Key points

- Dyspnoea is influenced by inspiratory muscle strength and the load placed upon the inspiratory muscles.
- Dynamic hyperinflation is a major cause of dyspnoea and exercise intolerance in patients with expiratory flow limitation due to its detrimental effect upon inspiratory muscle loading.
- Specific IMT improves inspiratory muscle strength, reduces dyspnoea and improves exercise tolerance, even in individuals without inspiratory muscle weakness or hyperinflation.
- Pressure threshold IMT is the most reliable, convenient and commonly used method of IMT, eliciting improvements in a wide range of muscle functional characteristics, including strength, shortening velocity, power and endurance.
- Inspiratory muscles adhere to the same training principles as other skeletal muscles, with respect to overload, specificity and reversibility.
- Training loads must exceed 30% of inspiratory muscle strength, with at least once daily training and weekly increases in training load. Programmes should be least 6 weeks in duration, after which frequency can be reduced to two sessions, three times per week.
- IMT can be implemented as a stand-alone intervention or as part of a comprehensive programme of rehabilitation.
- Monitored outcomes should include inspiratory muscle strength, an index of dyspnoea (*e.g.* BDI/TDI and/or Borg CR-10) and exercise tolerance (*e.g.* 6MWD).

Inspiratory muscle training in obstructive lung disease:

how to implement and what to expect

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Statement of interest
A.K. McConnell declares a beneficial interest in the POWERbreathe® inspiratory muscle trainer in the form of a share of licence income to the University of Birmingham (Birmingham, UK), as well as acting as a consultant to Gaiam Ltd (Leamington, UK).

Educational aims

- To provide an overview of the role of respiratory muscle function in the genesis of dyspnoea.
- To describe the response of inspiratory muscles to different types of training stimuli.
- To offer guidance on the implementation and monitoring of IMT.

Summary

Dyspnoea is strongly influenced by respiratory muscle function. Patients with obstructive lung disease become hyperinflated and experience an associated functional deficit in inspiratory muscle function, as well as a concomitant increase in the work of breathing. These changes result in a heightened sense of respiratory effort and a propensity for inspiratory muscle fatigue. There is now convincing evidence that specific inspiratory muscle training (IMT) improves respiratory muscle function, reduces dyspnoea and improves exercise tolerance. This review will describe the two most commonly implemented methods of IMT, and the specific functional adaptations that are elicited by each. It will also describe successful, evidence-based implementation and monitoring of the most commonly used method of IMT.

Glossary

Dynamic hyperinflation: a term that incorporates the influence of expiratory flow limitation (EFL) upon both resting and exercising lung volumes. Premature airway closure leads to an inability to achieve a condition in which all of the forces acting on the lung are in equilibrium (the true relaxation volume). This produces an increase in end-expiratory lung volume (EELV) and a reduction in inspiratory capacity. During exercise, when the demand for expiratory flow is increased, EFL induces an adaptive response that increases EELV still further in an attempt to minimise EFL. These are two different, but related, processes that may be present to differing degrees in different patients.

The lactate threshold: corresponds to an exercise intensity above which there is a progressive accumulation of the metabolite lactate in the circulating blood. Accumulation is brought about by an imbalance between production and catabolism of lactate such that production exceeds catabolism. Lactic acid is a potent stimulus to the respiratory system, inducing a compensatory hyperventilation that acts to minimise acid–base disturbance.

Dyspnoea, exercise limitation and reduced quality of life are common features of obstructive pulmonary disease. Sense of respiratory effort (dyspnoea) contributes to exercise limitation in healthy people, as well as patients with respiratory disease [1]. The first unifying hypothesis to explain the mechanistic basis of dyspnoea was that proposed by Moran Campbell's group in the 1960s. Campbell coined the term "length–tension inappropriateness" (LTI) to explain how the sensation of dyspnoea might be "transduced" to consciousness [2, 3]. Although dated, the paradigm has stood the test of time (see [3] for further details). Under the LTI paradigm, the intensity of dyspnoea is increased when changes in respiratory muscle length (*i.e.* volume) or tension (*i.e.* pressure) are inappropriate for the outgoing motor command, and/or when the requirement for respiratory work becomes excessive. These conditions prevail in obstructive lung disease, where alterations in inspiratory muscle function and respiratory mechanics disturb the normal inter-relationship of motor outflow and the mechanical response to that outflow, creating inappropriateness. Similarly, physical deconditioning, inefficient breathing patterns and gas exchange abnormalities increase the ventilatory requirement for exercise, creating a high demand for respiratory muscle work.

The single most important factor contributing to an increased requirement for inspiratory motor drive during exertion in obstructive lung disease is likely to be the dynamic hyperinflation generated by expiratory flow limitation (EFL) [4]. In the later stages of chronic obstructive pulmonary disease (COPD), this is exacerbated by development of static hyperinflation, due to irreversible structural deteriorations, such as loss of alveolar tethering, formation of bullae and/or loss of lung elasticity [5]. The requirement to breathe at higher ranges of the total lung capacity (reducing inspiratory capacity) increases the elastic load presented to the inspiratory muscles by the lungs and chest wall creating a "restrictive" ventilatory deficit. Hyperinflation and/or EFL also exacerbate inspiratory muscle loading in a further number of ways:

- by inducing functional weakening of the inspiratory muscles (caused by foreshortening of expiration, which flattens the diaphragm and moves the inspiratory muscles to a weaker portion of their length–tension relationship [6, 7])
- by forcing inspiratory time to shorten (to allow more time for expiration, which moves the inspiratory muscles to a weaker portion of their force–velocity relationship) [8]
- by generating intrinsic positive end expiratory pressure, whereby expiration ends before all of the forces acting on the lung are in equilibrium, so inspiration is initiated under a positive expiratory load [9]).

In addition, the ventilatory requirement for exercise is increased in patients with COPD by a range of factors, including increased dead space, low tidal volume and low lactate threshold [10], as well as the systemic manifestations of the disease. The latter include abnormal peripheral muscle structure/function and reduced muscle oxidative enzyme activities [11].

Thus, mechanical and metabolic/systemic deficits associated with COPD (impaired inspiratory muscle function and an increased requirement for ventilation) create a mismatch between the demand for inspiratory muscle work and the capacity to meet that demand. These conditions would seem to be ideal for implementation of an intervention that can increase capacity and restore its balance with demand, for example inspiratory muscle training (IMT).

Interpretation of the literature relating to IMT in patients with COPD has been hampered by some studies with inadequate experimental designs; flaws have included a failure to apply basic training theory. The negative outcomes of these studies contributed to early scepticism about the value of IMT. However, the most recent meta-analysis of IMT supports the notion that specific IMT increases inspiratory muscle strength, reduces dyspnoea and improves functional exercise capacity in patients with COPD [12]. LOTTERS *et al.* [12] examined 15 studies, seven of which incorporated measures of dyspnoea. Studies included in the analysis had randomised, controlled designs and set training loads ≥30% of the maximum strength of the inspiratory muscles in the treatment group. The effect sizes for changes in dyspnoea during exercise and daily activities (assessed using Baseline and Transition Dyspnoea Indexes (BDI and TDI, respectively) [13]) were statistically significant and the largest of the outcomes assessed. This positive influence of inspiratory muscle strengthening upon dyspnoea is also supported by observations in healthy young people [14, 15], where pressure threshold IMT has also been associated with a reduced whole body effort sensation and improved athletic performance (see [16]).

Collectively, these data support the notion that IMT attenuates respiratory effort sensation, irrespective of the functional status of the inspiratory muscles.

Techniques of IMT

The two most commonly used modes of inspiratory muscle training to have been implemented in patients with obstructive lung disease are inspiratory flow resistive loading and inspiratory pressure threshold loading. These training modes, their evidence base and relative merits are described below. The technique of expiratory muscle training is not covered here because there is, as yet, no clear evidence favouring its use. Interested readers are referred to a review on the merits of inspiratory and expiratory muscle training [17].

Inspiratory flow resistive loading

Inspiratory flow resistive loading (IFRL) requires individuals to inspire *via* a variable-diameter orifice, whereby, for a given airflow, the smaller the orifice the greater the resistive load. An inherent limitation of IFRL loading is that inspiratory pressure, and thus training load, varies with flow (according to a power function) and not just to orifice size [18]. Therefore, it is vitally important that breathing pattern is monitored during IFRL if a quantifiable training stimulus is to be provided. In their 1992 meta-analysis of IMT in patients with COPD, SMITH *et al*. [19] concluded that studies employing IFRL in which inspiratory flow rate was not controlled failed to elicit improvements in inspiratory muscle function. Several studies have supplemented IFRL with feedback control of flow rate (see [20]), and observed positive outcomes, including improvements in inspiratory muscle strength, dyspnoea and exercise tolerance [21–23]. However, such modifications require additional hardware, increasing the cost and complexity of implementing IMT with IFRL.

Inspiratory pressure threshold loading

Inspiratory pressure threshold loading (IPTL) requires individuals to produce an inspiratory pressure sufficient to overcome a negative pressure load and thereby initiate inspiration. Threshold loading permits variable loading at a quantifiable intensity by providing near flow independent resistance to inspiration. This can be achieved in several ways, for example, with a weighted plunger [24], a solenoid valve [25], a constant negative pressure system [26] or a spring-loaded poppet valve [27–30]. Figure 1, shows the mechanical inspiratory poppet valve held closed by the tension in the spring, which can be adjusted using a threaded adjustment knob. The spring characteristics are linear such that a given change in spring length results in the same change in valve opening pressure at all spring lengths. The valve only opens when the inspiratory pressure generated by the patient exceeds the spring tension. Expiration is unimpeded and occurs *via* the expiratory flap valve. Threshold loading has been shown to induce improvements in inspiratory muscle strength [12, 27], maximum rate of muscle shortening [15, 31, 32], maximum power output [31–33] and inspiratory muscle endurance [30, 33, 34].

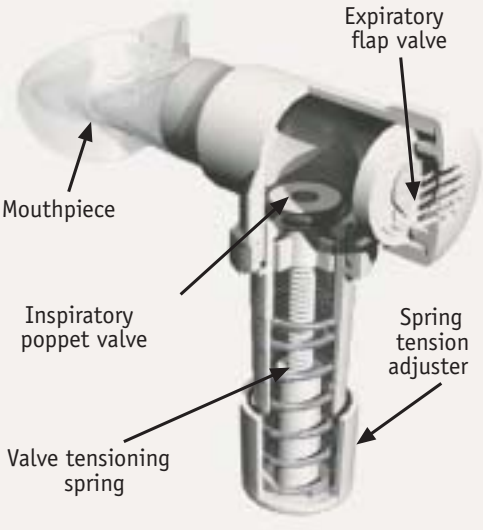

Figure 1
An exploded view of the internal construction of a mechanical pressure threshold training device.

Due to its flow independence, training using an IPTL can be undertaken effectively without the need to regulate breathing pattern. In addition, IPTL using a device with a mechanical poppet valve is both portable and easy to use, with evidence of efficacy when implemented in a domiciliary setting, as well as with long-term use

Figure 2
Commercially available pressure threshold inspiratory muscle training devices. A: The Threshold® (Respironics Inc., Murrysville, PA, USA) pressure threshold trainer; B: The POWERbreathe® (Gaiam Ltd, Leamington, UK) pressure threshold trainer.

[34]. Examples of two commercially available IPTL devices are illustrated in figure 2.

In summary, although there appears to be no difference in the efficacy of IMT using IFRL (with flow controlled) and IPTL [12, 35], the technique that has been implemented most widely is IPTL, using devices that employ a mechanical poppet valve. This is probably due to the simplicity, reliability and "user-friendliness" of these devices. They also benefit from being relatively easy to implement in placebo form for research purposes, when the threshold load can be set at a detectable, but not efficacious level.

Training principles underlying IMT

Respiratory muscles respond to the training principles established for other skeletal muscles; namely "overload", "specificity" and "reversibility" [32, 36]. The evidence supporting this statement is provided below.

Overload

To obtain a training response, the muscle fibres must be overloaded. Implicit within this principle is the concept of training duration, intensity and frequency. In the majority of reported studies in patients with respiratory disease, the duration of IMT has been 2–3 months, but structural adaptations are evident within 6 weeks [30]. A recent study in patients with COPD has demonstrated the efficacy of a 12-month programme (figure 3) [34]. Regardless of the type of training, interventions have generally been carried out in continuous bouts lasting 10–30 minutes, 1–2 times per day, for 5–7 days per week.

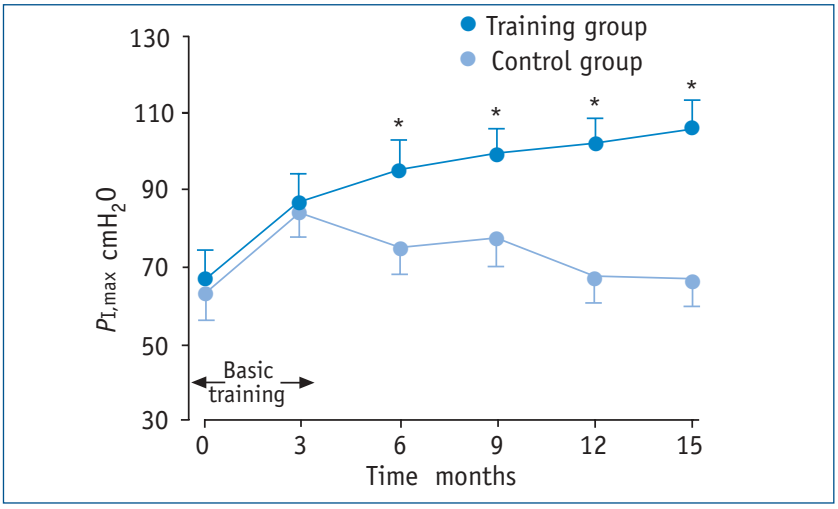

Figure 3
*Inspiratory muscle strength, as assessed by maximal mouth inspiratory pressure ($P_{I,max}$), before and following IMT (basic training 0–3 months; maintenance training in the IMT group 3–15 months; de-training in the control group 3–15 months). *: statistically significant difference between the groups. Figure modified with permission from [34].*

Data from seven studies collated by PARDY and ROCHESTER [37] suggest a significant positive relationship between the percentage increase in maximum inspiratory pressure (MIP) and the relative magnitude of the inspiratory training load (inspiratory pressure load/MIP; r = 0.85); in other words, the higher the load relative to the subject's inspiratory muscle strength, the greater the increase in strength that was induced by training. The collated data suggest that to achieve a 20% increase in MIP, a load of ≥30% MIP is required. This is supported by data from the LOTTERS *et al.* [12] meta-analysis of IMT, which included only studies where training loads had exceeded 30% of MIP. This resulted in a significant overall effect (using the fixed-effect model) upon MIP of 10.5 cmH_2O or 15%. The ineffectiveness of training loads <30% of MIP is supported by the observation of PREUSSER *et al.* [38] that MIP failed to improve significantly after 12 weeks of IMT at a load equivalent to 22% of MIP. However, although there was no significant increase in MIP, there were significant increases in indices of inspiratory muscle endurance and a modest increase in 12-minute walking distance (17 m); the magnitude of these changes was less than half that observed in the group who trained at 52% MIP. Overall, the literature supports the need for training loads to be in excess of 30% MIP.

A recent study has examined the feasibility of using high-intensity, interval-based IMT (alternating 2-minute IMT with 1-minute rest for a total of 20 minutes at a load equivalent to 68% MIP), and found it to be effective (32% increase in MIP), but no more so than conventional approaches that have employed training loads of 30–50% of MIP [39]. This area requires further systematic study in order to identify the most time- and outcome-efficient training approaches.

In the longest randomised controlled trial of IMT to date, WEINER *et al.* [34] noted the largest improvement in MIP during the first 3 months of their study (32%), followed by smaller increases (~6%) for the four subsequent 3-month blocks of IMT. LARSON *et al.* [27] observed a plateau in the improvement in MIP after 1 month of training, as did LISBOA *et al.* [40]. The existence of this "plateau" effect is also supported by data from IMT studies on healthy people, where MIP increased most rapidly during the first 3 weeks, and generally continued to improve with time, but began to plateau after ~6 weeks [14, 32]. The development of a plateau cannot be ascribed to a lack of load progression (increasing the training load to accommodate increases in MIP), since it

occurs regardless of this measure. Instead, it is a reflection of a basic property of muscle adaptation to strength training stimuli [41, 42], which necessitates periodic changes in the training stimulus in order to maintain the adaptation process; this is one of the reasons why athletes periodise their training.

Finally, a dose-dependent effect of IMT has been identified by WINKLER *et al*. [43], who found a significant positive correlation between the number of successfully completed respiratory muscle strength and endurance exercises and the improvement in respiratory muscle function in 10 patients with neuromuscular disorders ($r^2 = 0.85$ and 0.67 for MIP and maximum voluntary ventilation, respectively). This is confirmed by personal experience, and highlights the value and importance of patients maintaining IMT diaries (figure 4). However, it is pertinent to raise the related issue of training frequency at this point. Although most studies have implemented IMT on a daily basis, the issue of training frequency has yet to be studied systematically. In the meantime, it is advisable to be guided by current evidence, which indicates daily training.

Specificity

The nature of a training response depends upon the type of load to which the muscle is subjected. BELMAN *et al*. [44] investigated the characteristics of the load applied to the inspiratory muscles using four methods of overload: flow resistive, pressure threshold, maximal isometric contraction (Mueller manoeuvre) and unloaded hyperpnoea. Whilst there were some similarities between the methods in terms of the stimulus delivered to the muscles (*e.g*. inspiratory pressure load), the methods also showed considerable variation (*e.g*. in the work of breathing). The authors considered that these characteristics might be important in the design of IMT protocols.

This supposition is confirmed by experimental evidence. Generally, respiratory muscles respond to high-frequency, low-load contractions with an endurance-conditioning response, and to low-frequency, high-load contractions with a strength-conditioning response [32, 37, 45]. However, as well as load specificity, there is also an element of flow specificity that must be borne in mind [32, 45], this is because high loads cannot be overcome at high velocities of muscle shortening. Training stimuli with high loads and low velocities (*e.g*. a Mueller manoeuvre) elicit increases in MIP, but do not elicit increases in maximal shortening velocity (peak inspiratory flow rate). Conversely, training with low loads and high velocities of shortening (*e.g*. unloaded hyperpnoea), elicit increases in maximal shortening velocity, but not MIP [32, 45]. Interestingly, training stimuli with intermediate loads and shortening velocities elicit improvements in both qualities [32, 45], which arguably provides the "best of both worlds".

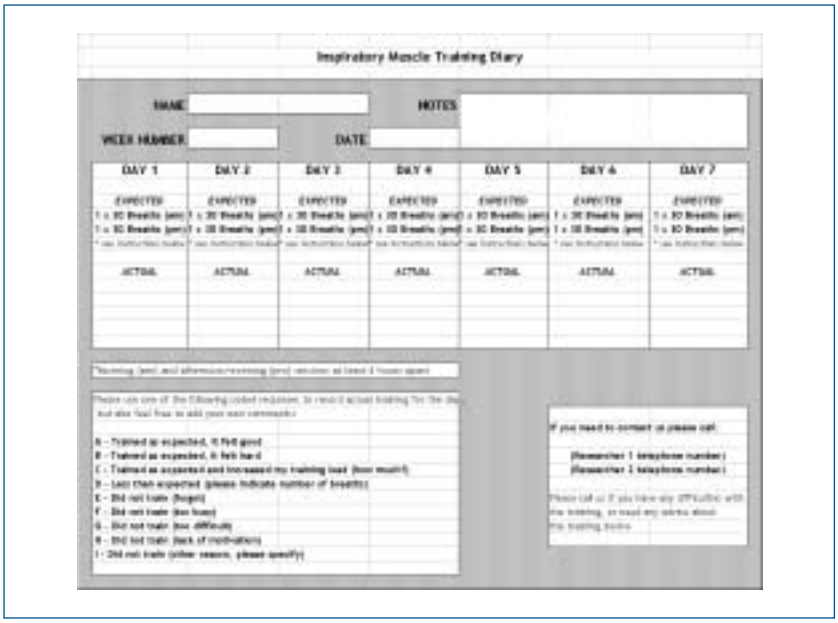

Figure 4
An example of a patient training diary record.

The endurance capacity of the respiratory muscles can also be enhanced by specific IMT regimens. Improvements in endurance have typically been achieved using prolonged voluntary isocapnic hyperpnoea [46], but can also be improved through strength training [21, 22]. There is a common misconception that muscle endurance can only be improved using a specific endurance training stimulus. However, stronger muscles perform a given task at a lower percentage of their maximum capacity than weaker muscles, which has beneficial consequences for fatigue resistance (endurance) [21]. Thus, inspiratory muscle strength training provides a dual conditioning response. Unfortunately, there is no evidence that a specific endurance training stimulus, such as hyperpnoea, improves MIP [47, 48]. Collectively, the data suggest that training regimens with a moderate strength bias have the capacity to improve maximal strength, velocity of shortening and power output [32], as well as endurance. This versatility supports the implementation of training with a moderate strength bias.

To date, only one study has examined whether the muscle length at which IMT occurs has any influence upon the outcome [49]. The data

indicate that improvements in inspiratory muscle strength are specific to the lung volume at which training occurs [49]. Three groups of healthy subjects performed 6 weeks of repeated static maximum inspiratory manoeuvres at one of three lung volumes: residual volume (RV) (functional residual capacity (FRC) or FRC plus one-half of inspiratory capacity). For each group, the greatest improvements in strength occurred at the volume at which the subjects trained, and were significantly greater for those who trained at low (36% for RV and 26% for FRC) than at high volumes. In addition, the range of vital capacity over which strength was increased was also greatest for those who trained at low rather than at high lung volume. These data suggest that IMT should be conducted over the greatest range of lung volume possible, commencing below FRC.

Reversibility

While normal whole-body endurance training promotes a variety of physiological adaptations, periods of inactivity (de-training) are associated with a reversal of many of the adaptations [50, 51]. Unfortunately, the extent and time course of regression following training are not well documented for the inspiratory muscles, but two studies do shed some light on these issues. In healthy young adults, ROMER and MCCONNELL [32] documented regression of IMT-induced changes in inspiratory muscle function (9 weeks of three differing IMT regimens) over an 18-week period of de-training. Decrements were observed at 9 weeks, with no further changes in strength-related measures at 18 weeks post-IMT. In contrast, endurance continued to decline between 9 and 18 weeks of de-training (unpublished observations). Inspiratory muscle function remained significantly above baseline at 18 weeks, with a loss of 32% of the improvement in strength, 65% of the improvement in maximum shortening velocity and 75% of the improvement in inspiratory muscle endurance.

In patients with COPD, WEINER *et al*. [34] observed the de-training response of a group of COPD patients who had completed a 3-month, intensive IMT programme. The de-training group undertook sham training (inspiratory load of 7 cmH_2O) for the next 12 months and were re-assessed at 3-month intervals. Over the 12-month period following IMT, MIP and inspiratory muscle endurance gradually returned to pre-training levels. After 3 months of de-training, both MIP and inspiratory muscle endurance remained elevated compared to baseline (MIP 19%, endurance 22%), but after 12 months, they were not significantly different from baseline. Collectively, these data suggest that inspiratory muscles respond in a similar manner to other muscles when a training stimulus is removed [50, 51], and that most of the losses of function occur within 2–3 months of the cessation of training.

On a more positive note, two de-training studies [32, 34] also demonstrated that IMT-induced improvements in inspiratory muscle function can be sustained with maintenance IMT programmes. Training frequency can be reduced by as much as two-thirds, without loss of function, *i.e.* to 2 days per week in healthy adults [32] and to 3 days per week in patients with COPD [34].

On the basis of the findings presented, it appears that the general training principles of overload, specificity and reversibility that have been established for peripheral skeletal muscles apply equally to the training of respiratory muscles. Further research is required to identify the specific aspects of inspiratory muscle function enhancement that are linked to improvements in dyspnoea and exercise tolerance. Once these are identified, it may be possible to design more targeted IMT programmes. However, in the meantime, some guidance is provided below, which is based upon the principles outlined above, and personal experience with many hundreds of patients and healthy adults.

Suggested indications for IMT

Current consensus statements confine recommendation of IMT to patients with inspiratory muscle weakness [52]. Whilst it appears to be the case that patients with inspiratory muscle weakness (MIP <60 cmH_2O) show greater improvements in dyspnoea and exercise tolerance, it is also the case that even patients with stronger inspiratory muscles experience clinically meaningful improvements [12]. Similarly, highly trained athletes with MIP values in excess of 120 cmH_2O also show improvements in dyspnoea and exercise performance [14, 15]. Since there are no known side-effects of IMT, it is arguable that the intervention could be applied to any patient with dyspnoea, particularly where other treatment options have been exhausted (see Patient selection).

As well as patients with obstructive lung disease, there is a theoretical rationale for strengthening the inspiratory muscles of any patient who experiences inappropriate

breathlessness, abnormal respiratory mechanics and/or inspiratory muscle weakness/fatigue. Accordingly, there may be some value in implementing IMT in patients with conditions such as sarcoidosis [53], obesity [54], type 1 diabetes mellitus [55], idiopathic pulmonary hypertension [56], amyotrophic lateral sclerosis [57], hypothyroidism [58], arthritis [59, 60] or cancer [61, 62]. Furthermore, there are a small number of randomised, controlled trials in which IMT has elicited a positive effect upon dyspnoea in cystic fibrosis [63], heart failure [64, 65], neuromuscular disease [66, 67], spinal cord injury [68], as well as less rigorously designed and executed studies in conditions such as kyphoscoliosis [69] and post-polio [70]. Finally, in a randomised controlled trial, IMT has been shown to offset the myopathic influence of high-dose oral steroids upon inspiratory muscle strength [71].

Implementation and monitoring of IMT

From the preceding sections, it is apparent that there are a number of factors that are associated with successful outcomes in the implementation of IMT. These are summarised in table 1.

When, where and who's involved?

IMT can be performed in in-patient, out-patient and in domiciliary settings, and is typically administered or supervised by suitably trained physiotherapists. The domiciliary setting is convenient for the patient and normally follows a period during which patients' IMT is closely supervised in an in-patient or out-patient clinic. Involvement of family members is also beneficial as they can provide encouragement and sustain patient motivation.

Contraindications

Although, IMT is associated with intra-thoracic decompression, there are almost no side-effects of the training itself. Even patients with heart failure experience no deterioration of their cardiac output during training (personal observation). Therefore, except for patients with unstable asthma and low perception of dyspnoea [72], or a history of spontaneous pneumothorax, there are no contraindications for IMT.

Patient selection

There is a general perception that only patients with evidence of respiratory muscle weakness or ventilatory limitation during physical activity benefit from IMT. However, it has been shown that even when inspiratory muscle weakness is not an inclusion criterion, improvement in dyspnoea and exercise performance follows IMT (see above). Furthermore, it has been shown repeatedly that there is a close correlation between the post-IMT decrease in dyspnoea, and the improvement in inspiratory muscle strength (MIP), regardless of MIP at baseline. This assertion is also supported by the existence of similar relationships in healthy young athletes (see Suggested indications for IMT). However, since there is a dose-response relationship that relates outcome to number of completed IMT sessions, patient motivation is arguably the most important criterion for enrolment. Accordingly, it is recommended that any well-motivated patient with breathlessness should be considered for IMT.

Table 1 Main characteristics of IPTL training protocols that are associated with positive outcomes in patients with COPD

Protocol characteristic	*Recommendation*
Inspiratory load	*30–60% of MIP*
Repetitions	*Up to 30 min, but dependent upon the load, i.e. higher loads cannot be sustained as long as lower loads*
Frequency	*Daily, one or two times per day (morning and evening)*
Duration	*>6 weeks*
Increments	*Weekly, to required percentage of new MIP*

Monitoring

In order to assess the efficacy of IMT in terms of patients' functional ability and quality of life, it is essential to assess the efficacy of the training in terms of its influence upon inspiratory muscle function. As discussed above, if inspiratory muscle function does not change, then neither does functional ability. The most straightforward non-invasive assessment of inspiratory muscle function is MIP. Other methods are available and the reader is referred to [73] and to the ATS/ERS statement on respiratory muscle testing [74] for further information and guidance on correct measurement of MIP. An argument favouring the use of MIP for monitoring is that functional improvements are linked to changes in dyspnoea, and that changes in dyspnoea only result when respiratory training improves inspiratory muscle strength. Regular monitoring of MIP also provides reassurance that patients are adhering to the prescribed training regimen, and provides a basis for resetting training loads. Ideally, monitoring should be undertaken once weekly.

Setting and incrementing the training load

The evidence overwhelmingly supports the use of training loads that exceed 30% of MIP, but also supports the presence of a load-response relationship whereby heavier loads yield greater improvements in MIP. Experience has shown that patients respond best if IMT commences at a load equivalent to 30–40% MIP, and then increments rapidly over the first 7–10 days up to 60–80% of baseline MIP. The first few days are the most problematic, as patients are understandably reluctant to engage in any activity that makes breathing feel harder and that provokes dyspnoea. However, with careful and sensitive coaching, including the allowance of short breaks, they can be encouraged to tolerate the sensation and to increase the training load progressively. Typically, increases in load of 5–10% per week can be achieved. An interesting area for future study is the setting of training loads based upon perceived effort, using tools such as the Borg CR-10 scale.

Coaching good training technique

Training is generally undertaken in the upright posture (normally sitting), but it is also possible to undertake IMT in patients who are supine or semi-recumbent. The operation of the inspiratory muscles differs between upright and supine postures, and this must be considered in the context of training specificity. Due to the influence of specificity upon the training response (upright or supine), it is advisable to encourage patients to train across the full extent of their vital capacity. Furthermore, evidence from limb muscles suggests that the percentage of muscle activation achieved is proportional to the velocity of muscle shortening [75]. In other words, a greater proportion of the muscle is activated when the effort is associated with the intent to shorten the muscle as quickly as possible. Accordingly, patients should be encouraged to breathe against the load as rapidly as possible in order to maximally recruit their inspiratory muscles. Since it is unclear whether functional improvements are associated with preferential increases in diaphragm and/or accessory muscle function, it is advisable to encourage patients to utilise all of their inspiratory muscles during training, and not just the diaphragm. As a result of the length–tension relationship of the inspiratory muscles, patients will experience "clipping" of their end-inspiratory lung volume during a training session, as the effects of training-induced fatigue are manifested first at higher lung volumes. Patients should be warned to expect this and discouraged from training beyond the point where they are able to achieve a "satisfying breath". Finally, patients may require breaks during a training session in order to cough or because they feel too dyspnoeic. It is important that the duration of these is minimised in order to maintain the training stimulus, but this too must be handled sensitively and supported by an explanation of why it is important to minimise the duration of these enforced "rest" periods. The use of supplemental oxygen may be beneficial, but has not been studied to date.

Stand-alone intervention or part of a comprehensive programme?

The evidence supports the use of IMT as both a stand-alone intervention and as part of a multidimensional rehabilitation programme. The ability of virtually all patients to tolerate IMT, even in the presence of co-morbidities that preclude exercise training, renders it promising management tool for severely compromised patients. The potential of IMT to enhance responses to physical exercise training, when used as a precursor to rehabilitation, also remains an intriguing possibility.

Training diaries

The use of specifically prepared training diaries is recommended, as this reminds patients about the requirements of their training schedule, as well as providing feedback to the clinical team about the patient's experience of the training and

Suggested further reading

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ATS Statement: Dyspnea: mechanisms, assessment, and management: a consensus statement. Am J Respir Crit Care Med 1999; 159: 321–340.

ATS Statement: Guidelines on six-minute walk test. Am J Resp Crit Care Med 2002; 166: 111–117.

Troosters T, Gosselink R, Decramer M. Respiratory muscle assessment. In: Lung Function Testing. Gosselink R, Stam H, eds. Eur Respir Mon 2005; 31: 57–71.

responses to it. Figure 4 shows an example of a training diary that can be modified for different applications. It contains information for the patient about when and how to train, as well as a mechanism for them to provide feedback on adherence and perception.

Maintenance training

There is now evidence that the improvements gained during a 3-month, intensive, daily IMT programme can be maintained by training just three times per week. Whether this is optimal in terms of the physiological benefits and adherence is unknown. An alternative approach might be to maintain benefits through short (*e.g.* 1 week) pulses of training every month. Firm recommendations await supporting evidence.

Outcomes

The data suggest that patients undergoing IMT should experience improvements in their MIP, inspiratory muscle endurance, dyspnoea (BDI/TDI and exertional), exercise tolerance and (perhaps) quality of life. There are well-established techniques for assessing all of these parameters and they can be applied equally well to patients undertaking IMT as to those engaging in exercise training.

Educational questions

1. What are the main mechanical and sensory consequences of EFL?
2. Why does exercise exacerbate hyperinflation and dyspnoea?
3. By what mechanism is inspiratory muscle training thought to reduce dyspnoea?
4. Which method of IMT requires simultaneous feedback of inspiratory flow rate: flow-resistive or pressure-threshold loading?
5. What are the three main principles of training?
6. Do the inspiratory muscles adhere to the principles of training in the same manner as other skeletal muscles?
7. Does muscle strength training increase muscle endurance?
8. What is the minimum training load that should be applied to the inspiratory muscles to increase strength?
9. How often should IMT loads be adjusted?
10. How many days per week should patients train during the first 6 weeks of their training, and how much can this be reduced by in the "maintenance" phase?

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Suggested answers

1. EFL creates a requirement to breathe at higher ranges of the total lung capacity, which increases the elastic load presented to the inspiratory muscles by the lungs, causes functional weakening of the inspiratory muscles, induces positive end expiratory pressure, and reduces time available for inspiration. These changes increase inspiratory muscle loading and intensify dyspnoea.
2. Exercise exacerbates hyperinflation because the increased requirement for expiratory flow forces the tidal–flow loop towards total lung capacity in order to minimise flow limitation.
3. The magnitude of the motor drive required to achieve a given muscle force is inversely proportional to muscle strength. Since the magnitude of the motor drive is believed to play a major role in determining the magnitude of effort sensation, it is thought that lower motor drive equates to lower effort. In the case of inspiratory muscles, lower effort is perceived as lower dyspnoea.
4. Flow resistive.
5. Overload, specificity and reversibility.
6. Yes, which means that principles of training that are applied to limb muscles apply equally to inspiratory muscles.
7. During any given muscle task, stronger muscles work at a lower percentage of their maximum capacity, which means they are less likely to fatigue.
8. Thirty per cent of MIP.
9. Weekly adjustments should be made in order to maintain the training load at the desired percentage of the new MIP.
10. During the first 6 weeks of training, patients should train daily (once or twice per day), two to three times per week in the "maintenance" phase.