

Voluntary decrease in breathing frequency in exercising asthmatic subjects

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Voluntary decrease in breathing frequency in exercising asthmatic subjects. F. Ceugniet, F. Cauchefer, J. Gallego. ©ERS Journals Ltd 1996.

ABSTRACT: Exercise rehabilitation programmes are increasingly recommended in young asthmatics, but it is unclear whether or not training should incorporate instructions on breathing pattern. In this study, we examined the effects of voluntarily decreasing breathing frequency on their ventilatory equivalents for oxygen and carbon dioxide (minute ventilation (V'_E)/oxygen consumption ($V'O_2$) and V'_E/CO_2 production ($V'CO_2$), respectively), noninvasively determined physiological dead space/tidal volume (V_D/V_T) and dyspnoea.

Fifteen young asthmatic subjects were assigned to two groups: low frequency breathing (LFB) and controls. They first underwent an exercise test at a cardiac frequency of 150 beats·min⁻¹. They were trained at this level for nine sessions. LFB subjects were instructed to decrease respiratory frequency by 40% during exercise. Control subjects received no instructions. A second test was then performed in the same conditions.

LFB subjects decreased $V'_E/V'O_2$, $V'_E/V'CO_2$ and V_D/V_T by 22, 19 and 12%, respectively. Arterial oxygen saturation (S_{a,O_2}) fell to 89±4% and end-tidal carbon dioxide tension (P_{ET,CO_2}) rose to 6.5±0.7 kPa (49±5 mmHg). In controls, these variables were identical in the two tests. Dyspnoea was lower in the second test in all subjects.

In conclusion, breathing pattern may be profoundly altered during exercise without concomitant increase in dyspnoea. However, directing breathing patterns for exercise rehabilitation requires an individual assessment of the desired pattern in order to prevent hypercapnia or hypoxia.

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Poor physical fitness in asthmatic children is a consequence of their low activity level, and it can be improved to a normal level by physical training [1, 2]. Training may lower the cardiorespiratory response for a given level of exercise, reduce exercise-induced asthma, increase thresholds for provocation of symptoms, lower medication demands and diminish the frequency of attacks [3]. For these reasons, exercise rehabilitation programmes are increasingly recommended in young asthmatics [1]. However, the way training should be designed to optimize its effects is a matter of research [4, 5]. In particular, it is unclear whether or not training should incorporate specific instructions on breathing pattern, especially respiratory frequency (f_R) and tidal volume (V_T).

Several arguments support the idea that a more appropriate breathing pattern may improve the ventilatory function in exercising asthmatic subjects. Firstly, exercise-induced asthma (EIA), whether its cause is respiratory heat loss or increased osmolarity due to respiratory water loss, seems to be related to minute ventilation (V'_E) and breathing pattern [4, 6, 7], although this relationship has been questioned [8, 9]. A slow and deep pattern of breathing makes the respiratory airflow more laminar, which reduces the heat and water exchange

within the tracheobronchial tree and, therefore, EIA [10]. This pattern also decreases the proportion of dead space ventilation to total ventilation, which allows a lower ventilation [11]. All these arguments support the idea that asthmatic subjects should benefit from adopting a slow and deep pattern of breathing during exercise.

An opposite view is that asthmatics should not be encouraged to change their breathing pattern because they spontaneously adopt the pattern best-suited to their pathophysiological status. The fact that exercise hyperpnoea is achieved by adopting higher V_T , and lower f_R than normal subjects may reflect these adaptive changes [12–14]. In addition to the fact that voluntary changes in f_R may cause hypercapnia, it may also increase dyspnoea at a given level of carbon dioxide tension (P_{CO_2}). In normal subjects, dyspnoea intensifies when the level or pattern of breathing is voluntarily changed [15]. Moreover, a recent study [16] has shown that the more a subject voluntarily lowers ventilation below the spontaneous level, the more he/she feels dyspnoeic. These and other arguments [17] suggest that ventilation and breathing pattern optimally minimize dyspnoea during exercise. If so, voluntary changes of this pattern would be detrimental to ventilatory exchange and dyspnoea.

In the light of these contrasting views, we have conducted an experiment on the effects of the voluntary decrease in f_R on pulmonary gas exchange and dyspnoea. Specifically, we tested the hypothesis that a low f_R during a submaximal exercise would decrease the ventilatory equivalent for O_2 ($V'E/O_2$ consumption ($V'O_2$)), and CO_2 ($V'E/CO_2$ production ($V'CO_2$)), physiological dead space/tidal volume ratio (V_D/V_T) and dyspnoea. One possible bias is that the difficulty of these voluntary changes may hamper the benefits of low f_R in terms of respiratory comfort or dyspnoea. We therefore postulated that the new pattern must be learned before its effects are considered; this requires sufficient practice [18, 19]. To do this, young asthmatic subjects were trained to breathe at a prescribed low frequency for nine sessions. These subjects were compared with asthmatic control subjects who performed the same amount of exercise training without specific ventilatory instruction.

Methods

Subjects

Sixteen subjects with chronic severe asthma participated in the study. All were in-patients of an educational rehabilitation centre located at 1,800 m above sea level. The subjects were aged 12–19 yrs. Anthropometric and spirometric data are presented in table 1. The current medication regimen comprised beta₂-agonists and inhaled corticoids, which were maintained during the study and kept baseline respiratory data within normal limits. The subjects were familiarized with the exercise testing and were physically active.

Apparatus

The graded exercise tests were performed on a computerized electronically-braked cycle ergometer (Medgraphics Exercise Testing System CPX/D). Prior to each test, the gas analyser system was calibrated according to standard procedures with known concentrations of CO_2 and O_2 : 21% O_2 , 0% CO_2 , balance N_2 ; and 12.4% O_2 , 4.6% CO_2 , balance N_2 . The turbine flow meter was

calibrated with room air using a syringe. Mixed expired concentrations of O_2 and CO_2 were measured continuously and used to calculate $V'O_2$ and $V'CO_2$. V_D/V_T was estimated noninvasively using end-tidal carbon dioxide tension (P_{ET,CO_2}) as an approximation of arterial carbon dioxide tension (P_{a,CO_2}). Arterial oxygen saturation (S_{a,O_2}) was estimated throughout exercise by pulse oximetry (Nihon-Kohden). Before the experiment, baseline spirometry was measured using a computerized spirometer (Dyn'Air). For each pulmonary function test, three successive volume manoeuvres were performed, and the value corresponding to the highest sum of forced expiratory volume in second (FEV₁) and forced vital capacity (FVC) was collected.

Cycle ergometry protocol

An individualized ramp protocol was used for exercise testing. The prescribed pedalling frequency was 60 rpm throughout. To achieve this, the subjects received a visual and vocal reminder to pedal at the prescribed rate. The power increment per minute was automatically calculated to reach the maximum predicted power in 15 min. This increment ceased when cardiac frequency (f_C) reached 150 beats·min⁻¹. The corresponding workload (W_{ref}) which was maintained for a 4 min period, ranged 51–158 W. This large disparity in workload was due to the relatively large range of the subjects' ages. This 4 min plateau was presumably below the lactate threshold, as suggested by the steady state reached by $V'O_2$. We considered that a steady state was attained because $V'O_2$ values remained constant throughout the 4 min of constant load [20]. These values were obtained by calculating averages from successive periods of 30 s.

Experimental design and procedure

All the subjects participated in one test (Test 1), nine training sessions scheduled over 3 weeks, and then a second test (Test 2). Temperature and barometric pressure were not significantly different between the two tests (21.6±2.3 vs 22.9±1.2°C, and 647±14 vs 636±7 mmHg). All the subjects performed Test 1 as described above. The subjects were then randomly assigned to two groups: low frequency breathing (LFB), n=9, and controls, n=7 (the first three subjects enrolled were deliberately assigned to the LFB group). Each subject performed nine 45 min training sessions, during which no data were collected. During these sessions, the subjects in the LFB group learned to lower f_R while performing pedalling exercise at W_{ref} . The subjects progressively decreased f_R by synchronizing breathing with pedalling frequency with an inspiratory to expiratory time (t_I/t_E) ratio of 1:2, thus reducing f_R to 20 breaths·min⁻¹. This caused f_R to decrease to about 40%, as a mean. Control subjects were not given any instruction on breathing. To standardize for mental activity and its possible influence on breathing and dyspnoea, control subjects were instructed to mentally count the pedal revolutions throughout the tests, to raise one finger and restart counting each time they reached 60. All the remaining aspects of the training were identical in the two groups. Finally, Test

Table 1. – Anthropometric data and spirometric values

	Group	
	Control	LFB
Subjects	7	9
Sex M/F	4/3	5/4
Age yrs	14±2	14±3
Height cm	158±17	161±8
Weight kg	54±16	58±15
FVC L	3.15±0.99	3.87±0.86
% pred	99±6	117±15
FEV ₁ L	2.54±1.02	2.90±0.83
% pred	91±10	106±26
FEV ₁ /FVC %	79±9	77±13
% pred	92±11	89±14

Values are presented as mean±SEM. LFB: low frequency breathing; M: male; F: female; FVC: forced vital capacity; FEV₁: forced expiratory volume in one second; % pred: percentage of predicted value.

2 was the same as Test 1 (the increment was also interrupted at a f_c of 150 beats·min⁻¹), except for the fact that the LFB group were asked to breathe as they have been trained to do, whereas control subjects counted mentally, and did not receive any instruction on breathing.

After each test, the subjects rated the intensity of breathlessness by writing a mark on a 10 cm horizontal visual analogue scale (VAS), between the words "Not at all breathless" on the zero-left extremity and "Extremely breathless" at the 10-right extremity of the scale. This latter point was defined as the highest level of dyspnoea the subject ever experienced whilst exercising. The dyspnoea score was expressed in millimetres from the zero end.

Data analysis

The dependent variables were f_R (breaths·min⁻¹), V_T (L corrected for body temperature atmospheric pressure, and water saturation (BTPS)), duty cycle *i.e.* fraction of inspiration of total breathing cycle (t_I/t_{tot}), V'_E (L·min⁻¹ BTPS), P_{ET,CO_2} (mmHg), end-tidal oxygen tension (P_{ET,O_2}) (mmHg), V_D/V_T (%), $V'O_2$ (mL·min⁻¹ corrected for standard temperature and pressure dry (STPD)), $V'CO_2$ (mL·min⁻¹ STPD), S_{a,O_2} (%), f_c (beats·min⁻¹), and dyspnoea scores (VAS, mm). These variables were averaged over the last 2 min of each constant load exercise in Test 1 and Test 2. The Jones equation [21, 22] for estimating arterial carbon dioxide tension (P_{a,CO_2}) from P_{ET,CO_2} was not used here because it has not been validated in asthmatic subjects.

LFB and controls were compared using analyses of variance (ANOVA) with group (two levels: LFB and controls) as a between-subject factor, and test (two levels) as a within-subject factor. A separate analysis was conducted for each dependent variable. Partial comparisons were made within groups when appropriate. All the interactions mentioned below are group by test interactions, and the corresponding p-values are denoted p_{int} . The software used for these analyses was Superanova (Abacus Concepts).

Results

Breathing pattern (fig. 1)

Test 1 and Test 2 were very similar in controls, whereas, in Test 2, the LFB subjects adopted much lower f_R ($p=0.0002$; $p_{int}=0.003$) and duty cycles ($p=0.015$; $p_{int}=0.011$). LFB subjects significantly increased V_T in Test 2 ($p=0.0009$; $p_{int}=0.001$). Partial comparison in the LFB group showed that V'_E was lower in Test 2 despite the increase in V_T , but this effect failed to reach significance ($p=0.079$; $p_{int}=0.105$).

In Test 1, V_T was significantly correlated with FVC ($r=0.901$; $p=0.0001$), and was $42\pm 7\%$ of FVC for the sample of subjects as a whole ($n=16$). In Test 2, V_T/FVC rose to $62\pm 10\%$ in LFB subjects, whereas it decreased slightly in controls ($37\pm 7\%$; $p_{int}=0.008$). In Test 2, V_T was still significantly related to FVC in the two groups (controls: $r=0.74$; $p=0.058$, and LFB: $r=0.83$; $p=0.005$).

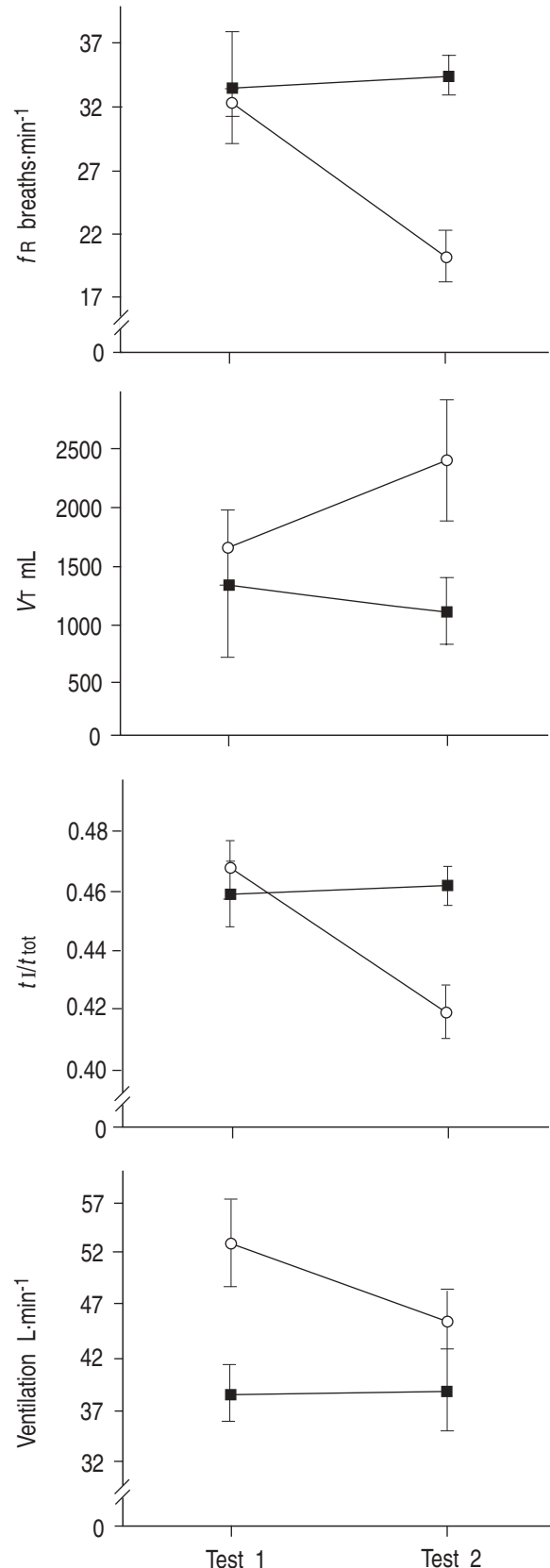


Fig. 1. – Effect of low frequency breathing (LFB) on breathing variables. Values are mean \pm SEM. LFB group (○—○; $n=9$), Control group (■—■; $n=7$). Test 1 and Test 2 were the same for the two groups. LFB was practised by the LFB subjects in Test 2 only. f_R : respiratory frequency; V_T : tidal volume; t_I/t_{tot} : fraction of inspiration to total breathing cycle duration (duty cycle).

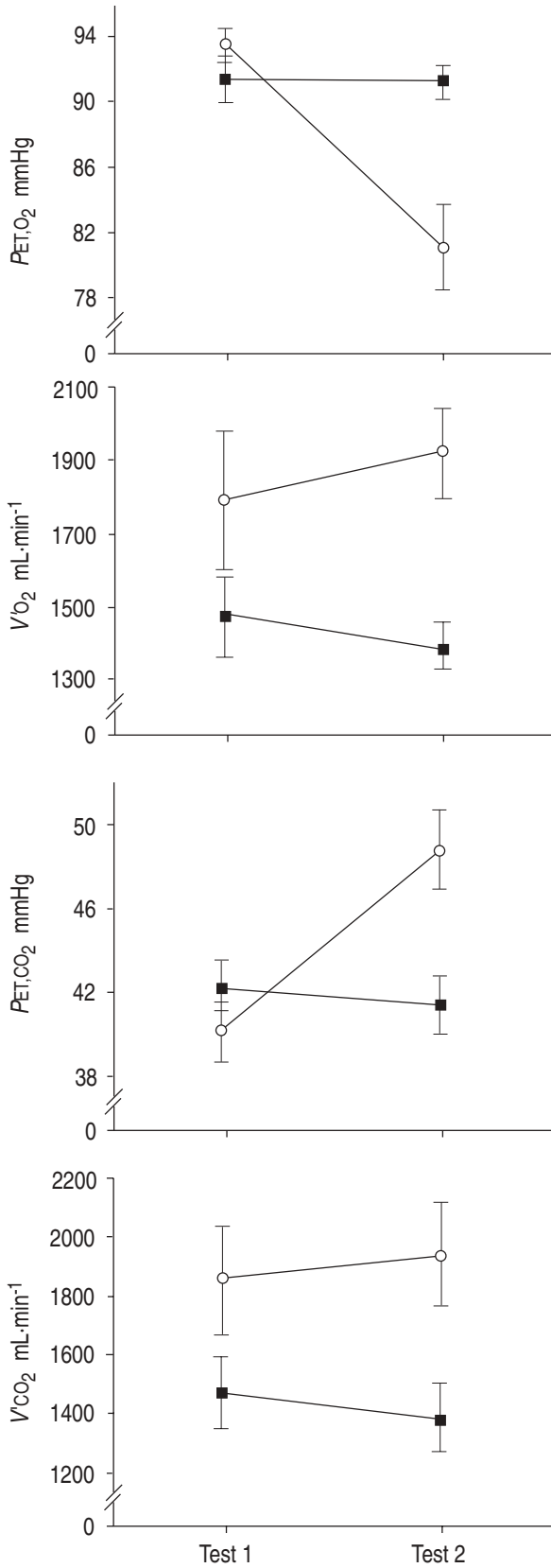


Fig. 2. — Effect of low frequency breathing (LFB) on ventilatory gas exchange. Values are mean±SEM. Test 1 and Test 2 were the same for the two groups. LFB was practised by the LFB subjects in Test 2 only. LFB group (○—○; n=9); Control group (■—■; n=7). P_{ET,O_2} : end-tidal oxygen tension; P_{ET,CO_2} : end-tidal carbon dioxide tension; $V'O_2$: oxygen uptake; $V'CO_2$: carbon dioxide production.

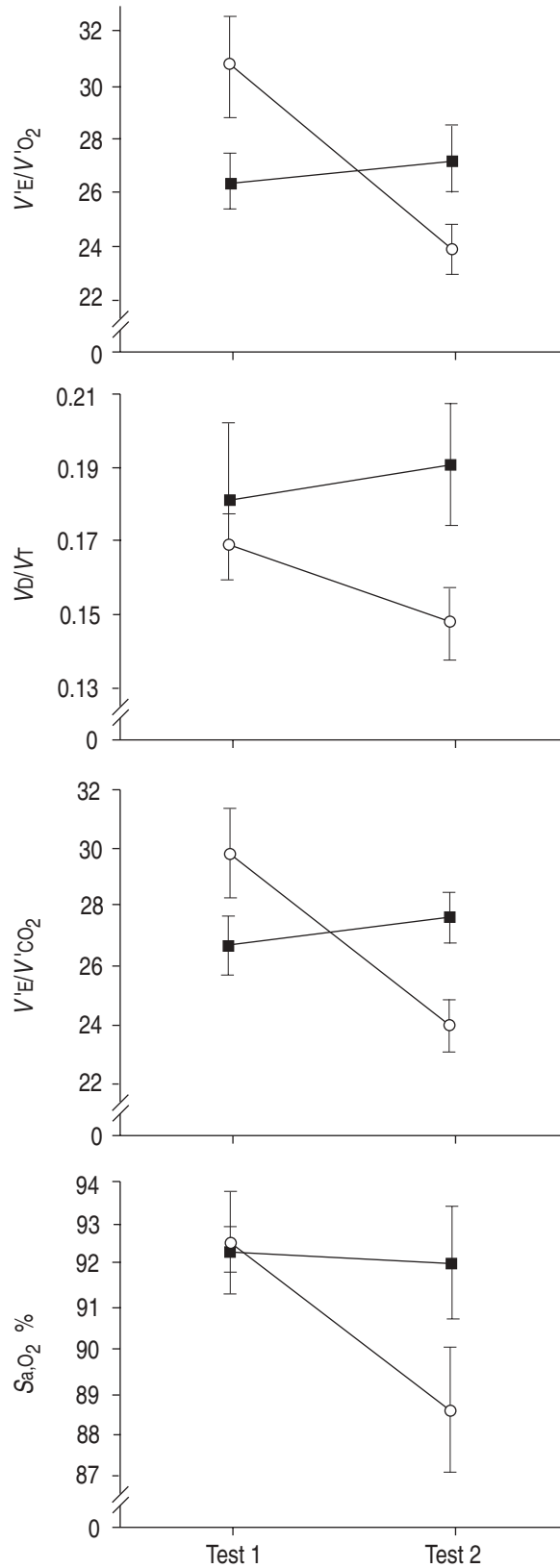


Fig. 3. — Effect of low frequency breathing (LFB) on ventilatory equivalents for O_2 ($V'E/V'O_2$), and CO_2 ($V'E/V'CO_2$), on physiological dead space/tidal volume ratio (V_D/V_T) and arterial oxygen saturation (Sa,O_2). values are mean±SEM. LFB group (○—○; n=9); Control group (■—■; n=7). Test 1 and Test 2 were the same for the two groups. LFB was practised by the LFB subjects in Test 2 only.

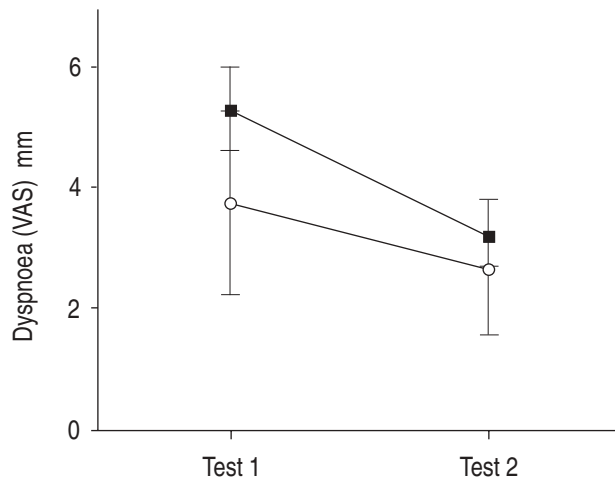


Fig. 4. — Effect of low frequency breathing (LFB) on dyspnoea. VAS: visual analogue scale. Values are mean \pm SEM. LFB group (○—○; n=9); Control group (■—■; n=7). Test 1 and Test 2 were the same for the two groups. LFB was practised by the LFB subjects in Test 2 only.

Pulmonary gas exchanges (figs. 2 and 3)

In controls, P_{ET,O_2} and P_{ET,CO_2} were very similar in the two tests. In LFB subjects, P_{ET,O_2} was lower in Test 2 than in Test 1 ($p=0.0026$; $p_{int}=0.004$), and P_{ET,CO_2} was higher in Test 2 than in Test 1 ($p=0.002$; $p_{int}=0.002$). $V'O_2$ and $V'CO_2$ were not significantly different in the two tests in either group. These values were higher in LFB subjects ($p=0.040$ and $p=0.047$, respectively). However, the $V'O_2$ to body weight ratio was not significantly different between the two groups ($p=0.27$).

In the LFB group, $V'E/V'O_2$, $V'E/V'CO_2$, and V_D/V_T were significantly lower in Test 2 ($p=0.008$, $p=0.001$ and $p=0.0001$; $p_{int}=0.012$, $p_{int}=0.001$ and $p_{int}=0.0003$, respectively). Compared with Test 1, mean changes in $V'E/V'O_2$, $V'E/V'CO_2$, and V_D/V_T were -22, -19.5 and -12%, respectively (*vs* 3, 4 and 14% in controls). In LFB subjects, Sa,O_2 was lower during LFB ($p=0.018$; $p_{int}=0.055$). Changes in f_c , and $V'O_2/f_c$ were not significant.

Dyspnoea (fig. 4)

Dyspnoea scores were missing for one control subject and three LFB subjects. All dyspnoea scores were lower in Test 2 than in Test 1 ($p=0.006$). No difference appeared between the two groups (main effects for group and interaction NS). Partial comparison within the LFB group confirmed that dyspnoea scores were lower in Test 2, although this failed to reach the 0.05 level ($p=0.057$).

Discussion

This experiment showed that young asthmatic subjects could exercise at high intensity with profound modification of their spontaneous breathing patterns and gas exchange, without concomitant change in dyspnoea. Low frequency breathing caused lower $V'E$, $V'E/V'O_2$, $V'E/V'CO_2$ and noninvasive V_D/V_T . The altered breathing pattern led both to a decrease in Sa,O_2 and an increase in P_{ET,CO_2} . It is likely, therefore, that P_a,CO_2 also increased under these conditions, thus indicating the potential drawbacks

of directing breathing patterns. The subjects did not exhibit exercise-induced asthma.

It was found that LFB caused about a 20% decrease in $V'E/V'CO_2$. This may have resulted in part from hypoventilation (as suggested by the elevated P_{ET,CO_2}) and from the decrease in V_D/V_T . This estimate of V_D/V_T differs from those calculated using P_a,CO_2 , particularly in patients with uneven distribution of ventilation and perfusion [23, 24]. The correlation between estimated V_D/V_T and the actual value derived from P_a,CO_2 , ($r=0.69$), for a sample of 68 patients, according to LEWIS *et al.* [23], is too low to decide whether V_D/V_T is above or below a given threshold of normality. However, this limitation does not necessarily preclude the use of noninvasive V_D/V_T to compare groups drawn from the same clinical population. For this purpose, the relevant issue is whether the estimation error of V_D/V_T is different among the groups. Specifically, if changing breathing frequency influences this error, no inference should be made on the physiological effects of frequency changes. In addition to these possible drawbacks, estimation of V_D/V_T should be considered with caution because small errors in estimated P_a,CO_2 may be amplified to larger relative errors in estimated V_D/V_T [23]. With these restrictions in mind, the drop in V_D/V_T caused by LFB may confirm the strong influence of breathing pattern on this ratio [25, 11].

The decrease in $V'E$, $V'E/V'O_2$, and $V'E/V'CO_2$ caused by LFB is in agreement with previous studies in healthy subjects, including trained athletes [26, 27]. Basically, the present data show that these previous findings may be extended to young asthmatic subjects. In the study by SHARP *et al.* [27], neither $V'O_2$ nor $V'CO_2$ were significantly affected by the voluntary decrease in f_R ; P_{O_2} was significantly lower and P_{CO_2} was higher during LFB than during spontaneous breathing. We observed similar effects in the present study. An interesting point reported by SHARP *et al.* [27] was that blood lactate concentrations were not influenced by LFB. This suggests that the O_2 delivery to exercising muscles was normal. A similar assumption may be true for the present study.

We observed that voluntary changes in breathing pattern did not increase dyspnoea. CHONAN *et al.* [15] reported that the level and pattern of breathing influenced dyspnoea in normal subjects at constant level of chemical drive. Because dyspnoea was minimal for breathing variables corresponding to spontaneous values, these authors suggested that $V'E$ and f_R may be optimally adjusted to minimize dyspnoea. In the present study, it was observed that dyspnoea was lower in Test 2, with or without LFB. Several arguments may explain this apparent discrepancy. Firstly, in the experiment by CHONAN *et al.* [15], the rating by the subject of the "intensity of the sensation of difficulty in breathing" may have been influenced by the perceived difficulty to adjust breathing to the prescribed pattern. The greater the difference between the target and the spontaneous patterns, the more the difficulty. Accordingly, the finding that the spontaneous pattern corresponded to the minimal dyspnoea rating may, in part, be due to the fact that this pattern was the easiest to produce voluntarily. The amount of previous training to control breathing is crucial in this respect because it makes it simpler to perform different target patterns. In the present experiment, the subjects practised the LFB for nine sessions, and they therefore

grew familiar with the target frequency. It is likely that their dyspnoea scores were not much influenced by the effort to control ventilation.

A second argument is that the results reported by CHONAN *et al.* [15] may not extend to asthmatic patients. Even assuming that breathing pattern is optimally adjusted to minimize dyspnoea in normal subjects, this may not be true in asthmatic subjects. In fact, breathing pattern in asthmatic subjects may be optimized according to other criteria than in normal subjects (for example, minimizing heat and water loss). If so, voluntary changes in breathing pattern for a given level of ventilation in asthmatic subjects would not necessarily cause an increase in dyspnoea. Finally, it should be noted that observed differences in dyspnoea between the two tests are not only due to changes in breathing pattern (the drop in f_R and t_I/t_{tot} and the increase in V_T), but also to the shift from automatic to voluntary breathing. The fact that dyspnoea at a given level of ventilation is less during voluntary than automatic breathing [28, 29] may have contributed to lower dyspnoea scores in Test 2 and have masked the opposite effect caused by the subjects departing from their spontaneous pattern.

The fact that dyspnoea scores were lower in the second test in control subjects contrasted with the remarkable reproducibility of physiological variables. The duration of practice during each session, in particular above 60–80% of maximal f_C (the critical training f_C) and the number of sessions were certainly too small to induce any training effect in either group [30]. Given the lack of any observable change in breathing pattern, pulmonary gas exchange, or power output in controls, the decrease in dyspnoea scores may reveal the effect of prior experience on dyspnoea sensation, an effect which has presumably influenced dyspnoea scores in LFB subjects as well. Prior experience may change the subjective sensation of dyspnoea by changing the reference levels used by the subjects in quantifying this sensation, as suggested by several recent experiments [31].

In fact, including LFB in rehabilitation programmes may facilitate a process which normally operates in some of these patients, through learning or conditioning processes which do not necessarily require the subject's awareness [32]. It has been reported that children with moderate asthma compensate their resistive load by using higher V_T and lower f_R [14]. However, training patients to lower frequency breathing should be considered with extreme caution, because the subjects practising LFB displayed hypercapnia and hypoxia. The abnormal blood gas levels were apparently due to insufficient compensatory increase in V_T . During spontaneous breathing, the subjects normally increase V'_E during exercise by increasing both V_T and f_R . At high work rates, the upper limit of V_T is about 50–60% of FVC, and further increases in V'_E are due to increases in f_R alone. During LFB, mean V_T rose to 62% of FVC. Further increase of V_T would have been very difficult and f_R and the t_I/t_{tot} were experimentally locked to prescribed values. The low prescribed values of f_R and t_I/t_{tot} , combined with the difficulty of increasing V_T above a certain fraction of FVC prompted the LFB subjects to hypoventilate. Although this effect failed to reach significance, it must have contributed to the observed increase in end-tidal P_{ET,CO_2} .

We speculate that the detrimental effect of hypoventilation and hypercapnia on dyspnoea [16] was masked by other factors described above. This suggests that the target breathing pattern should be determined individually, in order to allow an improvement in ventilatory efficiency without affecting normal blood gas values. However, it is not established that the individual determination of this pattern is easily achievable in clinical settings. At an experimental level, an alternative way to disentangle the effects of breathing pattern and changes in blood gas values on dyspnoea would be to replicate the present experiment with another freely-breathing group in which P_{CO_2} and S_{a,O_2} have been brought to the levels achieved in the LFB group, through appropriate manipulation of the inspired gas.

As noted above, one of the expected benefits of LFB is to reduce EIA. This is based on the hypothesis that LFB would reduce the heat and water exchanges within the tracheobronchial tree, which are supposed to trigger or to exacerbate EIA. Most of the patients who participated in this study displayed EIA after outdoor exercise, but since the present test was performed indoors at a warm temperature, EIA did not occur. Replicating the present experiment in cold and dry air may shed light on the possible beneficial effects of LFB. A complementary approach would consist of comparing asthmatic patients having EIA with asthmatic patients without EIA. It might be anticipated that EIA patients will display abnormally high V'_E or f_R in response to exercise, suggesting that LFB may be beneficial to these patients.

More generally, it has been a long-held belief that voluntary breathing can be a useful complementary therapy for asthma, but it has rarely relied upon appropriate controlled experiments. Amongst several exceptions, a randomized, double-blind, placebo-controlled experiment showed that asthmatic patients who had performed slow deep breathing during 15 min sessions, twice a day for 4 weeks, significantly decreased airway reactivity [33]. In another study, asthmatic patients practising deep diaphragmatic breathing were compared with those practising physical exercise, and with a waiting list group, all engaged in a 16 week programme [3]. Deep diaphragmatic breathing resulted in significant reductions in use of medication, intensity of asthmatic symptoms, and a threefold increase in time spent in physical activities. These studies supported the idea that voluntary control of breathing may be useful, but without providing clear evidence for this. Possibly, the ability to control breathing helped the subjects to prevent the exacerbation of the pathophysiological processes by hyperventilation and stress. We might speculate that the ability to control ventilatory behaviour during exercise may be beneficial for the same reasons.

In conclusion, the present experiment supports the idea that changes in breathing pattern during exercise do not necessarily increase dyspnoea. However, directing breathing patterns in the context of exercise rehabilitation requires an individual assessment of the target frequency in order to prevent any detrimental effect on blood gas values.

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