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Psoas muscle hypertrophy: mechanical cause for "jogger's trots?"

Although gastrointestinal disturbances occur in up to 30% of recreational or competitive runners,[1] they are poorly recognised by the medical profession, and the mechanism of their production remains speculative.[2 3] We report a case where "jogger's trots" may have been produced by mechanical compression of the colon.

Case report

A 36 year old man presented with a three year history of frequent bowel motions since starting competitive running. During training (10 miles (16 km), five times weekly) and competitive marathons he had the urge to defecate every 30 minutes; he passed formed stools without blood, mucus, or abdominal pain. He had less frequent bowel motions during fell running than road running, and cycling did not cause an increase in frequency. He had no other symptoms. When resting he produced two formed stools daily.

Results of physical examination, including sigmoidoscopy, were normal. Stools were formed and without occult blood. The following investigations gave normal or negative results: full blood count; plasma viscosity; liver function tests; urea and electrolyte concentrations; serum calcium, thyroxine, vitamin B_{12}, and folate concentrations; stool culture; three day faecal fat concentration; jejunal biopsy, including disaccharidase enzymes; and barium follow through. A profile of gut hormones was not performed. Abdominal ultrasonography showed grossly enlarged psoas muscles occupying half of the anteroposterior diameter of the abdomen. Barium enema showed reproducible extrinsic compression of the colon by both psoas muscles during flexion of the hip (figure).

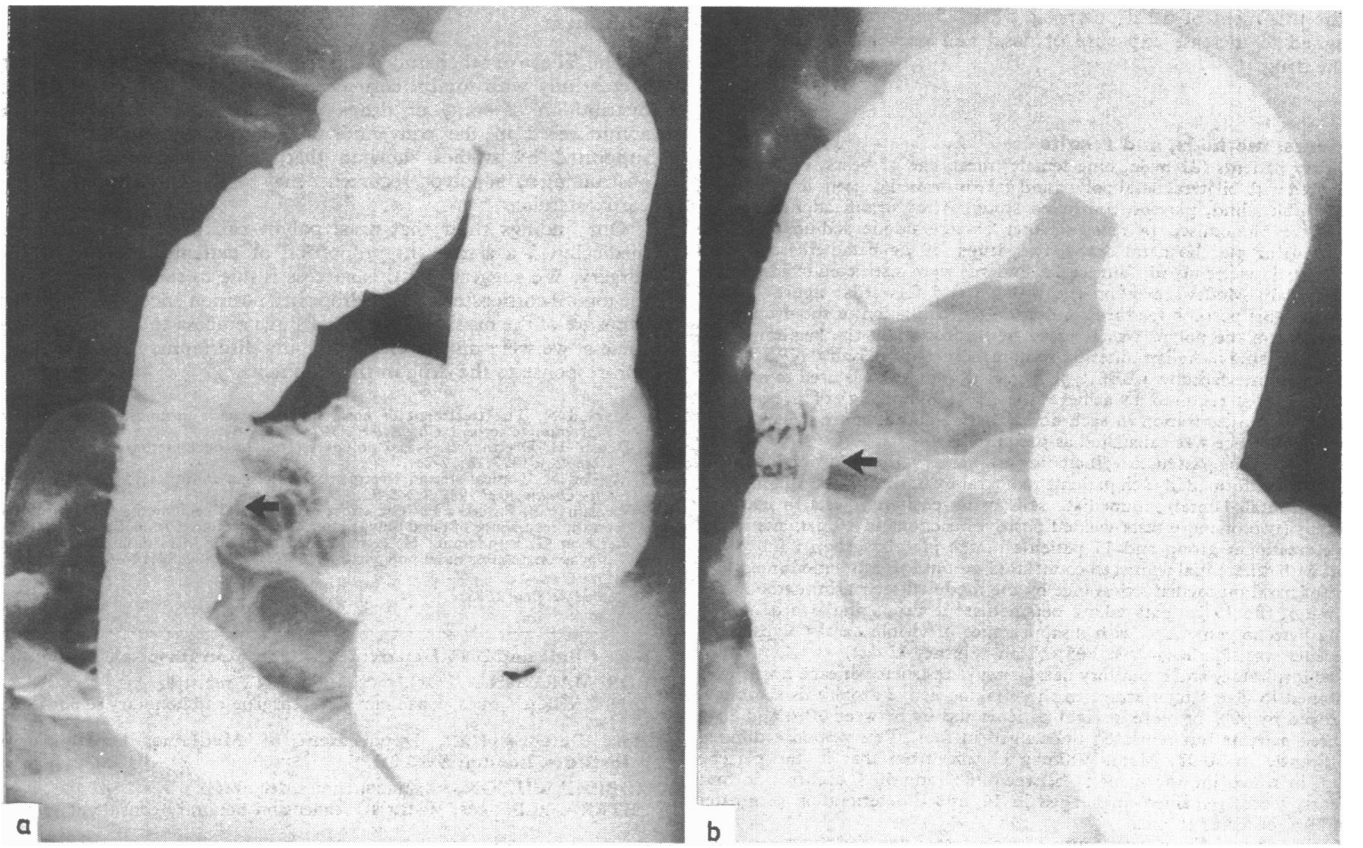
The median whole gut transit time of 50 faecal markers contained in a standard test meal[4] was 47·3 hours at rest and 44·5 hours after a 32 kilometre run. All markers were passed by 75·5 and 82·0 hours, respectively, and total weights of stools were 824·6 and 882·5 g, respectively.

His symptoms were controlled by not eating food on days of races and taking loperamide one hour before running.

Comment

The cause of gastrointestinal symptoms during running is unknown, but excessive parasympathetic activity is unlikely to be responsible as it is persistent in the trained athlete while diarrhoea is intermittent. Severe symptoms usually occur after a rapid increase in the amount of training or particularly severe exertion[2] when it is postulated that hypovolaemia and splanchnic vasoconstriction lead to ischaemia of the gut. This in turn may produce bloody diarrhoea and symptoms that mimic acute appendicitis or Crohn's disease.[3] Hormonal or vascular changes might produce diarrhoea by speeding intestinal transit or increasing intestinal secretion, but neither mechanism occurred in our subject as transit time and weights of stools were similar at rest and after exercise. Similarly, effects of stress during competition would not account for symptoms during training.

The gross psoas muscle hypertrophy in our patient led to obvious colonic compression during flexion of his hip, and this continuous mechanical message may have produced diarrhoea by emptying the colonic contents without increasing total transit or intestinal secretion. Interestingly, his symptoms were reduced by decreasing his intake of food, were worse during road running when mechanical jarring



Radiographs showing compression of ascending colon (arrow) during flexion of right hip—(*a*) extension, (*b*) flexion.

would be greater than on the softer surface of fells, and were absent during cycling when passive contraction of the psoas would produce less colonic compression. This mechanism may be similar to the repetitive mechanical trauma described to explain contusion of the bladder and haematuria in runners.[5]

"Jogger's trots" is a further example of the many hazards that face marathon runners. Although our patient's symptoms may be controlled with drugs, this seems contrary to the principle of guidelines for the control of abuse of drugs in sport.

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Double blind, placebo controlled trial of betamethasone nasal drops for nasal polyposis

The aetiology and pathogenesis of nasal polyps are poorly understood. Studies have shown no effect of topical corticosteroids on nasal polyps, although concomitant sneezing and hypersecretion of mucus were reduced.[1-3] We have investigated the efficacy of betamethasone drops in the treatment of nasal polyposis when administered in a position designed to increase exposure of nasal and paranasal sinus mucosa to the drug.

Patients, methods, and results

Thirty patients (21 male, nine female; mean age 42 years, range 14-66) presented with bilateral nasal polyps and gave informed consent to participate in a double blind, placebo controlled study. After stratification for atopy they were randomised to receive either betamethasone sodium phospate (Betnesol) or placebo nasal drops, two drops (50 μg betamethasone) into each nostril twice daily for four weeks. Patients were instructed to administer the drops in Moffat's position (head down and forwards; figure) and to remain in this position for three minutes after instillation of the drops.

The size of the polyps was assessed by rhinoscopy at the beginning and end of the study. Active anterior rhinomanometry (mercury NRS rhinomanometer interfaced to a BBC type B microcomputer) was used to measure the flow (ml/s) required to achieve 150 Pa (1·1 mm Hg) of pressure on inspiration and expiration in each nostril before and after treatment. Nasal airways resistance was calculated as pressure/flow.

After recording past history, including any surgery, the following investigations were performed in each patient: radiography of sinuses (occipitomental, occipitofrontal, lateral); immediate skin hypersensitivity tests to common allergens (two or more tests yielded positive reactions in 10 patients in the active treatment group and 11 patients in the placebo group); full blood count with differential white cell count; total serum IgE estimation; measurement of nasal mucociliary clearance by the modified saccharin method.

Nine of the 15 patients taking betamethasone drops and two of the 15 taking placebo responded with disappearance of visible nasal polyps. This difference was significant ($p < 0{\cdot}05$; Mann-Whitney U test).

The inspiratory and expiratory nasal airways resistance of each nostril was combined to give a total score. In those taking active drops this score was improved by 50% or more in eight patients and by between 20% and 50% in three patients but remained unchanged in four. This response differed significantly ($p < 0{\cdot}02$; Mann-Whitney U test) from that in the placebo group, in which improvement of between 20% and 50% occurred in four patients, scores remained unchanged in 10, and a deterioration of greater than 50% occurred in one.

No significant difference was found between responders and non-responders in respect of the other variables measured at the beginning of the study.

Head down and forwards position.

Comment

The failure of other studies to show that nasal polyps can be treated successfully with topical corticosteroids may be related to the poor distribution of spray or drops within the obstructed nose when administered in the conventional "head back" position. This is supported by studies showing that, after surgery to clear such obstructing nasal polyps, recurrence may be prevented by using topical corticosteroids.[4 5]

Our findings show that nasal polyps can be successfully treated medically in a significant proportion of patients, thereby avoiding surgery. We suggest that this success is due to the administration of the topical corticosteroid nose drops in a position encouraging maximal exposure of the nasal and paranasal sinus mucosa to the drug. Nevertheless, we were unable to identify any discriminant for response or non-response to the drug in these patients.

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