Discussion

Primary osteoarthritis was found in only 4% of the athletes, but in 8.7% of the controls. According to Danielsson 3.4% of persons over 55 have clear osteoarthritis,¹ and as many as 30% in Hermodsson's study.² So competitive sports cannot be considered as a factor that would make osteoarthritis more likely. Nor did any of the runners have changes indicating epiphyseolysis, which Murray and Duncan found in athletes.³ Those authors considered that growth disturbances due to stress on the proximal femoral epiphyses, comparable to minimal degrees of epiphysiolysis, were incurred in the early teens.

The aetiology of osteoarthritis remains a mystery, though many theories have been put forward, but our study excludes the possibility that the physical strain to which runner's hips are exposed would contribute to its development. Anatomically and physiologically it is not strange that the hips of competitive runners should be spared osteoarthritis, for the hip is designed for walking and running,⁴ and motion is necessary for the nutrition of the cartilage. By intermittent compression and release, the synovial fluid enters and leaves the cartilage very much in the same way as air enters and leaves the lungs under alternating pressures. On the other hand, both prolonged immobilization and continuous pressure for only six days across the living, cartilage-covered joint surfaces produces ulceration and destruction of the cartilage.⁵ Normally the femoral head can be loaded with up to 15 times body weight before the femoral neck is fractured.⁴ Wolff's law about the adaptation of structure to function may be applied to the hip joint as well as to other tissues of the body. The hip joint is made for movement, and there is no reason to worry about injuries even if it has to sustain a heavy daily load.

¹ Danielsson, L., *Clinical Orthopaedics*, 1966, 45, 67.

² Hermodsson, I., *Acta Radiologica Supplement*, 1947, 66, 5.

³ Murray, R. O., and Duncan, C., *Journal of Bone and Joint Surgery B*, 1971, 53, 406.

⁴ Ryder, N., *Clinical Orthopaedics*, 1973, 92, 6.

⁵ Salter, R. B., and Field, P., *Journal of Bone and Joint Surgery A*, 1960, 42, 31.

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Urinary Retention and Intestinal Obstruction associated with Ano-rectal *Herpes simplex* Virus Infection

We describe here two unusual cases of acute urinary retention and intestinal obstruction associated with *Herpes simplex* virus (H.S.V.) infection of the ano-rectal area.

Case 1

A 24-year-old male homosexual was first seen in March 1974 with a one-year history of persistent non-specific urethritis, treated elsewhere. At this stage, H.S.V. (untyped) was isolated from his urethra. The H.S.V. complement fixation test (C.F.T.) titre was <1:10. Six months later he complained of anal pain and constipation of several days' duration, inability to pass urine for 24 hours, and severe colicky lower abdominal pain. There were numerous tender ulcers in the ano-rectal area which were negative for *Treponema pallidum*. He had practised passive anal intercourse for the first time nine days before. Proctoscopy showed semi-solid faeces and severe proctitis. The bladder was palpable, gut sounds were increased, and a plain radiograph of the abdomen was compatible with large bowel obstruction (fig.). Gram-stained smears of rectal exudate showed many polymorphs but no gonococci. H.S.V. type 2 was isolated (human embryonic lung cells) from both rectal exudate and anal ulcers.

After admission his colicky abdominal pain was relieved by defaecation after glycerine suppositories. He later passed urine spontaneously. He was discharged six days later, while the anal ulcers healed in two weeks. The proctitis persisted for eight weeks, however, throughout which H.S.V. type

Erect plain radiograph of abdomen showing fluid levels.

2 was repeatedly grown from the rectal exudate, though not afterwards. The H.S.V. C.F.T. titre was still <1:10 on admission, but rose to 1:320 eight weeks later. Rectal cultures for the gonococcus and *Chlamydia* agent, and serological tests for syphilis, were negative.

Case 2

A 26-year-old male homosexual attended in November 1973 with painful perianal ulceration of a few days' duration. Anal intercourse had taken place 10 days before. The next day he had colicky lower abdominal pain and could not urinate. He had been constipated for seven days. On examination the bladder was palpable to the umbilicus. Numerous tender ulcers were present at the anal margin. These were negative for *T. pallidum*. Proctoscopy showed a diffuse proctitis and a large amount of semi-solid faeces. Many polymorphs, but no gonococci, were seen in a Gram-stained smear of rectal exudate. He was admitted, and passed 750 ml of urine after a hot bath. He defaecated after applying 1% lignocaine to the anal area. The results of cystourethroscopy and urography were normal. On discharge, two weeks later, the proctitis had resolved and bladder function was normal. Swabs taken from the ano-rectal ulcers grew H.S.V. type 1 (no swab for H.S.V. was taken from the rectal mucosa). Rectal cultures for the gonococcus, and serological tests for syphilis, were negative. The C.F.T. titre for H.S.V. on admission was 1:20, and on discharge 1:40.

Comment

Acute urinary retention and constipation may have resulted from reflex spasm of anal and vesical sphincters due to severe anal pain. Both patients had proctitis and in case 1 H.S.V. was isolated from the inflamed rectal mucosa. One possibility is that the proctitis resulted from H.S.V. introduced during anal intercourse, with perhaps trauma facilitating viral entry.

Since case 1 had proved urethral H.S.V. infection several months before the ano-rectal involvement, the latter may have resulted in another way. It is thought that between overt attacks of herpetic infection the virus persists in the ganglion cells of the correspondingly infected dermatome, as in sacral ganglia.¹ A possible mechanism for the ano-rectal lesions in case 1 might, therefore, be a primary urethral infection; persistence of the virus in sacral ganglia; and the trauma of ano-rectal intercourse then reactivating the virus, this leading to herpetic anal ulcers and rectal inflammation.

Cultures for H.S.V. should be taken from all cases of proctitis of uncertain aetiology, especially where anal intercourse is admitted.

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¹ Baringer, J. R., *New England Journal of Medicine*, 1974, 291, 828.

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