Exercise induced rhinorrhoea (athlete's nose)

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Rhinorrhoea is a common complaint caused by infection, allergy, and vasomotor rhinitis. Some noses drip in cold weather, and this extra secretion may be necessary to moisten the cold dry air, a task at which the nose is particularly good.¹ Healthy people are not infrequently seen clearing their nose or throat when actively participating in sport during all seasons, and our study investigated this habit.

Subjects, methods, and results

Eight healthy non-smokers (six men, two women) aged 16 to 45 were studied. None had resting rhinorrhoea. Two had had mild asthma in childhood but were currently clinically and physiologically normal. To measure nasal secretion we asked the volunteers to blow their noses using a previously weighed paper tissue, which was placed in a sealed container and weighed again. Nasal resistance was measured by posterior rhinomanometry,² which is sensitive to change induced by exercise. A mean of five readings was used at each time interval. Expiratory peak flow rates were measured with a Wright's peak flow meter. The exercise consisted of free running in a corridor. During this and the subsequent rest period the volunteers tried to breathe through their mouths to prevent direct nasal evaporation or air stimulus. Eight and four minutes before exercise the volunteers blew their noses, and these secretions were discarded. Exercise was performed from time 0 for 12 minutes, followed by 12 minutes' rest. Measurements were made every four minutes. A paired *t* test was used to analyse the significance of the results.

The figure shows that there was a significant ($p=0.023$) increase in nasal secretion during exercise, but this returned to normal 12 minutes later. Secretion had started to increase by four minutes of exercise. The exercise was sufficient to cause a significant decrease ($p<0.02$) in nasal resistance throughout exercise, which returned to baseline eight minutes later. Only

Weight (mg) of normal secretions collected over preceding 4 minutes for eight subjects at beginning and end of 12 minutes' exercise and at 12 minutes after exercise

one subject, who subsequently gave a history of mild asthma in childhood, decreased his peak flow rate by 15%.

Comment

These results directly show an increase in nasal secretion during exercise. The maximum value of 150 mg/min in one of these jogging subjects might well explain the rhinorrhoea observed in competitive football and other sports.

The combination of increased nasal secretion with decreased nasal resistance enables humidification of the larger volume of air taken in during exercise. The secretions were not chemically analysed but probably originated from the large anterior serous glands and the generally distributed seromucinous glands and goblet cells. Our method of measurement underestimated the increased secretion since this normally flows posteriorly and is swallowed or expectorated.

Decreased nasal resistance during exercise is probably due to the activity of sympathetic nerves on the capacitance vessels, although overall blood flow may increase because arteriovenous anastomoses are opened up.³ Secretion from both serous and mucous glands is thought to be increased by parasympathetic nerve stimulation.⁴ This would mean that during exercise the nasal mucosa is under the influence of both branches of the autonomic system rather than being subject to the more usual concept of a push-pull system. Stimulation of adrenergic nerves can increase watery secretion in the mucosa of some animals.⁵ Increased circulating catecholamines concentrations and adrenergic activity in the mucosal blood vessels during exercise may thus contribute to the production of the nasal secretions reported here. The relative influence of the non-adrenergic, non-cholinergic system is not clear.

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Acute laryngeal stridor with respiratory arrest in drug induced systemic lupus erythematosus

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Renal and nervous system dysfunction is rare in drug induced systemic lupus erythematosus. We report a case of bilateral 10th cranial nerve palsy in a patient due to hydralazine related systemic lupus erythematosus.

Case report

A 73 year old white woman was admitted with acute right sided pleuritic pain. For several months she had been feeling generally unwell with pain and swelling of forearms and wrists. She was noted by her general practitioner to have a mild anaemia and raised erythro-

cyte sedimentation rate. She had had hypertension diagnosed in 1960 and was treated initially with guanethidine, changing to a β blocker in 1981, but control was never satisfactory until hydralazine 25 mg twice daily was added.

She had a low grade fever, a purple, papular rash affecting both legs, and a pleural rub on the right. Haemoglobin concentration was 97 g/l with normochromic cells, and the erythrocyte sedimentation rate was 70 mm in the first hour. Chest x ray examination showed clear lung fields and a ventilation-perfusion scan showed no evidence of pulmonary emboli. Over several hours she developed a hoarse voice followed by acute stridor. Indirect laryngoscopy disclosed bilateral vocal cord palsies with both cords in the midline. Shortly afterwards she collapsed with an acute respiratory arrest requiring endotracheal intubation. Emergency tracheostomy was performed and post-operatively she collapsed again with rapid atrial fibrillation responding to cardioversion followed by digoxin.

Hydralazine induced systemic lupus erythematosus causing bilateral 10th nerve palsies was diagnosed. She was treated with pulsed doses of intravenous methyl-