



## Report

# Evaluation of an inspiratory muscle trainer in healthy humans

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The Powerbreathe<sup>®</sup> is an inspiratory muscle trainer promoted as improving inspiratory muscle strength (and consequently exercise performance) in athletes and patients with respiratory disease. No published evidence supports its efficacy. We performed a prospective randomized controlled study in which 12 normal subjects received either Powerbreathe<sup>®</sup> training or sham training for a 6-week period. The primary outcome measure was diaphragm strength evaluated as twitch transdiaphragmatic pressure (Tw Pdi) but secondary outcome measures were provided by full respiratory muscle assessment and cardiopulmonary exercise testing. An advantage to training was observed when outcome was assessed by maximal static inspiratory mouth pressure (mean advantage 14.5 cm H<sub>2</sub>O, 95% CI 2.2–26.9 cm H<sub>2</sub>O,  $P=0.025$ ). However, no significant difference was observed between the groups in any other parameter. In particular the  $\Delta$ Tw Pdi was not different between groups (mean 'advantage'  $-0.7$  cmH<sub>2</sub>O, 95% CI  $-7.0$ – $+5.5$  cmH<sub>2</sub>O,  $P=0.8$ ). The continued sale and use of the Powerbreathe<sup>®</sup> device is not justified by our data. A sample size calculation showed that 234 subjects would need to be randomized to definitively refute the hypothesis that Powerbreathe<sup>®</sup> improves Tw Pdi and we argue that such a study is required.

**Key words:** inspiratory muscle training; magnetic nerve stimulation.

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## Introduction

It has been claimed that inspiratory muscle training is of benefit in patients with chronic obstructive pulmonary disease (1) and in patients with respiratory muscle weakness (2). The Powerbreathe<sup>®</sup> is a threshold inspiratory muscle training device sold direct to the public which is claimed to improve inspiratory muscle strength and exercise performance in both athletes and patients with respiratory disease after 6 weeks of use. The device originated from the U.K. but can be bought from the internet world-wide. The cost of the device is currently US\$80 and, at least in the U.K., is not reimbursed to patients. For many patients this represents a more than trivial investment yet no data exists to support its efficacy.

The hypothesis that strengthening the inspiratory muscles, by training, results in improved exercise performance (or reduced dyspnoea at a given level of exercise

performance) is not new (3). Nevertheless, until recently it has not been possible to test this hypothesis because the methods commonly used to assess respiratory muscle strength did not distinguish between a true increase in strength and an improvement in the subject's ability to perform the test. However, diaphragm strength can now be measured in an effort-independent fashion using the twitch transdiaphragmatic pressure, Tw Pdi, elicited by bilateral supramaximal anterior magnetic stimulation of the phrenic nerves, BAMPS (4).

We therefore conducted a prospective randomized controlled study of the Powerbreathe<sup>®</sup> in which the primary outcome measure was Tw Pdi. Subjects also underwent full respiratory muscle studies, lung function tests and cardiopulmonary exercise testing.

## Materials and methods

The protocol was approved by our ethics committee and all subjects gave their informed consent to participate. Twelve subjects [nine men and three women; mean (sd) age 32 (4.8) years] were studied; all were free from respiratory or neurological disease. All participants were highly motivated and members of the pulmonary function or respiratory muscle laboratories.

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## STUDY DESIGN

Subjects were randomized to receive either active treatment with Powerbreathe<sup>®</sup> or sham therapy. The Powerbreathe<sup>®</sup> is essentially a valve which only allows inspiratory airflow once a threshold negative pressure is generated at the mouth. Once the valve has opened the resistance to inspiratory airflow is minimal.

The treatment group used the device in accordance with the manufacturers instructions; in brief this entailed performing 30 rapid inspirations from residual volume (RV) to total lung capacity (TLC) twice daily. The opening threshold of the device is adjusted by the trainee so that the trainee can only just complete 30 inspirations. With continued training the trainee finds it easier to reach the threshold and the opening pressure is then made more negative by adjustment of a screw controlling the valve. Thus, the opening pressure after 6 weeks training was more negative than at the start. The sham-training group were given Powerbreathe<sup>®</sup> trainers set at a minimal opening pressure threshold and instructed not to increase this, but their regime was otherwise identical. The compliance of the subjects with training was monitored with the use of a diary card.

## MEASUREMENTS

Measurements were made before and after a 6-week period of either real or sham training. The oesophageal pressure required to open the valve used by each individual for training was measured at the second assessment.

Lung function was assessed according to the guidelines of the British Thoracic Society (5) using spirometry, whole body plethysmography and 12-sec maximal voluntary ventilation (Jaeger, Wurzburg, Germany).

An incremental cardiopulmonary exercise test to exhaustion on a treadmill was performed using the Bruce protocol. During this test we obtained breath by breath measurements of ventilation, tidal volume, respiratory frequency, oxygen uptake and carbon dioxide production (Jaeger).

Inspiratory muscle strength was measured using the traditional method, mouth pressure during a maximal static inspiratory effort ( $PI_{max}$ ), according to the method of Black and Hyatt (6), modified by the use of a flanged mouthpiece. The oesophageal pressure during this maximal effort ( $Poes_{max}$ ) was also recorded. The pressure at the mouth was measured via a thin bore tube connected to a side aperture on the mouthpiece and linked to the recording system (see below).

Inspiratory muscle strength was also assessed using more elaborate laboratory tests. Details may be found elsewhere (7,8), but in essence we measured inspiratory muscle strength as oesophageal (9) and transdiaphragmatic (10) pressure during a maximal voluntary sniff (Sn Poes and Sn Pdi) as well as the unpotentiated supramaximal twitch Tw Pdi during BAMPS (4) using two 45-mm double-circular coils each powered by a Magstim 200 stimulator (The Magstim Co, Dyfed, U.K.) operating at 100% of maximal output. To make these measurements oesophageal pressure

(Poes) was measured using a 10 cm latex balloon attached to a 110 cm PTFE coated catheter and inflated with 0.5 ml air. Gastric pressure (Pga) was measured using a similar balloon catheter inflated with 2 ml air. The two balloon catheters were introduced pernasally and positioned in the oesophagus and stomach in the conventional manner. The catheters were connected to Validyne transducers ( $\pm 200$  cm H<sub>2</sub>O, Validyne Corp., Northridge, CA, U.S.A.). Electrical signals were amplified (Validyne Corp.) and passed via an analogue to digital converter (NB-MIO-16, National Instruments, Austin, Texas, U.S.A.) to an Apple Macintosh computer running LabView<sup>™</sup> software (National Instruments) and sampling at 100 Hz.

## ANALYSIS

Since a sham-training group was used the primary comparison was made between changes before and after training (or sham-training) in the two groups. Unpaired *t*-tests (Statview 4.0, Abacus concepts, Berkeley, CA, U.S.A.) were used for this purpose. For within-group before-after comparisons the Wilcoxon signed rank test was used. In both cases a level of  $P < 0.05$  was taken as significant.

## Results

All participants completed the trial. As judged by diary cards, no subject omitted more than 5% of training sessions. The mean (SD) opening pressure at the conclusion of training in the intervention group was 132 (22) cmH<sub>2</sub>O, or 82.6 (12.3)% of the oesophageal pressure observed during a maximal effort ( $Poes_{max}$ ). For the sham group the mean (SD) opening pressure at the conclusion of training was 37 (13) cmH<sub>2</sub>O, or 31.3 (12.1)% of  $Poes_{max}$ . Thus, the training group had a load which was a mean 95 cm H<sub>2</sub>O [95% confidence interval (CI) 72–117 cmH<sub>2</sub>O,  $P < 0.0001$ ] greater than the sham-training group or, expressed as %  $Poes_{max}$ , which was 51.3% of  $Poes_{max}$  (95% CI 37–67% of  $Poes_{max}$ ,  $P < 0.0001$ ) greater than the sham-training group.

There was no significant increase in lung function parameters (of note vital capacity, total lung capacity or peak inspiratory flow rate) in either the sham-training or the training group, nor were the differences between groups significant.

Strength data are shown in Table 1. At baseline inspiratory muscle strength was not significantly different between subjects allocated to training and those serving as controls except as judged by Sn Poes (mean difference 21 cmH<sub>2</sub>O, 95% CI 2–70 cmH<sub>2</sub>O,  $P = 0.03$ ). This difference is not unexpected since Sn Poes is recognized to be lower in women (9) and three of six controls were women. After training the mean observed increases in  $PI_{max}$ , Sn Poes, Sn Pdi and Tw Pdi were 12.2%, 9.8%, 7.7% and 3.8%, respectively. The corresponding figures for the sham-training group were 1%, 6.5%, 2.5% and 6.8%; these data are tabulated with confidence intervals in Table 2. Comparing the two groups for change in these parameters it is of note that we found a significant increase in  $PI_{max}$  in the training group (mean advantage with training

TABLE 1. Strength data

Subject No.	Group allocation	Gender	Tw Pdi (cmH <sub>2</sub> O)		PI <sub>max</sub> (cmH <sub>2</sub> O)	
			Before	After	Before	After
1	Training	Male	26.9	25.6	132	130
2	Training	Male	24.1	27.7	128	140
3	Training	Male	22.1	28.3	80	88
4	Training	Male	39.4	39.0	149	173
5	Training	Male	38.5	36.6	129	157
6	Training	Male	19.3	19.5	149	172
7	Sham	Female	29.5	25.7	89	95
8	Sham	Male	23.9	21.2	90	99
9	Sham	Female	24.5	22.2	104	98
10	Sham	Male	30.2	42.5	126	117
11	Sham	Male	27.3	30.3	127	132
12	Sham	Female	23.5	27.8	119	121

TABLE 2. Strength data (with 95% confidence intervals) for training and control groups

Test	Training group			Control group			Comparison of change in training vs. control group			
	Mean value pre-training	Mean value post-training	Mean change	Mean value pre-training	Mean value post-training	Mean change	Mean diff	<i>P</i> -value	Lower 95% CI	Upper 95% CI
PI <sub>max</sub> (cmH <sub>2</sub> O)	127.8	143.4	15.6	109.3	110.4	1.1	14.5	0.025	2.2	26.9
Sn Poes (cmH <sub>2</sub> O)	134.7	147.9	13.2	98.8	105.2	6.4	6.8	0.300	-7.1	20.8
Sn Pdi (cmH <sub>2</sub> O)	141.4	152.3	10.9	120.2	123.3	3.1	7.8	0.365	-10.7	26.4
Tw Pdi (cmH <sub>2</sub> O)	28.4	29.5	1.1	26.5	28.3	1.8	-0.7	0.800	-7	5.5

14.5 cmH<sub>2</sub>O, 95% CI 2.2–26.9 cmH<sub>2</sub>O, *P*=0.025) whereas other tests, and in particular the  $\Delta$ Tw Pdi, was not different between groups (mean 'advantage' with training -0.7 cmH<sub>2</sub>O, 95% CI -7.0–+5.5 cmH<sub>2</sub>O, *P*=0.8).

Performance data are shown with confidence intervals in Table 3. No significant improvement was observed in either exercise duration, intensity or maximal voluntary ventilation.

## Discussion

Our data show that PI<sub>max</sub> increased in the training group, but this was not confirmed by detailed tests to represent a genuine increase in respiratory muscle strength and there was no improvement in exercise performance. Since the PI<sub>max</sub> manoeuvre has many similarities with the training manoeuvre we suggest that this result represents a learning

effect; thus we reject the concept that use, by healthy subjects, of the Powerbreathe<sup>®</sup> device is beneficial. Although this study is limited by its small sample size (see below), we know of no data in the literature that do support its use. Chest physicians should not advise purchase of the Powerbreathe<sup>®</sup> unless further data become available.

## CRITIQUE OF THE METHOD

In the current study the subjects were well motivated and demonstrated their adherence to the programme with diary cards. At the completion of the study there was a clear difference in the training load of the two groups. Therefore three possible reasons exist why our study failed to show a benefit with Powerbreathe<sup>®</sup>. First, there could be a genuine benefit which our study failed to detect because the sample size was too small. Second, although inspiratory muscle

TABLE 3. Exercise data (with 95% confidence intervals) for training and control groups

Test	Training group			Control group			Comparison of change in training vs. control group			
	Mean value pretraining	Mean value post-training	Mean change	Mean value pretraining	Mean value post-training	Mean change	Mean diff	<i>P</i> -value	Lower 95% CI	Upper 95% CI
MVV ( $l \text{ min}^{-1}$ )	174	186	12	151	158	6	5	0.659	-21	32
Exercise time (sec)	848	887	39	750	754	4	35	0.229	-26	95
Max min ventilation ( $l \text{ min}^{-1}$ )	122	113	-9	105	101	-4	-5	0.711	-27	19
Max HR ( $c \text{ min}^{-1}$ )	182	184	2	185	185	0	2	0.534	-12	22
$V_{O_{2\max}}$ ( $ml \text{ min}^{-1} \text{ kg}^{-1}$ )	44	40	-5	39	39	0	-4	0.140	-10	2
$V_{CO_2}$ ( $l \text{ min}^{-1}$ )	4.9	4.1	-0.8	4.4	4.1	-0.3	-0.5	0.216	-1.4	0.4
RQ (end-exercise)	1.30	1.28	-0.02	1.30	1.28	-0.02	0.00	0.411	0.28	0.13

training may be beneficial the Powerbreathe<sup>®</sup> training regime may not load the muscles sufficiently to train the inspiratory muscles or, thirdly, inspiratory muscle training may not be of value in healthy humans.

Clearly, if our sample size were too small we could have missed a treatment effect. The use of parametric statistics and confidence intervals allows an estimation of the magnitude of difference we could have missed. Tables 2 and 3 show that we could have failed to detect an increase in inspiratory muscle strength of approximately 20% and an increase in exercise duration or peak ventilation of approximately 10%. Although these might seem substantial margins, it is important to recall that enthusiasts consider that inspiratory muscle training can generate bigger improvements; for example in the now classic paper by Leith and Bradley strength training was reported to increase inspiratory muscle strength, judged from a maximal static effort, by 55% (3).

The sample size required to demonstrate a benefit is of interest. All outcome measures used in previous studies depend on patient effort or aptitude at performing the test. This is particularly true of the  $PI_{max}$  which, because it has marked similarities to the training manoeuvre itself, may increase simply because the subjects are better at performing the manoeuvre. It is therefore of interest that the  $\Delta PI_{max}$  was significantly greater in the present study in the training group. We suggest that this represents a 'learning' effect which is unrelated to the contractile properties of the inspiratory muscles. Nevertheless it may offer an explanation for apparently positive results produced by inspiratory muscle training previously.

Because we were concerned that  $PI_{max}$  was not an adequate test to evaluate the effects of Powerbreathe we determined prior to the study that our primary aim would be to evaluate inspiratory muscle training using a non-volitional technique. We performed a sample size calculation using data from the control subjects with the premise that a 10% increase in Tw Pdi would be clinically worthwhile, and sought 80% power and a significance level of  $P < 0.05$ . This calculation showed that 117 subjects would be required in each group. Since the resources are not currently available to our laboratory to randomize and follow 234 subjects we opted to submit the present data for publication, since we believed them to be hypothesis-generating.

The remaining possibilities (that the Powerbreathe<sup>®</sup> regime is insufficiently rigorous to induce training or that inspiratory muscle training itself can not occur in normal subjects) are not distinguished by the current data. However the pressures generated in the training group were close to maximal and thus if the Powerbreathe<sup>®</sup> is insufficiently rigorous this must be because the quantity of training is insufficient rather than the intensity. We know of no data where phrenic nerve stimulation data has been used to test either of these hypotheses specifically.

An additional limitation of the current study is that the Powerbreathe<sup>®</sup> was assessed in healthy volunteers. To some extent this limits the extrapolation of our data, but it should be recalled that the Powerbreathe<sup>®</sup> is considered by its manufacturer to be equally efficacious in healthy subjects

and patients with respiratory disease and to have the same mode of action.

## SIGNIFICANCE OF THE FINDINGS

The use of Powerbreathe<sup>®</sup> has been justified almost entirely by publications in abstract form only. Indeed a Medline search performed using both the inventors name and the term 'Powerbreathe<sup>®</sup>' failed to yield a single study evaluating its efficacy. Despite this the device has been favourably portrayed in the print and television media, and indeed was exhibited in the Millennium Dome in London as an achievement of British science. The device has also been awarded a SMART Foresight award from the U.K. government in recognition of 'exceptional vision in developing new technologies'. Our data raise the serious possibility that the device is of no value in increasing inspiratory muscle strength or exercise performance and we suggest that its current popularity is not justified by scientific data. It is therefore a matter of concern that the device is sold direct to the public without any form of regulation.

The Powerbreathe<sup>®</sup> is simply one example of an inspiratory muscle training device; and strictly speaking, our conclusions cannot be extended to other training regimens. Nevertheless it is of interest that a meta-analysis of training regimes in chronic obstructive pulmonary disease (COPD) (11) concluded that the data provided little support for this treatment while, more recently, a controlled randomized study of 30 patients with COPD failed to find an increase in maximal inspiratory pressure in patients with COPD (1). Conversely,  $PI_{max}$  can rise by up to 20% in patients treated only with placebo (12), presumably as a result of a learning effect.

Magnetic stimulation of the phrenic nerves is a relatively new technique which is acceptable both to normal subjects and patients with respiratory disease (13,14). Magnetic stimulation has been shown to be sufficiently sensitive to track the changes induced by low frequency fatigue in man (13), and is especially advantageous for 'within-occasion' data (15). The principle advantage of using the Tw Pdi elicited by magnetic stimulation is that the data are entirely independent of the patients aptitude or motivation. This is particularly relevant where the test manoeuvre closely resembles the training manoeuvre. To our knowledge, no previous study has evaluated an inspiratory muscle training protocol using data obtained by phrenic nerve stimulation as an endpoint. The disadvantage of using Tw Pdi is that, because the twitch is usually about 20% of the value of the voluntary manoeuvre (16), the between-occasion variability is, as a proportion bigger than would be obtained with the sniff manoeuvre; this variation is present irrespective of stimulation modality (17). Thus to exclude change large sample sizes may be required and we argue that such a study is now warranted.

In conclusion our data provide no evidence base for the current use of the Powerbreathe<sup>®</sup> device. We suggest that a randomized controlled study of sufficient power be performed to evaluate its position more precisely.

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