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Effect of Treadmill Exercise on Ventilatory Functions in Bronchial Asthma

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The present study describes the effect of standardised treadmill exercise in 32 asthmatics and 20 healthy controls on airway resistance. The results show an overall incidence of 81.25% of exercise induced asthma (EIA) amongst asthmatics. None of the controls developed EIA. Following two minutes of exercise, asthmatics as well as controls showed a decrease in the airway resistance; manifesting as a rise in PEFR and FEV_1. The rise was significantly more ($P < 0.001$) in controls compared to asthmatics. At 5 and 15 minutes post exercise, however, PEFR and FEV_1 respectively showed significantly greater percentage fall from basal values in asthmatics, than in controls ($P < 0.001$). The increased airway resistance started to normalise 20 minutes after exercise and reached near basal values at the end of 30 minutes both in controls and asthmatics.

Introduction

Many asthmatics, especially children, experience acute, usually self limiting, airway obstruction. after strenous physical exertion. The exercise induced airway obstruction in a known asthmatic is termed 'exercise induced asthma,' (EIA). Earlier, it was believed that EIA is a seperate and a distinct clinical entity. It is now well established however, that exercise is but one of a number of methods of provoking an attack in an asthmatic subject.

The incidence of EIA has been reported to be from 75% to 90% in various studies. [3], [4], [13]. Whilst almost all children experience EIA, its variable and low incidence in adults is probably because they never take enough exercise. Thus the true incidence of EIA in asthmatics may be 100% and indeed, failure to elicit EIA should cause the diagnosis of asthma to be reconsidered.[9] EIA can therefore be used as a diagnostic test to confirm the presence of bronchial asthma and distinguish patients from those with other respiratory diseases with airway obstruction.

The present work describes the effect of treadmill exercise on ventilatory functions in known asthmatics.

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Patients & Methods

All patients studied had asthma as defined by American Thoracic Society (1962). Detailed clinical examination including X-ray chest and a resting electrocardiogram was done and subjects with co-existing cardiovascular disease; pulmonary tuberculosis or bronchitis were excluded. Patients with a history suggestive of allergic diathesis (hay fever, urticaria, eczema) early age of onset, seasonal variations were classified as extrinsic asthmatics. IgE estimation or skin tests with allergens were not done.

Controls included normal healthy volunteers with no past or family history of asthma or atopy.

A standardised protocol[8,10,13] of treadmill exercise was employed. All medications were stopped 12 hours prior to exercise which was performed with subjects on empty stomach. The protocol included measurement of resting peak expiratory flow rate (PEFR) on the Wright's peak flow meter and Forced expiratory volume first second (FEV_1) on Godart's pulmo test. After recording the resting heart rate, the subject was made to run on treadmill (model Venky's) for two minutes. The gradient of the treadmill was fixed at 10% for all subjects. The speed was adjusted to attain a heart rate of 85% of the predicted maximum for that individual. PEFR and FEV_1 were determined at the end of the exercise. The subject was made to rest till his heart rate returned to the basal pre-exercise level; at the end of which he was again exercised on the treadmill keeping the exercise protocol at the predetermined submaximal level, for a period of six minutes. PEFR and FEV_1 were again measured immediately on cessation of exercise and at 2,5,10,20 and 30 minutes respectively.

EIA was said to exist if PEFR and FEV_1 in the patient fell by 15% or more of the pre-exercise value.

EIA was graded as mild, moderate or severe as per the criteria of Cropp et al.[4,5]

The studies were conducted at ambient temperature and humidity. No environmental control was exercised.

For statistical analysis standard difference of mean and unpaired 't' test were employed.

Results & Observations

Details of subjects studied is given in Table 1. Of the 32 cases with bronchial asthma, 6 were children with a mean age of 10 years. Seventeen were adult males with a mean age of 30 years and 9 females with an average age of 28 years.

The controls included 10 adult males and females with a mean age of 30 years and 26 years respectively. The duration of asthma was variable and averaged 5.8 years in adults and only 2.5 years in children.

In the majority of children (66.6%) a past history suggestive of EIA was present; whereas only 23.5% of adult males had exacerbations of airway obstruction on exercise. None of the females interestingly had experienced asthma on exercise earlier.

Majority of children (83.3%) belonged to extrinsic group, whereas 61.6% of adults had intrinsic asthma.

Table 2 shows comparison of exercise tolerance on treadmill between healthy controls and asthmatic subjects. The average speed on treadmill required to reach submaximal exercise levels was significantly lower in asthmatics than in controls ($P<0.001$). PEFR and FEV_1 were, likewise, lower in both adult male and female asthmatics compared with healthy controls ($P<0.001$).

The incidence of EIA in the subjects studied is shown in Table 3. All children (100%) manifested EIA. Amongst adults the incidence was higher in females (88.8%) compared to males (70.5%). Overall incidence of EIA in this study was 81.25%. Incidence of EIA when compared between extrinsic and intrinsic group did not differ statistically ($P>0.01$).

Extrinsic asthmatics developed a severer attack of EIA than intrinsic asthmatics. This difference was statistically significant at $P<0.05$.

The changes in airway resistance as measured by PEFR and FEV_1 following 6 minutes of treadmill running in adult males and controls is shown in Fig. 1a. It may be noted that PEFR and FEV_1

Table I

Subjects	Age Yrs Mean (Range)	Duration of Symptoms Yrs Mean (Range)	History of E I A (%)	Extrinsic Group (% age)	Intrinsic Group % age
Children n = 6 (18.75%)	10 (7-12)	2.5 (3/12-6)	4 (66.6%)	5 (83.3%)	1 (16.7%)
Adult Males (53%) n = 17	31 (22-44)	7.3 (1/12-19)	4 (23.5%)	7 (41%)	10 (59%)
Adult Females (28.25%) n = 9	28 (19-38)	4.4 (6/12-6)	0	3 (33.3%)	6 (66.7%)

Table II

	Subjects	Treadmill Exercise Levels Kmph mean (Range)		Resting PEFR L/min mean (Range)	Resting FEV_1 in litres mean (Range)
Adult Males	Controls n = 10	6.8[x]	5-8	585[a] (520-660)	4.08[c] (3.8-4.3)
	Asthmatics n = 17	5.1[x]	3-7	475[a] (370-640)	3.1[c] (3.0-4.6)
Adult Females	Controls n = 10	3.8[+]	(2.7-5)	390[b] (290-480)	3.34[*] (2.7-4.0)
	Asthmatics n = 9	2.9[+]	(2.5-3.75)	290[b] (250-370)	2.46[*] (2.0-3.1)

[x] $P < 0.001$ [+] $P < 0.01$ [a] $P < 0.001$ [b] $P < 0.001$ [c] $P < 0.01$ [*] $P < 0.01$

Table III

Incidence of E I A

Total Number of subjects developing E I A	Children n = 6	Adult (males) n = 17	Adult (females) n = 9	Total n = 32
	6	12	8	26
Percentage %	100	70.5	88.8	81.25

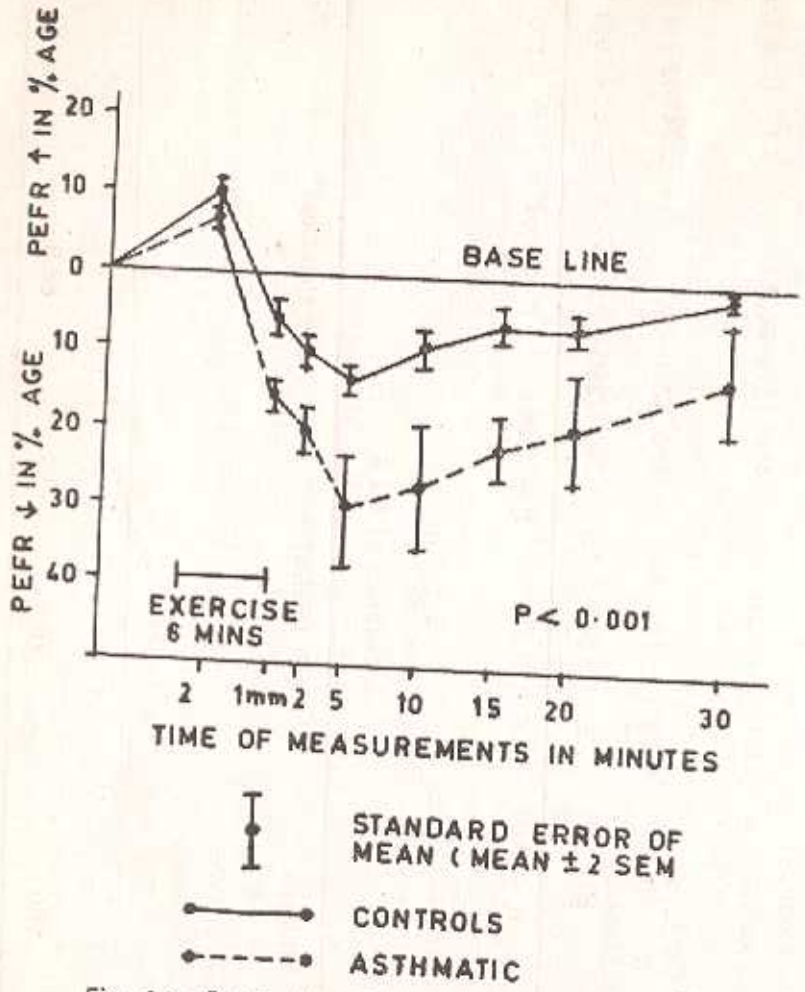

Fig. 1 a *Comparison of response to 6 mins of treadmill running between asthmatic male adults & controls showing changes in PEFR*

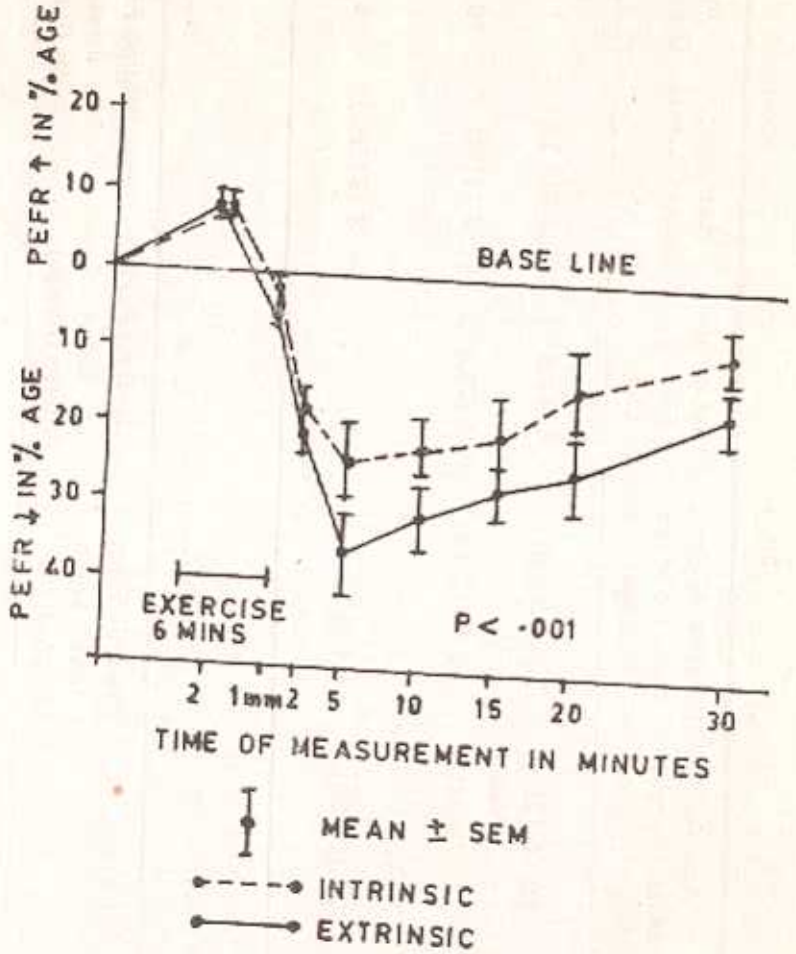

Fig. 1 b *Comparison between extrinsic and intrinsic asthmatics in their response to treadmill running for 6 mins showing changes in PEFR*

show a rise after 2 minutes of exercise: both in asthmatics and controls.

The rise is more in controls compared to asthmatics and this difference is statistically highly significant ($P<0.001$).

PEFR showed a maximal fall 5 minutes after exercise in both controls and asthmatics; however, the percentage fall was significantly more in asthmatics than in controls. ($P<0.001$).

FEV_1 also showed a fall, but the maximal fall was evident at 15 minutes, post exercise; significantly more ($P<0.001$) in asthmatics than in controls.

The airway obstruction improved 20 minutes after exercise and almost normalised at the end of 30 minutes, PEFR and FEV_1 returning to near baseline valves.

The ventilatory parameters showed similar changes in respect of asthmatic females and children.

Fig. 1(b) shows comparison between extrinsic and intrinsic asthmatics in their response to treadmill running for 6 minutes. The PEFR showed a greater percentage fall amongst extrinsic group ($35\% \pm 5$ 2SEM) at 5 minutes, as against ($24\% \pm 4.6$ 2SEM) amongst intrinsic asthmatics. The difference was statistically significant at $P < 0.001$.

Discussion

Exercise is a potent, non-immunological trigger for inducing bronchial asthma. Exercise induced asthma reflects bronchial hyperreactivity, and its presence is an indication not of active bronchial asthma but of the ability to develop an attack given an appropriate allergic, physical or emotional stimulus. EIA does not occur in non-asthmatic subjects.

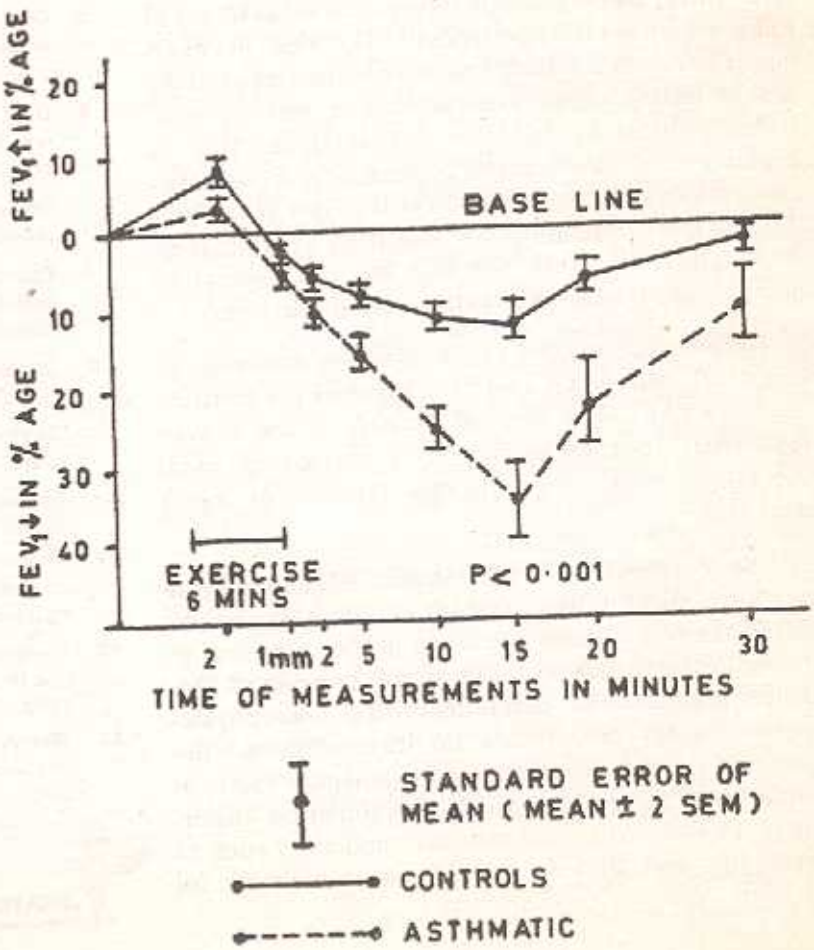

Fig. 2 *Comparison of response to 6 mins of treadmill running between asthmatic male adults & controls showing changes in FEV_1*

The overall incidence of EIA (81.25%) amongst asthmatics, in the present study, compares favourably with the reported studies.[9,13]

The ability to develop asthma after exercise did not show any relation to whether the individual was atopic (extrinsic) or non-atopic (intrinsic). Anderson et al[1] in their studies also found no difference in the response to exercise between extrinsic and intrinsic asthmatics. The severity of EIA was, however, more amongst extrinsic asthmatics as compared to the intrinsic group. The difference may relate to the increased bronchial liability in the atopic asthmatics. Eggleston et al also observed severer EIA amongst subjects with extrinsic asthma.

Even though the majority of test subjects were asymptomatic with no clinical evidence of bronchospasm, airway resistance as assessed by PEFR and FEV_1 showed a significant increase compared to normal subjects. The difference is probably due to sub-clinical increased airway resistance in asthmatics and to their poor physical conditioning. The latter may be particularly relevant in assessment of PEFR which is effort dependent. The rise in PEFR and FEV_1 seen 2 minutes after cessation of exercise is seen both amongst asthmatics and healthy controls ; suggesting initial bronchodilation. This is explained on the increased sympathetic drive associated with exercise. The percentage rise was significantly more in healthy controls than in asthmatics, ($P < 0.001$) ; possibly due to a blunted sympathoadrenal response in asthmatics during exercise.[2,14]

Normal subjects did not display any evidence of bronchial lability on exercise. None of the controls developed EIA. The minimal increase in the airway resistance (not exceeding 15% of resting basal valves) is consistent with the findings of other workers.[1,13]

The mechanism of EIA, remains elusive despite much work in the field. Recent studies have emphasised the role of heat exchange in the airways as the major contributing factor in the genesis of EIA. Others implicate as yet unidentified neurophysiological events responsible for its production.[2] Regardless of the initiating mechanisms, there is evidence to suggest mast cell degranulation resulting in release of bronchospastic mediators such as histamine and SRS-A, which are responsible for EIA.[9,11]

Conclusions

1. It is concluded that treadmill exercise is a potent stimulus and induces significant bronchoconstriction in majority of the asthmatics.
2. It is a non-invasive, simple and readily available diagnostic test, which should be used in separating asthmatics from non-asthmatics. To an extent thus, it may find a place in aeromedical evaluation, in selected subjects.

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