can be taken [7]. When compared with standard techniques, this method shows good agreement and can be applied to intubated patients.

Thus, vital capacity is most useful in the assessment of weakness in patients who are alert and co-operative and without co-existing pulmonary disease, as for example in the Guillain-Barré syndrome [8]. CHEVROLET and DELÉAMONT [8] studied 10 patients with Guillain-Barré syndrome, half of whom developed ventilatory failure requiring mechanical ventilation. The patients required intubation and ventilation when the VC fell to approximately 1 l (15 ml·kg⁻¹ body weight) (fig. 1). As the patients regained strength, VC rose, and successful weaning became possible when VC once again exceeded 15 ml·kg⁻¹ (fig. 2).

Maximum respiratory pressures

In the pulmonary function laboratory, strength of the respiratory muscles is most commonly assessed by documenting their capacity to generate pressure. Maximum mouth pressures are easily measured [4], and to document the strength of the inspiratory muscles the patient simply makes a maximum inspiratory effort against a closed airway, from either residual volume or functional residual capacity. Normal values for maximum inspiratory and expiratory mouth pressures have been widely reported [4]. In the ICU, measurements are much more difficult. When the intubated patient makes inspiratory efforts, it is a relatively simple matter to briefly occlude the airway and record the pressure within the endotracheal tube with an appropriate pressure gauge or transducer. Patients are encouraged to make maximal efforts and, in an attempt to achieve reproducible values, repeated measurements are made. Maximum inspiratory pressure (MIP) measurements are routinely undertaken in many ICUs and are often used to judge when weaning is appropriate. Conventional ICU wisdom considers that when MIP is greater than 30 cmH₂O, patients should wean successfully; when MIP is less than 20 cmH₂O, weaning will fail; and when MIP is 20–30 cmH₂O, the
outcome is uncertain. However, MIP results are of less value in predicting weaning outcome than conventional dogma suggests. Many studies have investigated MIP and weaning [9]; in some, weaning outcome was related to MIP, but in others it was not.

What is striking about these data is that in some studies weaning was achieved with low MIP values and in others weaning failed despite much higher MIP results. Several factors could explain this apparent discrepancy, including variability of ventilatory load, but a key factor is likely to be the variability of MIP measurements, because of the great difficulty in performing this test in such a way that it truly reflects inspiratory muscle strength. Indeed, many ambulant, relatively well, and highly motivated patients attending the pulmonary function laboratory, nevertheless, have difficulty with the maximum inspiratory mouth pressure manoeuvre. The normal laboratory ranges for mouth pressure are wide; for males, maximum inspiratory mouth pressure can be any value between 50 and 200 cmH₂O.

The measurement of MIP in the ICU poses additional problems that undermine its reliability. Frequently, ICU patients find it very difficult to make maximum voluntary efforts. Even for patients who are alert and cooperative, the MIP manoeuvre is unpleasant, and many are inhibited from making truly maximal contractions. Much is also likely to depend on the skill, attitude, encouragement and technique of the physician, nurse or physiotherapist making the measurements. Conventionally, MIP in the ICU is measured by briefly occluding the airway at end-expiratory volume. To try to improve the quality of MIP measurements, Marin et al. [10] modified this technique by attaching a one-way valve to the endotracheal tube, which allowed expiration but obstructed inspiration. With this technique, over a period of 20–30 s they noted that repeated MIPs progressively increased in amplitude. The MIP values were higher than for the occluded airway technique. These investigators also found that the one-way valve technique could be used in patients whose conscious level did not allow them to make voluntary efforts to command.

Comparison of the one-way valve technique and the more conventional occlusion approach, by Kamarek et al. [9], in 50 patients about to wean from mechanical ventilation showed a mean MIP of 30.8 cmH₂O when the airway was occluded at end-expiration and 39.8 cmH₂O when a one-way valve was attached to the airway. Despite modification of the MIP technique, it remains doubtful whether it reliably provides useful information about inspiratory muscle weakness. When MIP is high, important respiratory muscle weakness is excluded and this information is useful, but, in the majority of patients, the MIP is low, certainly when compared to maximum inspiratory mouth pressure results for normal subjects.

In practice, most MIP values range from 20–60 cmH₂O, but do such values really indicate weakness?MULTZ et al. [11] investigated the reliability and reproducibility of MIP measurements in the ICU in 14 stable patients, using the one-way valve technique of Marin et al. [10]. Five experienced investigators made repeated measurements over seven days. Different investigators obtained different MIP values on the same patients studied on the same day, the best MIP value for a particular patient varying by 40% between investigators. Because the "true" MIP must be equal to or greater than the largest MIP value obtained, they concluded that maximum inspiratory pressure is commonly underestimated by the technique (fig. 3). The difficult question remains of what a low MIP really means.

In our experience, the best results for MIP are obtained with co-operative, well-motivated patients. Ventilatory drive should be high, the patients should not be hypocapnic, and we try to allow patients to breathe spontaneously for several minutes before attempting the MIP manoeuvre. Great care is taken to explain to the patients what is required of them, in particular they are reassured that they can make repeated efforts, but they will not be exhausted. We attach a positive end-expiratory pressure (PEEP) valve to the endotracheal tube to act as a one-way valve. Pressure is measured from within the endotracheal tube using an air-filled arterial pressure catheter and a conventional vascular pressure transducer. The baseline of the pressure transducer is offset, so that it can indicate negative pressures, by applying a standing positive pressure to one side of the diaphragm. Pressures are displayed on the bedhead monitor. The patient is vigorously encouraged to make repeated maximum inspiratory sniff-like gasps.

Using presently available techniques, MIP measurements in the ICU are of limited value. It is likely that much of the literature that reports clinical studies based on MIP measurements is undermined by the unreliability of the measurement. Improvements in the methods available to measure inspiratory muscle strength are urgently needed, either by refinement of the MIP technique, or, more likely, by adopting different strategies.

**Phrenic nerve stimulation**

Phrenic nerve stimulation [4] has the great advantage of being a nonvolitional test of diaphragm contractility.
In the laboratory, the nerves are usually stimulated using percutaneous electrodes applied at the posterior border of sternomastoid at the level of the cricoid cartilage. Unilateral electrical stimulation produces a transdiaphragmatic pressure (Pdi) twitch response of approximately 10–15 cmH₂O, bilateral stimulation 25–40 cmH₂O. By recording the surface diaphragm electromyographic (EMG) activity it is easy to measure the phrenic nerve conduction time, normally less than 9.5 ms.

Although electrical phrenic nerve stimulation is often satisfactory in normal subjects, it is frequently less successful in patients referred to the laboratory for testing. Some patients find supramaximal stimulation uncomfortable and, in practice, it can be difficult to achieve reproducible maximal responses. Nerve stimulation is critically dependent on the precise positioning of the electrodes. Any small movement results in suboptimal activation. In the ICU the technical problems of phrenic nerve stimulation are greatly increased. Alert patients frequently find the technique painful and neck cannulae or trauma and swelling often make stimulation difficult or impossible.

The recent introduction of magnetic phrenic nerve stimulation is an important advance [12], and patients find this technique more acceptable than electrical stimulation [13]. For patients with substantial soft tissue thickness over the back of the neck, magnetic stimulation may not elicit a maximal response, and in the ICU it may also be difficult to position some patients in the best posture for stimulation. The development of small coils, that allow unilateral local phrenic nerve stimulation, is likely to be useful.

The assessment of diaphragmatic strength by recording Pdi requires oesophageal and gastric balloons. However, it is feasible to record pressure at the mouth during phrenic stimulation and obtain a useful index of diaphragmatic contractility [14]. Similarly, it is possible to measure endotracheal tube pressure in patients in the ICU during phrenic stimulation. This technique has yet to be formally evaluated, but in the future may become an important noninvasive method for assessing diaphragmatic strength in the ICU.

### The load: capacity ratio

In the ICU, assessment of the clinical importance of reduced respiratory muscle capacity must take into account the ventilatory load. Both sides of the load:capacity equation are equally important.

Laboratory studies demonstrate that respiratory muscle fatigue develops when the load is excessive relative to capacity, when the mean inspiratory pressure during each breath (Pm) becomes a high proportion of maximum inspiratory pressure (PImax), when Pm/PImax is greater than 0.15–0.20, fatigue occurs.

Studies in the ICU show that long-term ventilator-dependent patients fail a weaning trial when the oesophageal pressure required to achieve adequate ventilation is a large fraction of maximum inspiratory pressure [15] (fig. 4). In the ICU, an assessment of ventilatory load,
for example by documenting dynamic compliance, provides crucial information when interpreting the significance of respiratory muscle strength measurements.

**Respiratory muscle fatigue in the ICU**

There are few data evaluating respiratory muscle fatigue in the ICU. Early studies demonstrated that during weaning failure the diaphragmatic EMG signal altered, the EMG "high/low" ratio fell, thereby indicating (in EMG terms) diaphragmatic fatigue [16]. More recently, the maximum relaxation rate (MRR) of the inspiratory muscles has been documented in ICU patients, and weaning failure shown to be associated with slowing of MRR; whereas, no slowing occurred in patients that were successfully weaned [15]. These data strongly suggest that in acute weaning failure a "fatiguing process" is initiated in the excessively loaded inspiratory muscles (fig. 5). It is likely that refinement of the magnetic phrenic nerve stimulation technique will allow further and more comprehensive studies of diaphragm fatigue in the ICU.

**Conclusions**

The measurement of respiratory muscle strength in the ICU is difficult and inaccurate. It is likely that voluntary manoeuvres usually underestimate strength, and this unreliability undermines much of the available data in the literature. Phrenic nerve stimulation has the advantage of being a nonvolitional test, but further advances are required to make the technique generally applicable in the ICU. The importance of weakness in the ICU depends on the ventilatory load imposed on the respiratory muscle pump; the load:capacity ratio is the crucial issue. It is likely that in some clinical situations, for example weaning failure, the excessive load on the respiratory muscles, in relation to their capacity, initiates a fatiguing process.

**References**