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The haematuria of the long-distance runner

One rare complication of strenuous muscular exertion and heat exhaustion is acute renal failure.[1] The kidneys are damaged by a combination of factors including hypotension, dehydration, high temperature, hypokalaemia, rhabdomyolysis, myoglobinuria, and hyperuricaemia.

For many years strenuous exertion has been known to lead to transient abnormalities in the urine, including microscopic and (less commonly) macroscopic haematuria, proteinuria, and cylindruria. In 1907 Collier reported proteinuria after strenuous running or rowing.[2] Gardner[3] found that after matches a high proportion of American football players had abnormalities in the urinary sediment, but that these disappeared rapidly, usually within 24 hours with rest. Because of the similarity of the urinary changes to those found in glomerulonephritis, Gardner coined the term "athletic pseudonephritis."

The presence of casts and in particular of red blood cell casts points to the renal parenchyma as the source of the red blood cells. Haematuria may occur in contact sports such as boxing[4] and American football,[5] where one explanation may be repeated renal trauma. Nevertheless, Alyea and Parish[6] found a high incidence of proteinuria, haematuria, and cylindruria in non-contact sports such as rowing, swimming, and track running. They also showed that the duration of the event was a major determinant of the transient urinary abnormalities. Renal vasoconstriction is known to occur as part of a general splanchnic vasoconstriction during severe exercise[7]; other factors that may be relevant include raised renal venous pressure[8] and increased fibrinolytic activity of the blood.

Marathon runners have been particularly well studied in recent years and myoglobinuria, rhabdomyolysis, proteinuria, and haematuria have been reported in a high proportion of them.[7 9 10] Nevertheless, while Siegel *et al*[10] reported the expected high incidence of haematuria, they did not find any casts—and in particular no red blood cell casts. They suggest that the appearance of casts after running should not be attributed to exertion but to underlying renal disease until proved otherwise.

Another mechanism sometimes responsible for microscopic or macroscopic haematuria after long-distance running is bleeding from the neck of the bladder or the posterior urethra.[11] Blacklock[12] has described a characteristic cystoscopic appearance with ecchymoses or frank contusions in the region of the interureteric bar, the posterior half of the rim of the internal meatus, and the posterior bladder wall. He postulated that the mechanism of "10 000 metres haematuria" may be repeated impaction of the flaccid posterior wall of the bladder against the base of the bladder during running. The first urine voided after a long-distance run often contained frank blood, sometimes with clots; and occasionally there was associated suprapubic discomfort and pain at the tip of the penis.

Whether the haematuria occurring after severe exercise is derived from the kidney or from the bladder, the abnormalities resolve rapidly, usually over 24-48 hours. If such episodes recur, however, the doctor should take steps to exclude any serious underlying abnormality of either the kidneys or the lower urinary tract.

Exercise may unmask underlying disorders, such as idiopathic paroxysmal myoglobinuria,[13] or a susceptibility to march haemoglobinuria[14]—which is thought nowadays to be due to intravascular haemolysis from repeated trauma to circulating red blood cells in the soles of the feet. Symptoms may be alleviated if the runner wears thick-soled shoes. Myoglobinuria and haemoglobinuria (separately or together) have also been reported after prolonged conga drumming[15] and karate.[16]

Unless he is fanatical the average jogger is unlikely to encounter any of these renal abnormalities, and the doctor should assume that any haematuria (whether microscopic or macroscopic), proteinuria, or cylindruria is due to underlying urinary tract disease until proved otherwise.

1 *British Medical Journal*, 1979, **1,** 1233.
2 Collier, W, *British Medical Journal*, 1907, **1,** 4.
3 Gardner, K D, *Journal of the American Medical Association*, 1956, **161,** 1613.
4 Kleiman, A H, *Journal of the American Medical Association*, 1958, **168,** 1633.
5 Boone, A W, Haltiwanger, E, and Chambers, R L, *Journal of the American Medical Association*, 1955, **158,** 1516.
6 Alyea, E P, and Parish, H H, *Journal of the American Medical Association*, 1958, **167,** 807.
7 Dancaster, C P, and Whereat, S J, *South African Medical Journal*, 1971, **45,** 547.
8 Bruce, P T, *British Journal of Urology*, 1972, **44,** 724.
9 Schiff, H B, MacSearraigh, E T M, and Kallmeyer, J C, *Quarterly Journal of Medicine*, 1978, **NS 47,** 463.
10 Siegel, A J, *et al*, *Journal of the American Medical Association*, 1979, **241,** 391.
11 Fred, H L, and Natelson, E A, *Southern Medical Journal*, 1977, **70,** 1394.
12 Blacklock, N J, *British Journal of Urology*, 1977, **49,** 129.
13 Wheby, M S, and Miller, H S, *American Journal of Medicine*, 1960, **29,** 599.
14 Spicer, A J, *British Medical Journal*, 1970, **1,** 155.
15 Furie, B, and Penn, A S, *Annals of Internal Medicine*, 1974, **80,** 727.
16 Russell, S M, and Lewis, A, *New England Journal of Medicine*, 1975, **293,** 941.