Asthmatic Child and Sport

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Motto. *If from running, gymnastic exercise, or any other work, the breathing becomes difficult it is called asthma. The symptoms of its approach are heaviness of chest, difficulties of breathing in running or on a steep road.*

Aretaeus the Capadochian, 2nd Cent. A.D.

Paradoxically, 1800 years had to elapse before the condition, so graphically described by Aretaeus, achieved international prominenece a gold medal winner in 1972 Summer Olympic Games was disqualified for having taken an ephedrine containing bronchodilator prior to swimming.

Most people with asthma, experience an attack when they exercise, which is symptomatically and physiologically quite similar to asthmatic attacks induced by other stimuli. At the peak of an attack the person is coughing, dyspnoeic and wheezy and a variety of pulmonary functions, if tested, are abnormal. Although exercise-induced asthma (EIA) is almost a universal finding in asthmatic subjects if sought intensively, it is largely seen in children, probably because they exercise more than adults and because of the prevalence of allergic asthma in them. The condition is so frequent in children that it alone can be used as a simple diagnostic test (EIA occurs also in approximately 40 per cent of those who have only allergic rhinitis or hay fever). It is most likely EIA to be a clinical problem in school children as it interferes with their physical education activity and games. What can be done to prevent it? The most obvious solution would be to avoid exercise and that was what the asthmatic children were advised to do up to some 8-10 years ago. Since then the attitude has changed towards the physical activity in asthmatics, thanks to a substantial body of knowledge concerning EIA acquired during the previous 15-20 years; and the increasing participation of asthmatics in competitive sports, many with distinction, clearly indicate that most asthmatics should not be considered physically handicapped. So, nobody familiar with children and young adults will advise them to abstain from physical exercise at a time when psychosocial growth depends on interaction with peers and interaction centers around physical activity. Yet,even today, because of the restrictive symptoms of EIA and anachronistically overprotective attitude of some parents and teachers, the asthmatic child may choose inactivity while the doctors are asked to cover it by writing excusing notes. In the light of the increasing demand placed upon paediatricians and family physicians for authoritative opinion concerning their asthmatic patiens' ability to be engaged in individual or team sport activity, it is timely to try to answer the question: Where do we actually stand with regard to the physical activity in young asthmatics? The series of excellent contributions presented recently at a symposium on specific problems facing athletes with asthma, provides us with the most up to date answer to the question[1]. Though the issue reached us in the wake of the 1984 Olympic Games, the symposium was held on the eve of the Los Angeles events. Eight years ago, while in Paris studying the ventilatory effects of physical exercise in asthamtic children[2], I profited a great deal from the proceedings of a similar symposium which was a prelude to the 1976 Olympic Games[3]. By comparing these two reviews, which reflect the state of the art of today and that of eight years ago, one realizes how far our knowledge of the EIA has advanced in a relatively short period of time. Though many of the facts established by 1976 have remained unchallenged, in the past 8 years we have begun to understand better some of the enigmas surrounding EIA. New advances in the understanding of the underlying pathophysiological mechanisms involved in EIA have paved the way for its pharmacological control, which enables asthmatic children to live as normal a life as possible, asthmatic athletes to compete effectively in their respective sports, even to win gold medals in Olympic Games.

Exercise is an integral part of the life of asthmatic children and youths. Physical education, individual athletics and team sports activity interact with asthma in many ways and are thus a legitimate area of interest for present and future physicians. I feel that it is therefore, worth here to reitirate some of the basic points of the phenomenon of EIA.

EIA is characterized by a post-exercise bronchospasm with maximal fall in lung function some 5-10 minutes after stopping the exercise. At the beginning of exercise there is usually some mild bronchodilation that is replaced by the onset of bronchospasm after 4 to 8 minutes. Different types of exercise of identical severity result in different amount of EIA. Free range running causes the most severe asthma, cycling somewhat less and swimming least. The variable effects of different activities are an important factor to stress the young asthmatic patients who want to compete in athletics. Swimming is their sport of choice and no wonder that of 5 asthmatic gold medalists in the 1972 Munich Olympic

Games, all of them were swimmers. While the severity of the attack augments in linear fashion with increasingly arduous exercise, the relationship between exercise duration and severity of the attack is more complex. Severity increases linearly up to 5-6 minutes of exercise but after that the response levels off, then gradually diminishes. After the attack of EIA the subject is relatively refractory to another attack, the magnitude of the refractiveness diminishing during the next 3 to 4 hours. This suggests that either the ability of bronchial smooth muscle to respond to stimuli is diminished or that mediators were released through mechanisms that require a definite time to reactivate. Thus *running through* phenomenon, although poorly understood, allows asthmatic youths to participate successfully in games, such as basketball, hockey or soccer, which involve interrupted exercise which is less likely to cause EIA than are continuous endurance events. As a matter of fact, a series of short sprints is a useful technique for inducing a refractory period of EIA. Furthermore, the warm-up techniques used by all athletes tend to induce a measure of refractoriness[4] [5].

8 years ago it was widely accepted that EIA was caused by the release of mediators from mast cells through some unknown exercise-related mechanism (hyperventilation was invoked among many other possible triggering mechanisms). At that time it was not clear why swimming, even if of severe intensity, failed to induce bronchospasm. Later on a Boston group of investigators noted that breathing humid air attenuated EIA and this could explain the low asthmogenicity of swimming[6]. They advanced a theory which postulates that cooling of the airways is the primary stimulus for the onset of EIA. Physical exertion produces an increase of minute ventilation to provide sufficient oxygen for the metabolic needs of working muscles. This results in large volumes of incompletely conditioned air being inspired, drying and cooling the upper airways as heat and water are transferred from their surface. The total quantity of heat exchanged varies directly with minute ventilation and inversely with the temperature and water content of the inspired air. Variations in temperature and humidity of inspired air accounts for differences in asthmogenicity of such tasks as running, cycling, walking and swimming rather than intrinsic properties of different types of exercise. This attractive concept has been more recently challenged by a group of authors, who think that the initiating event in EIA might well be the loss of water and not the loss of heat from the airways during the hyperventilation of exercise[7] [8]. The loss of water could possibly trigger EIA via a transient increase in osmolarity of the airway surface. The same as the original heat loss theory, water loss theory fails to explain how hyperosmolarity of the airway surface fluid generates bronchoobstruction. A vagal reflex, induced via irritant receptors seems likely but this does not exclude the possibility that mediators are released from airway mast cells.

What all this means for the asthmatic athlete is that he should try to avoid exercising in cold, dry conditions and choose swimming rather than skiing (or ice skating, or ice hockey) as his preferred sport. In this context it might be of interest to know that some 8-10 per cent of the Australian Olympic Team were asthmatic in 1976 and 1980 and of those slightly more than a half were swimmers[5].

Increased bronchial reactivity is the fundamental abnormality in asthma. The underlying cause of this abnormality is uncertain but the most widely accepted hypothesis attributes it to imbalance of autonomous control with predominance of excitatory (cholinergic and alpha-adrenergic) or a deficiency of inhibitory (beta-adrenergic) control. Very recently this concept has been modified by involving the recently discovered third autonomous, non-adrenergic non-cholinergic (NANC) nervous system[9]. It postulates that non-adrenergic inhibitory nerves may exert a braking effect on bronchoconstriction and a functional defect, presumably present in asthmatics, would lead to exaggerated responses to constrictor stimuli. Their neurotransmitter, vasoactive intestinal peptide (VIP) has been shown to relax smooth muscle in vitro and is bronchodilator in both animals and man. The results of very recent studies provide further evidence that VIP given intravenously or inhaled may have a place in the treatment of severe bronchospasm [10] [11]. The demonstration of non-cholinergic substance P-containing nerves in human lungs is also clinically relevant since this peptide can produce bronchoconstriction, bronchial mucosal oedema and mucus hypersecretion, all of which are features of asthma. On the other hand, cells other than mast cells and mediators other than histamine and neutrophil chemotactic factor (NCF), such as leukotriens and prostaglandins, must certainly play some role in the production of hyperreactivity of the airways in asthmatics.

What concerns the young asthmatic athlete on this point is that severity of EIA depends on the degree of bronchial reactivity which can be influenced by both allergic and non-allergic stimuli. A previous contact with the causative allergens either in naturally occuring asthma or in bronchial challenge tests, renders them more hyperreactive to exercise. The same applies to the atmospheric pollutants, such as ozone, sulphur dioxide and nitrogen oxide, which enhance bronchial activity and cause more severe EIA [12]. It has been shown recently that the level of ionization of the air can also affect EIA[13]. In the existing polemics over the use of negative ion generators in the treatment of asthma, this makes their prescription even less justified[14]

Despite all the advances made to improve our understanding of the pathophysiology of EIA, the mechanisms by which exercise produces acute episodes of bronchoconstriction has not been worked out. It has been established, however, that EIA can be prevented by several pharmaceutical agents. The most important point with regard to managing patients with EIA is to emphasize the fact that it can be controlled

and they should not refrain from normal physical activity.

Numerous drugs have been tried as modulators of the bronchial response to exercise. Some of them have established themselves as effective or partially effective in prevention of EIA, others have already been discarded as ineffective while trials with the third ones are still under way.

EIA can be prevented by Beta-adrenergic drugs, theophylline and cromolyn sodium. Beta-sympathomimetic agonists, especially the more recent selective Beta-2 agents, are generally agreed to be the most effective. (They act through adenyl-cyclase to increase intracellular concentrations of cyclic adenosine monophosphate - c-AMP) Most researchers suggest that the inhaled route is the most practical as the drug is effective when used immediately before exercise[15]. Oral sympathomimetics are equally effective but should be taken at least 1 hour, and preferably 2 hours before exercise[16]. There is also an agreement that cromolyn and theophylline are less effective than Beta-2 selective agents administered by aerosol (Methyl-xanthines act through inhibition of phosphodiesterase which degrades c-AMP). On the other hand disodium cromoglycate (DSCG, cromolyn) has the advantage of preventing both the immediate and delayed type of response and is virtually free of side effects. The precise mode of action of DSCG is still unclear. It is difficult to account for all its properties on the basis of mast cell stabilization since relatively high concentrations of the drug are required to inhibit mediator release from sensitized human lung fragments, challenged with specific allergen[17]. An important aspect of bronchial asthma is bronchial hyperreactivity and it has been suggested that the drug might have a direct influence on smooth muscle tone[18]. This action is, probably, responsible for the most important therapeutic effect of DSCG, the ability of the drug to reduce bronchial hyperreactivity in the long run[19]. Alternatively or in addition, the drug may act on certain neurological/reflex pathways, such as irritant receptors[20]. For patients regularly being treated by cromolyn it is worth taking an extra dose, alone or with Salbutanol, immediately before strenuous exercise. The need for a dose of Salbutamol would also apply to patients under regular treatment with theophylline which seems to have a similar efficacy to DSCG in prevention of EIA.

Atropine and its isomer Ipratropium bromide *(Atrovent)* appear to be effective in selected patients, especially in those with chronic bronchitis which condition is rare in young asthmatic patients. These drugs may have a place for the treatment of those patients unresponsive to Beta-agonists but are unlikely to be useful for the athlete with EIA. The same applies to the newly introduced preparation *Duovent*, in which Ipratropium bromide is combined with the bronchoselective Beta-agonist Fenoterol [21] [22].

Although steroids are effective in chronic asthma and in restoring Beta-agonist responsiveness in acute severe asthma, their benefit in protecting against EIA is minimal. However, a recent study of Budesonide, a new steroid aerosol, showed protection against EIA. after 4 weeks treatment[23]. But, further evaluation will be needed before any definite recommendations are to be made.

While classical antihistamines are ineffective in preventing EIA, prophylaxis by Ketotifen, an orally active antihistamine with cromolyn - like antiallergic effects, has been controversial[24]. According to a very recent study, Terfenadine, a new potent histamine receptor antagonist, has been shown to be effective in prevention of EIA[25].

Some Alpha-adrenergic blocking agents have been shown to be effective in EIA protection (Indoramin, Phentolamine. Isoxsuprine). These drugs may be of particular benefit to patients with both asthma and coronary disease[26], though it is hardly to expect such patients to participate in more strenuous sport activities.

The intracellular concentration of free calcium ions regulates many functions of the cells, including secretion, contraction, transport process, and mobility among others. All the pathogenetic processes in asthmatic airways are calcium dependent phenomena: excitation - contraction coupling in smooth muscle, stimulus - secretion coupling in mast cells and mucosal glands, nerve impulse initiation and conduction , and development of inflammatory infiltration. The intriguing concept of the possible role of calcium ions in the development of hyperreactivity of the airways in asthma has already led to therapeutic trials with calcium channel blockers, such as niphedipine and verapamil. Although they may effect EIA, airway tone, mast cell mediator release and experimental anaphylaxis, given the existing evidence, it seems unlikely that present agents will play a major role in the treatment of asthma. Maybe a new generation of calcium ion antagonists will find better place in management of asthma, including the prevention of EIA[27] [28].

The philosophy of total rehabilitation of the asthmatic child aims at enabling him/her to live as normal a life as possible, *comme tout le monde et avec tout le mond,* as the French nicely put it. The present body of knowledge concerning EIA and the existing pharmacologic arsenal for its prevention make this goal feasible.

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In Malta plain *Xylocaine* is used for local infiltration prior to perineotomy but the dose has to be less than 3 mg per kg body weight.

Conclusion

Improvement in the control of labour pain by inhalation methods can be *enhanced* if midwives cooperate, and *ensured* if an anaesthetist is present in the labour ward. If we are to venture into the field of routine epidural blocks for labour and delivery, adequate budgeting has to be done beforehand in the provision of manpower, in terms of both nurses and doctors, so that the same good results can be obtained in regional analgesia as in general anaesthesia.