AFRICAN JOURNAL OF MEDICINE and medical sciences

VOLUME 22, NUMBER 2, JUNE 1993

EDITOR: B.O. ONADEKO
ASSISTANT EDITORS:
B.O. OSOTIMEHIN and A.O. UWAIFO

SPECTRUM BOOKS LIMITED
Ibadan • Owerri • Kaduna • Lagos

ISSN 1116–4077

Afr. J. Med. med. Sci. (1993) 22, 33-37

Exercise induced bronchoconstriction in Nigerian asthmatics

* G E ERHABOR, ** A A AWOTEDU and * M O BALOGUN
** Department of Medicine, Obafemi Awolowo University Teaching Hospital, Ile-Ife and ** Department of Medicine, University College Hospital, Ibadan, Nigeria.*

Summary

This study describes the result of free-running exercise performed on 40 asthmatic subjects and 40 age and sex matched controls. A percentage fall of more than 15% in PEFR was used for the diagnosis of exercise-induced asthma (EIA). 77.5% of our asthmatic patients had exercise-induced bronchoconstriction (EIB) while none of the control subjects exhibited EIB.

Twenty-seven patients gave positive history of EIA out of which 25 (92.59%) had positive exercise testing. Conversely, 13 patients with asthma did not give history of EIA but 6 of this (46.15%) were found to have EIB on exercise. History alone is therefore insufficient to diagnose patients with exercise-induced asthma.

Resume

Cette etude a pour but la description de l'effet de l'exercise non-controlle chez 40 sujects asthmatiques et 40 temoins de l'age et sexe correspondants. Une baisse de plus de 15 pourcent a ete utilise pour la diagnose de l'asthme induit par l'exercise (EIA). 77,5 pourcent de nos sujects asthmatiques ont developpe la bronchoconstriction induit par, l'exercise (EIA), alors que less 40 temoins n'ont pas developpe de bronchoconstriction.

27 malade donnaient une positive histoire de l'EIA, parmi lesquels 25 (92,5 pourcent) avaient un test positif d'exercise. Reciproquement, 13 malades avec l'asthme n'ont pas donne de positive histoire de EIA, mais 6 parmi eux (46,15 pourcent) ont ete, trouves d'avour EIA suivant l'exercise. Alors, l'histoire seule ne suffit pas pour la diagnose de l'asthme induit par l'exercise.

Correspondence to: Dr. G E Erhabor, Department of Medicine, Obafemi Awolowo University Teaching Hospital, Ile-Ife, Nigeria.

Introduction

Exercise is one of the non-specific stimuli that have been identified in the pathogenesis of asthma[1]. Historically, exercise as a provocating factor of asthma was recognised as far back as the 17th Century; athough it was not until 1946 that Herxheimer conclusively documented a causal relationship between exercise and asthma[2]. Many patients with asthma frequently develop acute, reversible and often self-limiting bronchospasm after or near the end of muscular exercise[3]. This response may require quantitative assessment, because of the poor correlation that exists between symptoms and laboratory evidence of exercise-induced bronchospasm[4].

The general mode of action of exercise is now believed to involve the autonomic (vagal) discharge as well as mast cell mediator release[5-7]. During the active phase of exercise the autonomic discharge is believe to maintain bronchodilatation which is more intense than mediator release. The discharge ends abruptly towards the end of exercise with the release of mediators from mast cells which now produce bronchoconstriction. There after, there is a refractory period of some hours during which further exercise fails to induce bronchospasm[5].

The refractory period following exercise is explained on the basis of the time delay for re-synthesis of fresh mediators. Drugs such as disodium cromoglycate and nifedipine (a calcium antagonist) abolish EIA when given prior to exercise by preventing mediator release[8].

While this general hypothesis may explain the major features of EIA it however does not explain the mechanism by which both vagal and mediator pathways are stimulated. It also does not explain the reasons why various exercises have different potentials for inducing bronchospasm in the

asthmatic subjects. While cycling, swimming and walking can induce bronchospasm in the asthmatic it is free running that is the most sensitive[2]. Earlier hypothesis to explain this phenomenon include hypocapnoea and ventilation perfusion abnormality[9,10]. The heat loss theory and water (also referred to as osmolarity) hypothesis have been advanced in recent times[7].

Incidence of EIA vary from 60-100% in most European studies[4,5]. However, there is no information about its incidence in the African although anecdotal report suggests that it is not common. Since many adults in this environment do not engage in asthmatogenic exercises such as free-running, the above observation is suspect. It is therefore in an attempt to find out the true picture about the relationship between asthma and exercise that we embarked on this study.

Materials and methods

Forty asthmatic patients attending the Chest Clinics of both the Obafemi Awolowo University Teaching Hospital Complex, Ile-Ife and the University College Hospital, Ibadan, were recruited into the study. The diagnosis of asthma was based on the criteria of the American Thoracic Society[11]. Forty age and sex matched patients without history of asthma or any cardio-respiratory diseases served as control subjects. Both subjects and controls were aged between 10 and 50 years; and did not have any cardiovascular disease that would make exercise hazardous.

Only patients with mild to moderate asthma were studied. These patients must not require more than inhaled steroid or bronchodilators for maintenance. Furthermore, the peak expiratory flow rate (PEFR) must be above 70% of their predicted value for age and sex.

Exercise test

All medications were withdrawn at least 12 hours before exercise test. The exercise test was done in an open field in each of the hospitals. The subjects were requested to run as fast as they could during the first minute and to maintain a steady pace during the next 6 minutes. The exercise was of sufficient severity to give a heart rate of 80% of predicted maximal heart rate[12]. Peak expiratory flow rate was measured using the Wright's Peak Flow meter. The exercise was only interrupted when the PEFR was being measured and the best of 3 measurements obtained at any instance was recorded. The PEFR was recorded every minute during the exercise and also at 3, 5, 8, 10, 15 and 20 minutes after the exercise. The control subjects were also exercised under the same environmental conditions as the asthmatic subjects. Equal number of asthmatics and control subjects were tested in the 2 towns (i.e. Ile-Ife and Ibadan). This is to allow for standardization of results.

The following indices were calculated for each of the subjects:

i. $\% \text{ rise} = \dfrac{[\text{Highest PEFR attained}] - [\text{Resting PEFR}]}{\text{Resting PEFR}}$

ii. $\% \text{ fall} = \dfrac{[\text{PEFR}] - [\text{Lowest Post Exercise PEFR}]}{\text{Resting PEFR}}$

Exercise-induced bronchospasm was considered present if there is a percentage fall of more than 15%.

The student '*t*' test was used to test the level of significance of PEFR values between asthmatic patient and normal controls.

Results

The total number of asthmatic and control subjects studied was 40 subjects in each group. The age and sex distribution is as shown in Table 1. Twenty males and twenty females were studied in both the asthmatic and control groups and they were matched for age and sex.

Table 1: Age and sex distribution of asthmatic subjects and controls

	Asthmatics (n = 40)		Controls (n = 40)	
Age (Years)	M	F	M	F
10 - 19	6	6	4	6
20 - 29	7	11	12	13
30 - 39	6	3	3	1
40 - 49	1	—	1	—
Total	20	20	20	20

The mean age of the asthmatics was 24.12 ± 7.72 years while that of the control was 23.70 ± 5.18 years ($P > 0.05$). Fig. 1 shows the graph of the percentage rise and percentage fall in both the asthmatic patients and control group.

The percentage rise in the asthmatic and control groups were 12.47 ± 9.70 and 8.50 ± 6.63 respectively ($P > 0.05$). The difference between the two being non significant. The percentage fall in PEFR in the asthmatic and control groups were 26.60 ± 16.3% and 0.27 ± 4.11% respectively ($P < 0.001$).

The distribution of the maximum fall is shown in Fig. 2. All the control subjects had a less than 15% fall, no change or an actual increase in PEFR. The asthmatic subjects had the greater fall in PEFR, 38 (95%) of them had a fall in PEFR post exercise but it was greater than 15% in only 31 (77.5%) of them. It is only these 31 patients who could be said to have Exercise-Induced Bronchospasm. Further breakdown of the 31 patients revealed that 11 (27.9%) of them had percentage fall of between 15 - 20%, 17 (42.5%) had a fall of 21 - 20% while 3 (7.5%) had a fall greater than 50%.

From the above, it could be inferred that at 15% fall, the sensitivity of the exercise test was 77.5% while the specificity was 100%. Fig. 3 shows the maximum fall in PEFR as a percentage of pre-exercise value for each asthmatic subject and control.

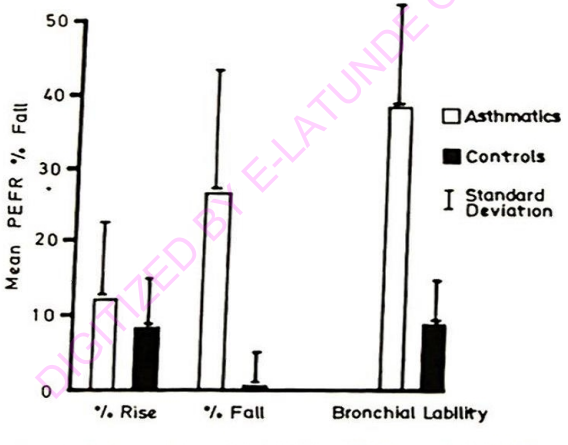

Fig. 1: Indices of bronchial liability (% Rise and % Fall) in asthmatics and controls

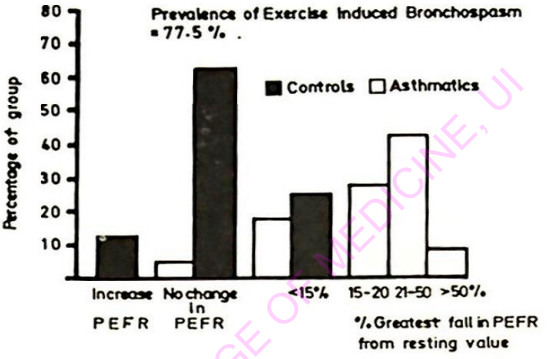

Fig 2: Distribution of greatest fall in PEFR of asthmatics and controls (Post exercise) in this study.

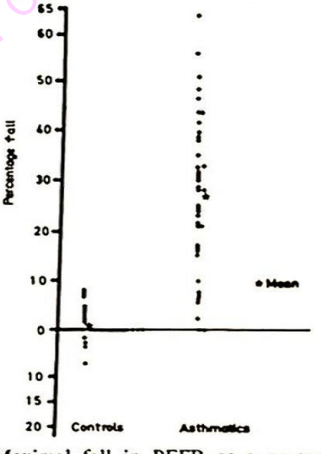

Fig 3: Maximal fall in PEFR as a percentage of pre-exercise value for each subject (each point represents the single most severe response to exercise in each subject).

The relationship between the history of exercise-induced bronchospasm and exercise test was ascertained. The results are noted in Table 2. This showed that 27 patients gave a positive history of EIB (Exercise-Induced Bronchospasm) out of which 35 (92.5%) had a fall in PEFR of 15% or more on exercise test.

Table 2: The relationship between the history of exercise-induced asthma (EIA) and exercise-induced bronchospasm (EIB)

History	Proven EIB		
	No.	No.	%
Positive	27	25	(92.59)
Negative	13	6	(46.15)

Correspondingly, 6 (46.15%) of the 13 subjects who denied any history of EIB had a positive exercise test results.

Discussion

This study has confirmed that majority of Nigerian asthmatics will develop bronchospasm after exercise like free- running. Prevalence of EIA in this study was found to be 77.5% which is low when compared with figures reported from European studies. McNeil *et al*[13] reported 100%, Jones *et al* 99%[14] and Godfrey *et al* 91%[2]. The differences in these results is more likely due to the varying cut-off points of percentage fall in either FEV_1 or PEFR used in the studies.

Free running has been reported to be the most sensitive exercise for exercise-induced bronchospasm. A major problem with this is that the study is usually conducted outdoors with no control of temperature, allergens or pollutants. It is an established fact that the climatic condition under which exercise is conducted should influence the severity of EIA. Patients who exercise in hot humid conditions are known to have less bronchospasm when compared with those who exercise in a cold dry climate[15].

The above factors which may influence EIA partially explain the different incidence rates of EIA in the different studies earlier mentioned. Another major source of variation in the incidence of exercise responsiveness has been the use of arbitrary selected criteria of abnormality. Earlier studies were not well standardized as cut-off percentage fall in PEFR or FEV_1 varied from 10%, 15% and 20%[2]. It is however now agreed that a 15% or more fall in FEV_1 is diagnostic of exercise induced asthma[2,16,17].

In this study, most of the patients who have history of EIB developed bronchospasm on exercise, however, 46.15% of patients who denied any history of wheezing on exercise developed EIB when exercised. It is therefore apparent that history alone is not a totally reliable means of identifying individuals with EIA. The reason for this is that in this environment, many patients with or without asthma do not engage in physical exercise such as free running.

Patients with exercise-induced bronchospasm need to be recognised and advised about the relationship between asthma and the various exercises.

References

1. McFadden. Exercise and asthma. N. Eng. J. Med 1987; 317: 502-504.
2. Godfrey S. 'Exercise-Induced Asthma' In Asthma Clark J H and Godfrey S. (eds) London: 'Chapman and Hall', 1983.
3. Gropp O A A. Grading, time course and incidence of exercise-induced airway obstruction and hyperinflation in asthmatic children. Paediatrics 1975; (56): 868-879.
4. Derman C W, Kawabore L, Pierson W X. Incidence of exercise-induced asthma in children. Paediatrics 1975; (56) (Suppl.): 847-850.
5. Edmunds A T, Tooley M and Godfrey S. The refractory period after exercise-induced asthma, its duration and relationship to severity of exercise. Am. Rev. Resp. Dis. 1978; 117: 247-
6. Deal E C, Wasserman S N, Soter N A, Ingram R H, and McFadden E R. Evaluation of role played by mediators of immediate hypersensitivity in exercise-induced asthma. J. Clin. Invest. 1980; 65: 659.
7. Lee T H and Anderson S D. Heterogeneity of mechanisms in exercise-induced asthma. Thorax 1985; 40: 481-487.
8. Barnes B J, Wilson N M and Brown M J. A calcium antagonist, nifedipine, modifies exercise-induced asthma. Thorax 1981; 36: 276.
9. Rebuck A S and Ready Y. Exercise-induced asthma. Lancet 1986; 2: 429-436.
10. Katz R M, Whipp B J, Helmlich E M and Wasserman K. Exercise-induced bronchospasm ventilation and blood gases in asthmatic children. J. Allergy 1971; 47: 148-157.
11. American Thoracic Society. Definitions and classifications of chronic bronchitis, asthma and pulmonary emphysema. Am. Rev. Res. Dis. 1962; 85: 762-768.
12. Balogun M O and Ladipo G O A. Cardiovascular responses to maximal treadmill exercise in healthy adult Nigerians. Afr. J. of Med. 1989; 18: 109.

13. Mcneil R S, Nairu J R, Miller J S and Ingram C G. Exercise-induced asthma. Q.J. Med. 1966; 35: 52-67.
14. Jones R S, Bustan M H and Wharton M J. The effect of exercise on ventilatory function in the child with asthma. Br. J. Dis. Chest 1962; 56: 78-86.
15. Kattan M, Keens T, Mellis C M and Levison J. The response to exercise in normal and asthmatic children. J. Paediat. 1978-721.
16. Crofton and Douglas's Respiratory Diseases 4th Edition Seaton A, Seaton D, and Leitch A G. (eds) Oxford: Blackwell Scientific Publications, 1989.
17. Harrison B D W. In Thoracic Medicine Emerson P. (eds), Buttersworths Publication. 1981.

(Accepted 14 August, 1990).