Review Article

The Assessment of Maximal Respiratory Mouth Pressures In Adults

John A Evans MSc and William A Whitelaw MD PhD

Maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) are simple, convenient, and noninvasive indices of respiratory muscle strength at the mouth, but standards are not clearly established. We review recent literature, update the 2002 American Thoracic Society/European Respiratory Society statement, and propose as the best choice using a flanged mouthpiece for reference values and lower limit of normal (LLN) values as a function of age for adults age up to about 70 years. Because male pressures are higher than female and MEP exceeds MIP, we present 4 linear regression reference equations as a function of age for adults age up to approximately 70 years: Male MIP $= 120 - (0.41 \times$ age), and male MIP LLN $= 62 - (0.15 \times$ age). Male MEP $= 174 - (0.83 \times$ age), and male MEP LLN $= 117 - (0.83 \times$ age). Female MIP $= 108 - (0.61 \times$ age), and female MIP LLN $= 62 - (0.50 \times$ age). Female MEP $= 131 - (0.86 \times$ age), and female MEP LLN $= 95 - (0.57 \times$ age). (Pressure in cm H_2O and age in years.) We discuss normal values in older subjects, estimation of LLN values, and the relationship between vital capacity and respiratory muscle strength, and offer a guide to interpretation of maximal pressure measurements. The approach should allow direct implementation of MIP and MEP in a pulmonary function laboratory. *Key words: maximal inspiratory pressure, MIP, maximal expiratory pressure, MEP, muscle strength, respiratory pressures, mouth pressures, normal values, prediction equations, reference equations, mouthpiece.* [Respir Care 2009; 54(10):1348–1359. © 2009 Daedalus Enterprises]

At the time of this study, John A Evans MSc was affiliated with the Pulmonary Laboratory, Foothills Medical Centre, Calgary Health Region, Calgary, Alberta, Canada. William A Whitelaw PhD MD FRCP is affiliated with the Division of Respiratory Medicine, Department of Medicine, University of Calgary, Calgary, Alberta, Canada.

Correspondence: E-mail: evansja@shaw.ca.

Introduction

This paper is intended to supplement guidelines for use of maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) given in the American Thoracic Society/European Respiratory Society (ATS/ERS) statement[1] on respiratory muscle testing of 2002, in order to aid their implementation in a pulmonary function laboratory.

Results of some of the basic tests used to assess pulmonary function depend not only on the lungs themselves but also on the respiratory muscles. Total lung capacity (TLC), the volume reached at the end of a maximal inspiration, is usually determined by lungs that cannot be expanded further, even by very large negative pressures, but if inspiratory muscles are weak, their maximum effort may not be enough to fully expand the lungs. Similarly, if expiratory muscles are weak, they may not be able to compress the lungs to the normal residual volume. A low vital capacity (VC) or TLC can thus be a sign of either "restrictive" lung disease or weakness of inspiratory muscles, while a high residual volume or small expiratory reserve volume can be a sign of either gas trapping from airways obstruction or weakness of expiratory muscles. To decide between muscle weakness and lung disease requires tests of respiratory muscle strength that are independent of the condition of the lung. Maximal inspiratory pressure (MIP) and maximal expiratory mouth pressure (MEP) are simple tests in which patients generate as much inspiratory or expiratory pressure as possible against a blocked mouthpiece. Because lung volume cannot change significantly during measurement, results are to a large extent independent of the properties of the lungs. They are general tests of neuromuscular function of the combined diaphragm, abdominal, intercostal, and accessory muscles.

The simple apparatus needed consists of a well fitting mouthpiece connected to a small chamber, to which a pressure gauge is connected (Fig. 1). A small leak in the chamber prevents the patient from using buccal muscles to generate the pressure. For MIP the patient stops breathing at a set volume, usually residual volume, and tries to sustain a maximal negative pressure, usually for one second or more. For MEP the volume is usually TLC, and positive pressure is generated. Details of how the test is performed, from choice of mouthpiece to instructions and motivation of the subjects to analysis of the data, make important differences to the result. Standardization of these is very important.

MIP and MEP are not usually done on all patients referred for pulmonary function tests (PFTs). They are indicated if muscle weakness could be contributing to abnormal results from routine testing, such as a low VC
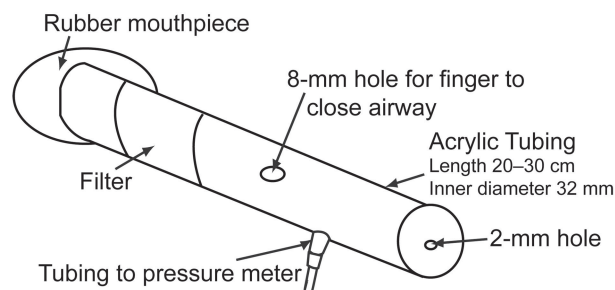

Fig. 1. Apparatus for testing maximal inspiratory pressure and maximal expiratory pressure.

without signs of obstruction or an abnormality of the flow-volume loop that is recognized to be associated with muscle weakness, or if muscle weakness is a possibility given the clinical scenario. Respiratory muscle weakness may be present in patients with dyspnea, respiratory failure, malnourishment, or debility,[2] in neuromuscular diseases such as myasthenia gravis, Guillain-Barré syndrome, amyotrophic lateral sclerosis, stroke, polio, or quadriplegia,[3-7] and in multisystem diseases such as polymyositis and sarcoidosis. MIP is used to monitor patients with acute conditions such as myasthenia gravis at risk of rapid loss of strength of the diaphragm, to follow the progress of patients with chronic diseases such as muscular dystrophy, and to detect muscle weakness in undiagnosed patients.

As for other PFTs, interpretation of results depends in part on comparison with population normal values. An important purpose of this paper is to try to make a synthesis of the many published sets of normative data. In many cases, however, comparison with normal values does not give a definite answer. Judgment must then be based on analysis of all the clinical and pulmonary function data and sometimes more extensive testing. During the last 5 years our pulmonary function laboratory in one tertiary referral center in Calgary, Alberta, Canada, performed MIP and MEP on approximately 5% of the patients having general PFTs.

Unresolved Issues

The ATS statement[1] discusses normal values only briefly, indicating that reported normal values in the literature vary considerably, probably because of differences in technique, and recommends the normal values of Wilson et al,[8] who used a protocol similar to the one proposed as the ATS standard. The ATS authors review studies of flanged versus tube mouthpieces. They make the point that with a flanged mouthpiece the values obtained are less than with a tube mouthpiece, but recommend the flanged mouthpieces as the standard because they are easier for patients to use. Since 2002, 2 large studies have added to the lit-

erature on normal subjects. Wohlgemuth et al[9] in 2003 reported MIP and MEP data using a mask. Windisch et al[10] in 2004 reported MIP data using a flanged mouthpiece and discussed methods of analyzing MIP and MEP pressure tracings. Hautmann et al[11] reported MIP in healthy subjects using "a stiff rubber mouthpiece," clarified as "an oval, rubber, flanged ("scuba") mouthpiece" in a personal communication (Hubert Hautmann MD, Pneumologie, Klinikum Innenstadt, Medizinische Klinik, Muenchen, Germany, 2008).

While reference mean values are reasonably well established, the lower limit of normal (LLN) values remain uncertain. Authors have employed a variety of statistical expressions to describe central tendency, variation, and the LLN values. The 2002 ATS statement[7] indicates that a MIP of -80 cm H_2O usually excludes clinically important inspiratory muscle weakness. However, Ruppel, in his 2003 textbook,[12] states that a MIP greater than -60 cm H_2O is normal.

Review of the Literature

Normal Values

Predicted normals and LLN values vary considerably.[8-11,13-31] There are relatively few studies for MEP, with fewer for females than males, and many of them have relatively few subjects. We reviewed all periodical sources available to us and made a selection based on the following criteria

- Study population and sampling method

- Number of subjects

- Number of test trials

- Rest time between trials

- Duration of each trial

- Definition of maximum pressure

- Maneuver used to generate maximal effort

- Number and experience of technologists performing tests

- Type of mouthpiece

- Type of pressure gauge

- Technique (including feedback to the subject and one-second duration of measurement)

- Method of defining limits of normal

Various methods are used to measure maximal respiratory mouth pressures. The choices made of mouthpiece, maneuver made by the subject, and definition of maximum pressure make a difference to the result.

Rounded "tube" mouthpieces inserted in the mouth for MIP, and placed on the lips, like a bugle, for MEP, generally give the highest values and have been used for most published reference data. Somewhat lower values are found in studies that used "flanged" mouthpieces.[8,10,11,18] By comparing MIP and MEP results with flange and tube mouthpieces in the same subjects, Koulouris et al[31] confirmed this in males and females, and Tully et al[6] found that tube MEP exceeded flange MEP in men by a mean difference of 20.7 cm H_2O. The reason for the difference is unknown. The ATS/ERS statement nevertheless recommends the flanged mouthpiece, because it ensures the least leak at the mouthpiece, particularly in subjects with weakness of the mouth muscles.[1] A mask held to the patient's face can also be used for such patients; MIP and MEP using a mask, compared to values obtained in the same patients with a flanged mouthpiece, were found to be 3% and 16% lower, respectively.[9]

The maneuver made by the subject is usually a maximal inspiration or expiration sustained for at least one second against a blocked airway, These are evidently effort-dependent tests, and the maneuvers required are not natural ones, so they are not easy for patients to perform well. For this reason, a maximal sniff pressure is sometimes used for MIP. Maximal inspiratory sniff pressure from the oral cavity proved to be 22% less than MIP from a flanged or "scuba" mouthpiece in normal subjects.[32] In dystrophy patients, after repeated visits, MIP results with the standard maneuver become similar to nasal sniff MIP.[33]

Some older studies derived the peak pressure reached during a brief maximal effort by watching the needle on the dial of an aneroid manometer. Modern apparatus displays the time course of pressure during the effort. From this can be measured the peak value, or the highest value sustained for some minimum period (plateau pressure), or the maximum mean pressure over one second. A recent comparison between peak pressure and highest value sustained for one second (plateau pressure) has shown that the plateau so defined is approximately 84% of peak pressure.[10] The ATS guidelines recommend using the maximum mean pressure over one second, which requires specific software to calculate.[1] By definition it must lie between peak and plateau pressure, but it has not been directly compared with them. Most published reference values are for 1-s plateau pressures.

We amalgamated results from published normal values for adults using flange mouthpieces to give composite mean MIP and MEP reference values as a function of age. For this purpose, only studies that report data as a function of age could be used. Reports of multiple regressions were also difficult to integrate into a composite. Studies using older aneroid manometers were omitted. Excluded studies are presented in Table 1, where sources of normal data for tube mouthpieces may be found.

Table 1.   Studies Excluded From This Review of Flanged Maximal Inspiratory Pressure and Maximal Expiratory Pressure

First Author	Year	Subjects (n)	Mouthpiece Type	Gauge Type	Notes
Arora[2]	1982	16	Tube	Aneroid	Unclear technique
Berry[15]	1996	101	Tube	Both	Varying gauges with data pooling
Black[23]	1969	120	Tube	Aneroid	Unclear technique
Bruschi[20]	1992	625	Tube	Electric	Multivariate predictors
Carpenter[29]	1999	13,005	Tube	Electric	Multivariate predictors
Charfi[24]	1991	253	Tube?	Electric	No pressure-age relationship
Cook[26]	1964	56	Tube	Aneroid	—
Harik-Khan[19]	1998	267	Tube	Electric	Multivariate predictors
Karvonen[17]	1994	200	Tube	Aneroid	Age grouping, not continuous
Leech[21]	1983	924	Flange	Electric	Physiologically untenable LLN
McConnell[32]	1999	41	Flange	Electric	Multivariate predictors
McElvaney[16]	1989	104	Tube	Electric	No pressure-age relationship
Ringqvist[25]	1966	110	Not stated	Electric	Mouthpiece not described
Rubinstein[27]	1988	28	Tube	Electric	MEP (only) from FRC
Tully[6]	1997	50	Both	Electric	MEP only in spinal-cord-injured patients: not repeated measures

LLN = lower limit of normal
MEP = maximal expiratory pressure
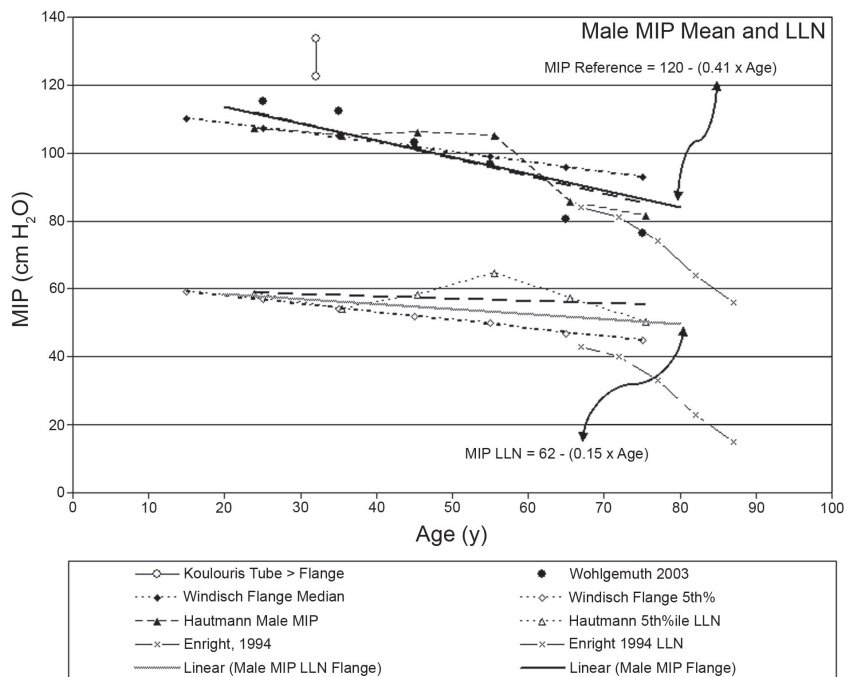FRC = functional residual capacity

Fig. 2. Relationship between reference and lower limit of normal (LLN) in male maximal inspiratory pressure (MIP) versus age.

Male and female data and norm sets are reported separately. Male exceeds female MIP by 34–66%, and male exceeds female MEP by 41–57%, depending on age.[11,17-19] Aside from sex, the correlation with age is the strongest in most of the studies mentioned[11] and was therefore used as the independent variable in our plots and prediction equations. For male and female MIP we used the weighted means of the slopes and intercepts for flange mouthpiece data of Hautmann et al[11] and Windisch et al.[10] Since the number of subjects tested was high, an electronic trans-
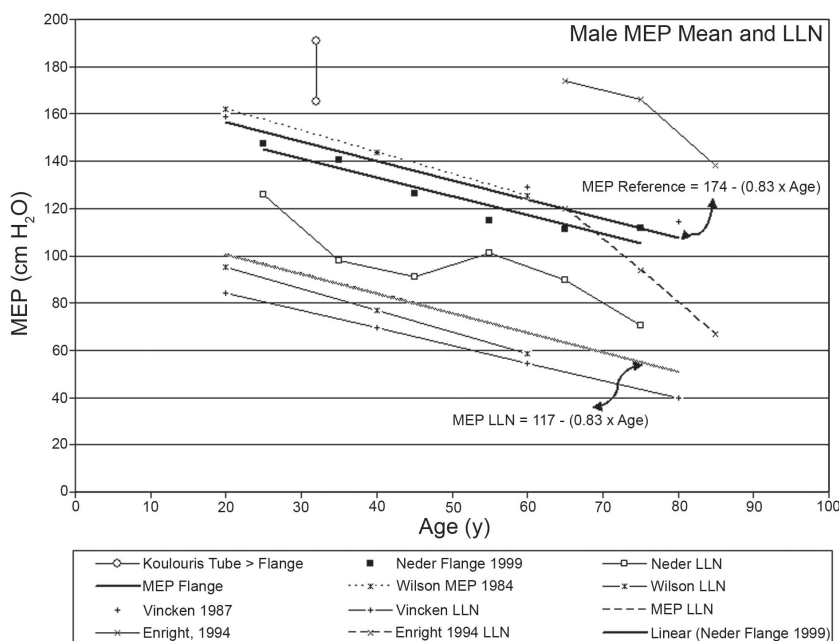
Fig. 3. Relationship between reference and lower limit of normal (LLN) in male maximal expiratory pressure (MEP) versus age.

ducer was used to obtain a one-second plateau value, and a flanged mouthpiece allowed testing without leaks of air. Both used the 5th percentile for LLN, while Wilson et al[8] and Vincken et al,[18] smaller studies, did not. Similarly, for male MEP we recommend the weighted means of the slopes and intercepts for flange mouthpiece data from Vincken et al,[18] Wilson et al,[8] and Neder et al.[28] (Hautmann et al[11] and Windisch et al[10] looked only at MIP). For female MEP we used the data of Neder et al,[28] since they were the only ones reporting a slope with age. They used an aneroid transducer but had a chart readout and took the peak pressure from tracings that lasted at least one second. We plotted predicted lines for flanged mouthpieces and derived a predicted normal line with the MEP versus age for each of the male and female data from the selected studies (male MIP, male MEP, female MIP, and female MEP) (Figs. 2 to 5). The difference between the tube and flange mouthpieces in the same subjects, as determined by Koulouris et al,[31] is shown on each plot. The range for these predicted mean values, the percent of predicted, and the LLN values are plotted for age 20–80 years.

Lower Limit of Normal

In medical practice, mean normal population values are of very little interest. To decide whether a patient has pathological weakness of respiratory muscles, the value of interest is the LLN. Unfortunately, it has not been possible to establish any but vague LLN values for MIP and MEP. To decide whether the muscles are weak enough to explain

either dyspnea or a low VC may require not only MIP or MEP but additional measurements, such as a lung pressure-volume curve or an exercise test.

Limits for the range of normal are usually given as mean less 2 standard deviations or 95% of the mean, which assumes a normal distribution. A possible problem is that the distribution may be significantly skewed, especially in older patients, where normal mean MIP and MEP are small, because the distribution cannot go below zero. For such populations a better indicator is the lower 5th percentile, which does not assume the distribution is symmetrical about the mean. The calculation has to make an assumption about whether variance of the distribution is the same at all ages. Since the degree of skewness cannot be assumed to be the same at all ages, the better indicator theoretically is the 5th percentile in each age group, as reported by Hautmann et al[11] with MIP. However, a very large number of subjects would be required to estimate this with any accuracy. Confidence intervals for any proposed cutoff value must therefore be very broad. The LLN lines shown in Figures 1 to 4 are based on $1.96 \times SD$, except for the data of Windisch et al[10] and Hautmann et al.[11]

It is important to remember that MIP and MEP are generated by muscles that are used for many purposes other than respiration. Their strength is not determined primarily by requirements for breathing. If MIP or MEP falls below the LLN, it may still be well above that needed to maintain normal VC and to support breathing during exercise. However, the minimum possible values of MIP
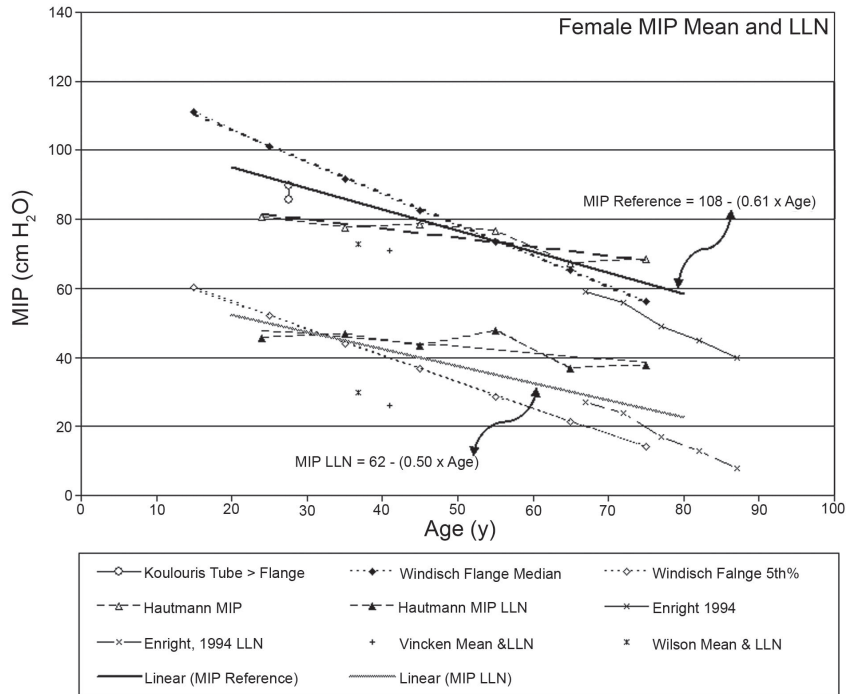
Fig. 4. Relationship between reference and lower limit of normal (LLN) in female maximal inspiratory pressure (MIP) versus age.
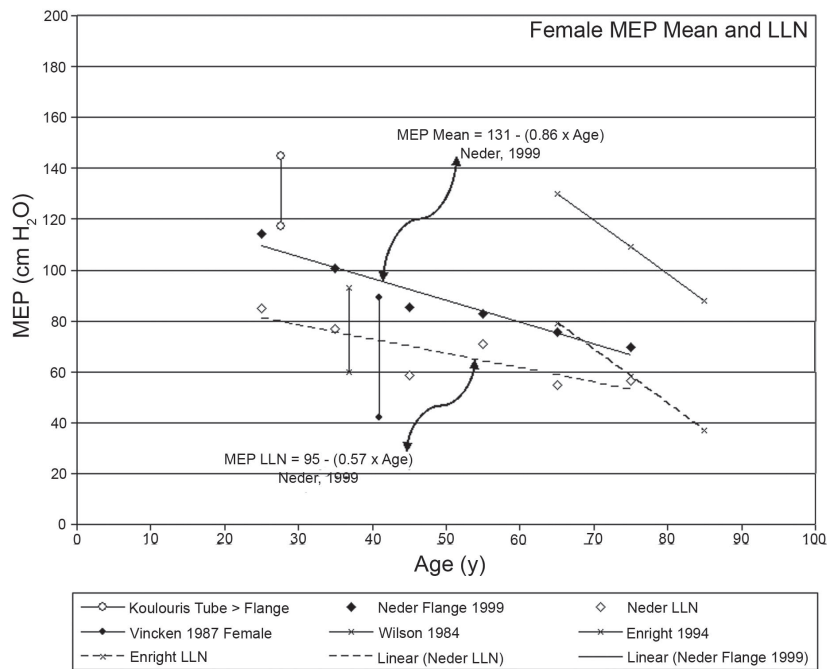
Fig. 5. Relationship between reference and lower limit of normal (LLN) in female maximal expiratory pressure (MEP) versus age.

and MEP are those required to sustain respiration at rest. Higher values are needed to support exercise.

Most studies show a decline in mean MIP and MEP with age, and the slope of decline with age is similar from one study to another. However, it is not so clear that the LLN changes with age. Review of all the data suggests that the LLN for MIP is constant up to age 70, approximately 60 cm H_2O for men and 40 cm H_2O for women.

Table 2. Prediction of Maximal Mouth Pressures in Adults With a Flanged Mouthpiece

Male MIP reference = 120 – (0.41 × age)	Male MIP LLN = 62 – (0.15 × age)
Male MEP reference = 174 – (0.83 × age)	Male MEP LLN = 117 – (0.83 × age)
Female MIP reference = 108 – (0.61 × age)	Female MIP LLN = 62 – (0.50 × age)
Female MEP reference = 131 – (0.86 × age)	Female MEP LLN = 95 – (0.57 × age)

MIP = maximal inspiratory pressure
LLN = lower limit of normal
MEP = maximal expiratory pressure

Certainly, there are not enough data to refute that possibility. It is physiologically plausible, given that the respiratory muscles, certainly in normal young people, are much stronger than needed for respiration, because they are used for non-respiratory purposes. But even deconditioned subjects must have the minimum strength reserve needed for respiration, and that will not change much with age. Mc-Connell et al[32] calculated from known resistance and elastance of the respiratory system plus data on fatigability of respiratory muscles that 35 cm H_2O should be the minimum MIP compatible with sustained exercise with oxygen consumption of 1 L per minute. A lower value would suffice for a sedentary person. Such an approach is probably more valid than looking at population statistics, but still needs to be supported by clinical data. Experience in intensive care indicates that MIP must be greater than about 20 cm H_2O before a patient can be successfully weaned from a ventilator.

For MIP on flange mouthpieces we suggest combining the Hautmann et al[11] and Windisch et al[10] values for LLN. This is not much different from the numbers given by the Enright et al studies on old people.[13,14] Because of the wide confidence intervals on any value of the LLN, it seemed most reasonable to choose simple numbers and straight-line approximations, as we have calculated in Table 2.

Values of MIP below the LLN, like all "abnormal" values in PFTs, have to be interpreted in relation to the clinical situation. The LLN is a statistical definition derived from studies of normal people. Of these normal subjects, none of whom had a clinical problem, 5% had results outside the range defined as normal. A low MIP may therefore be found in a normal subject. On the other hand, a person who begins by being very strong, with a value of MIP well above average, can suffer a major loss of respiratory muscle strength due to disease and still be able to generate MIP well within the normal range. As with other respiratory function tests, MIP is much more reliable for following the progress of a disease by repeated testing than for deciding at the outset if muscles are affected by a disease.

In practice, MIP is usually performed only in patients with a recognized or suspected respiratory muscle problem. They may have a low VC, with no obvious explanation, or dyspnea on exertion of unknown cause, or a known muscle disease where the question is whether the respiratory muscles are affected, or unexplained ventilatory failure. A patient with systemic lupus erythematosus who has shortness of breath, a low VC, and a chest radiograph showing normal but small lungs (vanishing lung syndrome), needs only a MIP low enough to cause a decrease in VC to confirm paralysis of the diaphragm, a recognized complication of the disease. A patient with fatigue and an unexplained high arterial P_{CO_2} with a low MIP may have a rare muscle disease, such as acid maltase deficiency. On the other hand, a person with normal exercise capacity and normal pulmonary function whose MIP is somewhat below the LLN can be considered normal. When a patient with reasons for suspecting weakness of respiratory muscles has inconclusive results, repeating the tests at intervals may show progression and confirm a diagnosis.

Maximal Inspiratory Pressure and Maximal Expiratory Pressure in the Elderly

For older subjects there is more uncertainty in predicting values for diverse reasons. In most studies the number of normal subjects older than 75 years is small. When subjects are grouped according to age, the oldest group in the study is often listed simply as ≥ 74.9 years,[19] > 70 years,[9-11,16] or 85+ years.[13] In addition, it is likely that the relationship of MIP to age is nonlinear, having a greater negative slope when age exceeds 60 years. Disagreement among studies about this conclusion can be explained by selection and small numbers of subjects. Some small studies did not find a significant relationship between MIP and age in older subjects.[34] The larger studies of older people by Enright et al[14] and Wijkstra et al[22] found greater negative slopes than those reported for whole sets of subjects with ages ranging from 20 to 70 or 80 years. A huge and definitive study reported by Carpenter et al[29] was done with cardboard tube mouthpieces held between the lips on 13,005 subjects age 47–68 years, as part of the Atherosclerosis Risk in the Community (ARIC) project. Mean peak MIP at age 55 was above the values obtained in studies with flange mouthpieces: about 10 cm H_2O for men, and about 6 cm H_2O for women. MIP showed very similar declines with age, of 1.1 cm H_2O per year in men, and 0.9 cm H_2O in women. These slopes are greater than those for populations that run in age from 20 to 60 or 70 years, and close to the values reported by Enright at al[14] and Wijkstra et al.[22]

These studies verify that there is a decline in MIP with age, and that extrapolation of regression lines for MIP against age in younger populations is not a valid way to estimate normal values for older people. They provide reliable values for mean MIP in male and female subjects for ages up to about 70 years. However, age and sex account for only a small percentage in the observed variation between subjects. The ARIC study found that male current smokers averaged about 10 cm H_2O less than never-smokers; that there was a curvilinear relation between body mass index and MIP, with both low and high body mass index associated with lower MIP than intermediate values; and that height was positively correlated with MIP. Even with these and other factors taken into account, a great deal of the variance in MIP was left unexplained.

The best available estimates for mean MIP in normal older people using tube mouthpieces are those reported by Enright et al as a function of age.[14] They had very few male subjects over 80 years old, however. It is reasonable to use the age-MIP relations from the ARIC study with a correction for the bias produced by a different mouthpiece.

Maximal pressures in older subjects seem to depend more on fitness of the subject than on age per se. Rendas et al[35] studied 2 groups of 25 height, weight, body mass index, and age matched women, of mean age of 67 years. The control group was sedentary while the active group performed aerobic and gymnastic activity. MEP was 23% higher in the active group, although the difference in MIP was not statistically significant. Berry et al[15] found that MIP and MEP correlated with hand-grip strength in older men and women. Watsford et al[34] found that older people with higher fitness, evaluated via a walking performance test, had better respiratory muscle strength. McConnell et al,[32] in subjects with mean age 70 years, found poor correlations of maximal pressures with age, height, and weight, but a strong correlation with habitual physical activity. The large ARIC study also showed a strong correlation with physical activity.[29]

Lower limits of normal are even less well defined for people over 65 years than for younger ones. McConnell et al[32] give an extensive and thoughtful discussion of the problem of judging the importance of low MIP values, and Zeleznik[36] offers sound general advice on evaluation of respiratory function in older people, emphasizing that they have more variation in physiologic measurement and varying risk factors for disease.

Maximal Inspiratory Pressure and Maximal Expiratory Pressure in Different Races

Few studies have looked systematically at racial or ethnic differences in mouth pressures. Chinese men in Singapore had higher MEP than Malays and Indian men, and probably higher MIP than Malays, while mean MIP was lower than published values for whites; there were no differences for women.[37] In North America, Carpenter et al[29] found no difference between whites and Afro-Americans, although they may have missed a small difference because their large study was not designed to look specifically at race, and in elderly subjects Enright et al[13] found no differences.

Relationship Between Vital Capacity and Respiratory Muscle Strength

How do VC and respiratory muscle strength interact in neuromuscular disease? Vincken et al[18] studied 2 groups of neuromuscular patients, one with normal muscles (76–122% of predicted), as measured via MIP and MEP, and a second with weak respiratory muscles (16–61% of predicted) combined with restriction, normal diffusion, and inspiratory and expiratory flow limitation. The second group showed a 25% reduction in VC corresponded to a 60% reduction in MIP in neuromuscular patients. De Troyer et al[3] and Estenne et al[38] showed similar effects, illustrated in Figure 6, which illustrates the theoretical normal and abnormal curvilinear relationships between VC and respiratory muscle weakness (measured via pleural pressure). However, they emphasized that chronic muscle weakness is often associated with reduced pulmonary compliance, which makes VC less insensitive than expected as an indicator of weakness.

Considering disproportionately greater decrements in MIP than in VC, Demedts et al[39] investigated 2 groups of patients with moderate neuromuscular disease who had virtually no respiratory complaints. By fluoroscopy, group A patients had radiologically preserved diaphragm displacement; group B had a maximal diaphragm displacement of less than 4 cm and generally sluggish movement. Group A had an average VC of 88% of predicted but MIP of 57% and MEP of 60% of predicted (from functional residual capacity [FRC]), while group B showed a somewhat lower average VC of 75% but a very low average MIP of 46% and MEP of 26% of predicted. They concluded that maximal mouth pressures were disturbed earliest and were the single most sensitive test for respiratory muscle weakness.

Further decrements in VC and respiratory muscle strength indicate progressive respiratory dysfunction. Braun et al[40] concluded that patients with uncomplicated neuromuscular disease are likely to have hypercapnic respiratory failure if MIP is less than 30% of predicted, but in this situation a VC below 55% of predicted is equally useful as an indicator.

Technique

The method of doing the tests varies: ATS[1] covers most details of the technique. For optimal results, testers
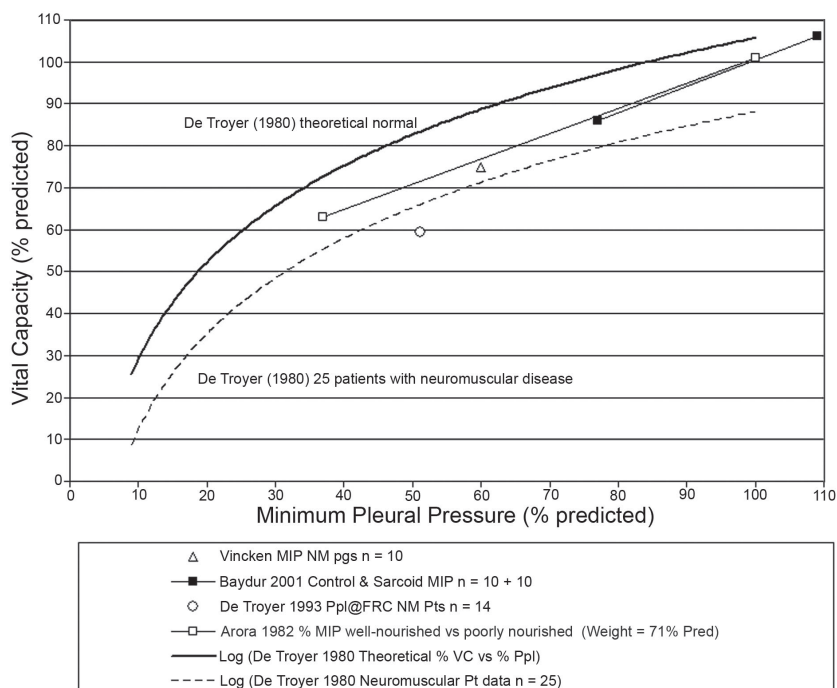
Fig. 6. Relationship between percentage vital capacity (VC) and percentage pressure in patients with and without neuromuscular disease. The solid curve is a theoretical curve with normal pressure-volume relationship, normal chest wall, and uniform inspiratory and expiratory muscle involvement. The dashed line is a log regression of patients with various neuromuscular diseases. P_{pl} = pleural pressure. MIP = maximal inspiratory pressure. FRC = functional residual capacity. NM = neuromuscular disease. Pt = patient. (Based on data from References 1–4 and 38.)

need to consider the points in Table 3. A particular factor affecting results is operator dependence. In the ARIC study of 13,000 people,[29] 27 specifically trained technicians took part, each one testing on average 480 subjects. The mean value of MIP obtained by one technician varied from 71 cm H_2O to 103 cm H_2O. (Omitting the lowest as an outlier, the range was still from 82 cm H_2O to 103 cm H_2O). Not only some outliers, but also the population means for MIP in series of several hundred subjects may have been affected by technician performance. Examples are the 2 papers by Enright et al,[13,14] both with large numbers of subjects in older age groups. The sample populations are not quite the same, but are not expected to have different MIP values. Mean values from the study where the operators were regular PFT technicians were nevertheless about 12 cm H_2O lower than from the one where operators were specially trained to do MIP. Another example is in the study by Leech et al[21] in young adults that found much lower pressures than other authors, which is hard to account for by anything except the operators. In cases where a low MIP result is important in the clinical evaluation, a repeat test with a second experienced technician should be considered. Technicians need to carefully judge radical outliers, which need to be eliminated

if the pressure reading showed no gradual increase to the plateau value, suggesting a sharp peak value.

As part of a PFT or as ordered alone, the MIP and MEP may be recorded using a portable electronic pressure meter. As part of a pulmonary neuromuscular test battery, the MIP and MEP can be done on a recording system with pressure-time waveforms to optimize validity of measurement.[1]

Interpretation of Results

Normal and Abnormal Mouth Pressures

1. The normal values and especially the LLN values are not securely based and should be regarded as rough guides. This is especially true for people over 70 years of age.

2. Respiratory muscles have many non-respiratory purposes and may be much stronger than needed simply for respiration. A pressure well below the population mean – 2 SD may be sufficient to give a normal VC, especially in young people.

3. Inspiratory pressures are largest at residual volume, smallest at TLC. The difference between values measured at residual volume and FRC is relatively small, about 16%.[39] This is helpful because patients who are generally weak

Table 3.   Emphases on Technique and Performance With Maximal Inspiratory Pressure and Maximal Expiratory Pressure

Issue	Recommendation	References
Type of mouthpiece	Prefer flange to tube	1, 6, 31
Leak at the mouth	Seal with fingers during MEP	24
Glottic closure	2-mm hole in apparatus, and 20–30 cm length	1, figure 1
Volume at which performed	MIP from RV, MEP from TLC	1
Duration	1-s plateau in 3–4-s effort	1
Number of efforts	Prefer 5 to ATS recommendation of 3	1
Time between efforts	1 min between trials	8-11, 29, 31
Criterion for stopping	Highest pressure varying < 20%	1
Operator dependency	In-services, coaching technique	None
Diurnal variation	Not important	41
Order of MIP and MEP	MIP trials before MEP	42
Patient position	Sitting is safer than standing. Sitting upright is preferred. If inclined in a bed, record the approximate angle in report.	43
Patient fatigue	Wait or rebook with patients with dyspnea during PFT	None
MEP cheek "ballooning"	Support cheeks with fingers and palms	1

MEP = maximal expiratory pressure
MIP = maximal inspiratory pressure
RV = residual volume
TLC = total lung capacity
ATS = American Thoracic Society
PFT = pulmonary function testing

cannot get down to predicted residual volume, and so have their MIP measured from FRC.

4. In muscle weakness, the loss in VC may be more than expected for a given loss in MIP. There are at least 2 reasons for this: people with chronic weakness develop some lung stiffness, or loss of compliance, even if they have no primary lung pathology; they do not get as much volume for a given negative pleural pressure as normal. When muscles shorten they lose force through the force-length relationship. If they have asymmetrical muscle weakness (eg, intercostal but not diaphragm loss), when they inspire they get paradox (eg, rib cage goes in, diaphragm descends much more than it would normally for the same tidal volume) and can therefore lose pressure more rapidly with increase in volume during inspiration than a normal subject.

5. Some patients have a muscle disease that affects mainly endurance. In that case they can get dyspnea or hypoventilation in exercise but have normal results for VC and MIP.

6. When in doubt, MIP and MEP need to be supplemented by additional tests, such as exercise, compliance, transdiaphragmatic pressure, electromyogram, phrenic-nerve conduction, and sleep studies.

There are 3 different questions that mouth pressure may help to answer

Are the respiratory muscles at all weak? For this, reference should be made to the predicted values for normal and LLN, as shown in Table 1 and Figures 2 to 5. A useful rule of thumb is that muscles are probably weak if MIP is less than 60% of predicted.

Are the muscles weak enough to be responsible for a low VC? In patients with chronic neuromuscular disease, male MIP less than 60 cm H_2O or female MIP less than 40 cm H_2O could be responsible for a VC less than 80% of predicted. When there is a low VC and a question of either muscle weakness or restrictive lung disease as a cause, it should be helpful to measure a lung pressure-volume curve from FRC to TLC. In normal subjects at TLC, inspiration is halted, not because the muscles have reached their maximum effort, but because the lungs just cannot be expanded any further. This shows up in the graph of lung volume against the pressure expanding the lungs, which is mouth pressure minus pleural pressure. This graph is a curve that ascends steeply from FRC but flattens out at TLC, with further increases in pressure giving no further increase in volume. If a patient has TLC limited by a disease that causes increased lung stiffness, the lung pressure-volume curve will flatten out at the patient's TLC. If the low TLC is caused by weak muscles, however, the curve will still be rising when the patient stops at TLC. The problem is failure to make more pressure, rather than lungs that just won't expand further. In some diseases, polymyositis, for example, TLC may be limited by both stiffness of lungs and weakness of respiratory muscles. As well, in chronic neuromuscular disease, the lungs tend to become somewhat stiffer and contribute to the loss of TLC and VC (see Fig. 6).

Are the muscles weak enough to be responsible for dyspnea? Patients with progressive weakness of respiratory muscles would be expected to develop dyspnea on exertion before muscles became weak enough to limit VC, but there is no quantitative data from which to draw cutoff values for this. In patients with sarcoidosis,[44] chronic obstructive pulmonary disease,[45] and general cardiorespiratory diseases,[46] lower MIP is correlated with reduced exercise tolerance, but exercise may be limited by endurance of leg muscles rather than respiratory muscles.

Discussion

Assessment of respiratory muscle function is difficult and complicated. It begins by examining the patient, with careful study of how the chest wall moves and which observable respiratory muscles seem to be active. This is followed by routine PFTs, MIP, and MEP. Further, more elaborate or invasive tests that are often needed are not covered in this review.

Maximum inspiratory and expiratory pressures are simple to do and a helpful indicator of muscle weakness. They are also good for repeated testing to follow the course of disease. Interpretation, as for all PFTs, must be based on the clinical situation. They measure the combined effect of all muscles used in the maximal effort, rather than just the diaphragm in inspiration. In expiration they assess a large array of muscles, many of which probably do not play an important role in normal breathing. All of the muscles used for breathing also serve other functions, and their strength may depend to a large extent on what is needed for these functions, rather than for breathing. Patients with some muscle diseases can have MIP in the normal range but develop fatigue abnormally rapidly on exercise. Some patients with MIP below the LLN are just poor performers of the test, and some have completely normal breathing. A decision about whether a patient does or does not have important respiratory muscle weakness cannot reliably be based on MIP and MEP alone.

All available normal values have been drawn from normal subjects. Since the pressure developed by respiratory muscles depends on their resting length and mechanical advantage, it is bound to be abnormal in patients with any disease that changes the resting volume and shape of the ribcage or diaphragm. For this reason, maximal pressures are of little value for assessing muscle function in these diseases, notably chronic obstructive pulmonary disease.

If weakness of respiratory muscles is present or suspected, further, more detailed tests can be done. They are reviewed extensively in the ATS statement[1] and are beyond the scope of this article. Strength and function of the diaphragm can be assessed separately by measuring transdiaphragmatic pressure by means of pressure sensors placed in the esophagus and stomach. By stimulating the phrenic nerve, the isolated muscle can be made to contract, nerve conduction can be assessed, and the question of patient effort and cooperation can be bypassed. Participation and coordination of diaphragm, intercostal, accessory, and abdominal muscles can be studied by simultaneous measurement of ribcage and abdominal expansion, along with lung volume and pleural and abdominal pressures. Exercise testing can expose problems of pathological fatigability of muscles, as illustrated in the case study by Voduc et al.[47]

Summary

The purpose of this paper is to update and amplify the recommendations of the ATS/ERS task force on respiratory muscle testing with respect to MIP and MEP. Based on a critical review of the literature and amalgamation of available data, reference equations are proposed for both mean values and LLN values according to age and sex, using a flanged mouthpiece. More data on MEP values, especially female, with a flanged mouthpiece, are needed. Details of technique are discussed. An approach to interpretation is offered, with particular attention to LLN values, results in the elderly, and addressing specific clinical questions. The information should aid optimal implementation of the findings from evidence-based literature directly into practice in a pulmonary function laboratory.

REFERENCES

1. American Thoracic Society/European Respiratory Society. ATS/ERS statement on respiratory muscle testing. Am J Respir Crit Care Med 2002;166(4):518-624.
2. Arora NS, Rochester DF. Respiratory muscle strength and maximal voluntary ventilation in undernourished patients. Am Rev Respir Dis 1982;126(1):5-8.
3. De Troyer A, Borenstein S, Cordier R. Analysis of lung volume restriction in patients with respiratory muscle weakness. Thorax 1980;35(8):603-610.
4. Baydur A, Alselek M, Louie S, Sharma O. Respiratory muscle strength, lung function, and dyspnea in patients with sarcoidosis. Chest 2001;120(1):102-108.
5. Vincken W, Elleker M, Cosio M. Flow-volume loop changes reflecting respiratory muscle weakness in chronic neuromuscular disorders. Am J Med 1987;83(4):673-680.
6. Tully K, Koke K, Garschick E, Lieberman S, Tun C, Brown R. Maximal expiratory pressures in spinal cord injury using two mouthpieces Chest 1997;112(1):113-116.
7. DePalo V, McCool F. Respiratory muscle evaluation of the patient with neuromuscular disease. Semin Respir Crit Care Med 2002;23(3):201-209.
8. Wilson S, Cooke NT, Edwards RHT, Spiro SG. Predicted normal values for maximal respiratory pressures in Caucasian adults and children. Thorax 1984;39(7):535-538.
9. Wohlgemuth M, van de Kooi EL, Hendriks JC, Padberg GW, Folgering HT. Face mask spirometry and respiratory pressures in normal subjects. Eur Respir J 2003;22(6):1001-1006.

10. Windisch W, Hennings E, Sorichter S, Hamm H, Criee CP. Peak or plateau maximal inspiratory mouth pressure: which is best? Eur Respir J 2004;23(5):708-713.

11. Hautmann H, Hefele S, Schotten K, Huber RM. Maximal inspiratory mouth pressures in healthy subjects: what is the lower limit of normal? Respir Med 2000;94(7):689-693.

12. Ruppel G. Manual of pulmonary function testing. St Louis: Mosby; 2003:72-74.

13. Enright PL, Kronmal RA, Manolio MB, Schenker MB, Hyatt RE. Respiratory muscle strength in the elderly. Am J Respir Crit Care Med 1994;149(2 Pt 1):430-438.

14. Enright PL, Adams AB, Boyle PJ, Sherrill DL. Spirometry and maximal respiratory pressure references from healthy Minnesota 65- to 85-year-old women and men. Chest 1995;108(3):663-669.

15. Berry JK, Vitalo CA, Larson JL, Patel M, Kim MJ. Respiratory muscle strength in older adults. Nurs Res 1996;45(3):154-159.

16. McElvaney G, Blackie S, Morrison NJ, Wilcox PG, Fairburn MS, Pardy RL. Maximal static respiratory pressure in the elderly. Am Rev Respir Dis 1989;139(1):277-281.

17. Karvonen J, Saarelainen S, Nieminen MM. Measurement of respiratory muscle forces based on maximal inspiratory and expiratory pressures. Respiration 1994;61(1):28-31.

18. Vincken W, Ghezzo H, Cosio MG. Maximal static respiratory pressures in adults: normal values and their relationship to determinants of respiratory function. Bull Eur Physiopathol Respir 1987;23(5):435-439.

19. Harik-Khan R, Wise RA, Fozard JL. Determinants of maximal inspiratory pressure. Am J Respir Crit Care Med 1998;158(5):1459-1464.

20. Bruschi C, Cerveri I, Zoia MC, Fanfulla F, Fiorentini M, Casali L, et al. Reference values of maximal respiratory mouth pressures: a population-based study. Am Rev Respir Dis 1992;146(3):790-793.

21. Leech JA, Ghezzo H, Stevens D, Becklake MR. Respiratory pressures and function in young adults Am Rev Respir Dis 1983;128(1):17-23.

22. Wijkstra PJ, Van der Mark TW, Boezen M, Van Altena R, Postma DS, Koeter GH. Peak inspiratory mouth pressure in healthy subjects and in patients with COPD. Chest 1995;107(3):652-656.

23. Black LF, Hyatt RE. Maximal respiratory pressures: normal values and relationship to age and sex. Am Rev Respir Dis 1969;99(5):696-702.

24. Charfi MR, Matran R, Regnard J, Richard MO, Champeau J, Dall'ava J, Lockhart A. [Les pressions ventilatoires maximales a la bouche chez l'adulte: valeurs normales et variables explicatives.] Rev Mal Respir 1991;8(4):367-374. *Article in French*

25. Ringqvist T. The ventilatory capacity in healthy subjects: an analysis of causal factors with special reference to the respiratory forces. Scan J Clin Lab Invest Suppl 1966;88:5-179.

26. Cook C, Mead J, Orzalesi M. Static volume-pressure characteristics of the respiratory system during maximal efforts J Appl Physiol 1964;19(5):1016-1022.

27. Rubinstein I, Slutsky A, Rebuck A, McClean P, Boucher R, Szeinberg A, Zamel N. Assessment of maximal expiratory pressure in adults. J Appl Physiol 1988;64(5):2215-2219.

28. Neder JA, Andreoni S, Lerario MC, Nery LE. Reference values for lung function tests. II: maximal respiratory pressures and voluntary ventilation. Braz J Med Biol Res 1999;32(6):719-727.

29. Carpenter MA, Tockman MS, Hutchinson RG, Davis CE, Heiss G. Demographic and anthropometric correlates of maximum inspiratory pressure. Am J Respir Crit Care Med 1999;159(2):415-422.

30. Chen H-I, Kuo C-S. Relationship between respiratory muscle function and age, sex, and other factors. J Appl Physiol 1989;66(2):943-948.

31. Koulouris N, Mulvey DA, Laroche CM, Green M, Moxham J. Comparison of two different mouthpieces for the measurement of PI_{max} and PE_{max} in normal and weak subjects. Eur Respir J 1988;1(9):863-867.

32. McConnell AK, Copestake AJ. Maximum static respiratory pressures in healthy men and women: issues of reproducibility and interpretation. Respiration 1999;66(5):251-258.

33. Terzi N, Orlikowski D, Fermanian C, Lejaille M, Falaize L, Louis A, et al. Measuring inspiratory muscle strength in neuromuscular disease: one test or two? Eur Respir J 2008;31(1):93-98.

34. Watsford ML, Murphy AJ, Pine MJ, Coutts AJ. The effect of habitual exercise on respiratory-muscle function in older adults. J Ageing Phys Act 2005;13(1):34-44.

35. Rendas AB, Gamboa T, Ramilo T, Botelho AS, Barbara C, Mota-Carmo M. Respiratory muscle function in physically active elderly women. Arch Geron Geriat 1996;22(2):123-130.

36. Zeleznik J. Normative aging of the respiratory system. Clin Geriatr Med 2003;19(1):1-18.

37. Johan A, Chan C, Chia H, Chan O, Wang Y. Maximal respiratory pressures in adult Chinese, Malays, and Indians. Eur J Respir Dis 1997;10(12):2825-2828.

38. Estenne M, Gevenois P, Kinnear W, Soudon P, Heilporn A, De Troyer A. Lung volume restriction in patients with chronic respiratory muscle weakness: the role of microatelectasis. Thorax 1993;48(7):698-701.

39. Demedts M, Beckers J, Rochette F, Bulcke J. Pulmonary function in moderate neuromuscular disease without respiratory complaints. Eur J Respir Dis 1982;63(1):62-67.

40. Braun N, Arora N, Rochester D. Respiratory muscle and pulmonary function in polymyositis and proximal myopathies. Thorax 1983;38(8):616-623.

41. Aguilar X, Fiz JA, Texido A, Vilata P, Abad J, Richart C, Morera J. Maximum inspiratory and expiratory pressures have no daytime variation in healthy men. Respir Med 1996;90(4):231-233.

42. Fiz JA, Carreras A, Aguilar J, Gallego M, Morera J. Effect of order on the performance of maximal inspiratory and expiratory pressures. Respiration 1992;59(5):288-290.

43. Fiz JA, Texido A, Izquierdo J, Ruiz J, Roig J, Morera J. Postural variation of the maximal inspiratory and expiratory pressures in normal subjects. Chest 1990;97(2):313-314.

44. Wirnsberger R, Drent M, Hekelaar N, Breteler M, Drent S, Wouters E, Dekhuijzen P. Relationship between respiratory muscle function and quality of life in sarcoidosis. Eur Respir J 1997;10(7):1450-1455.

45. Wijkstra P, TenVergert E, Van de Mark T, Postma D, VanAltena R, Kraan J, Koeter G. Relation of lung function, maximal respiratory pressure, dyspnoea, and quality of life with exercise capacity in patients with chronic obstructive pulmonary disease. Thorax 1994;49(5):468-472.

46. Hamilton A, Killian K, Summers E, Jones N. Muscle strength, symptom intensity, and exercise capacity in patients with cardiorespiratory disorders. Am J Respir Crit Care Med 1995;152(6 Pt 1):2021-2031.

47. Voduc N, Webb KA, D'Arsigny C, McBride I, O'Donnell DE. McArdle's disease presenting as unexplained dyspnea in a young woman. Can Respir J.2004;11(2):163-167.