Letters

Muscle hypoxia in polymyositis

Sir: The pathogenesis of polymyositis is unknown, but there is circumstantial evidence that pathological changes in the intramuscular blood vessels may play a role. Ultrastructural abnormalities have been found in the endothelium of the microvessels, and capillary basement laminae have been reported to be thickened. Capillaries are lost in polymyositis, particularly in the dermatomyositis subtype.¹

These changes, possibly triggered by immune complex deposits in the vessel walls,² may be responsible for the reported decline in muscle blood flow in polymyositis,³ but it is not known whether they are able to render the muscle ischaemic enough to contribute to the necrotising damage in the muscle tissue.

We measured tissue gas tensions in five adults (four women, one man, age 42-67 years) with 2-7 years history of definite polymyositis as diagnosed by clinical, neurophysiological and muscle biopsy examinations. One of them had skin symptoms suggesting dermatomyositis. All of these patients were ambulatory, and were at a chronic stage of disease with only minimal clinical symptoms (one further patient—with a low resting PO_2 is not included in the results, as her exercise test had to be interrupted because of fatigue). Five healthy volunteers (two women, three men, age 42-68 years) served as controls. The measurements were carried out by means of an implanted Silastic tonometer.^{4,5} Under local anaesthesia, a 16cm long silicone elastomer tube with an external diameter of 1.4mm and an internal diameter of 1.0mm was inserted into the medial belly of the gastrocnemius muscle. Care was taken to insert the tonometer at the same depth in the patients and controls. The recordings were made four days after implantation. The tube was slowly perfused with hypoxic saline, and the PO_2 and PCO_2 of the efflux were continuously monitored both at rest and during a heel lifting exercise.⁵

As shown in the figure, the calf muscle PO_2 levels were consistently lower in the polymyositis patients than in the controls. The differences are statistically significant both at rest and during exercise ($p = 0.004$, Mann-Whitney U test). The lowest values were found in the dermatomyositis patient. PCO_2 , on the other hand, behaved similarly in the two groups. The response of PO_2 to exercise in the polymyositis patients was

different from that in patients with occlusive arterial disease: in the latter condition the PO_2 is close to normal at rest but decreases during exercise.⁵ It seems, therefore, that in polymyositis the larger vessels are patent and they are able to respond to the needs of autoregulation. The finding of low average PO_2 in polymyositis patients both at rest and during exercise suggests that the capillaries are unable to maintain normal tissue oxygenation. Thus, the behaviour of PO_2 in polymyositis is consistent with the hypothesis that circulation is hampered at the level of small peripheral vessels.

It may be asked whether the disability and inactivity of the polymyositis patients causes diminished blood flow and drop of temperature in the muscle tissue with resultant unspecific changes in tissue gas tensions. The patients, however, were ambulatory and had only minimal weakness at the time of the study. Furthermore, low tissue temperature would enhance the solubility of O_2 in the extracellular fluid, and higher (not lower) PO_2 would be found in measurements carried out at the standard temperature of 37°C.

The finding of marked relative muscle hypoxia in idiopathic inflammatory myopathy suggests that microvascular obstruction plays a role in its pathogenesis. The result may also have therapeutic implications.

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Fig Calf muscle PO_2 in polymyositis patients and normal subjects during rest and heel lifting exercise.

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References

- Whitaker JN. Inflammatory myopathy: a review of etiologic and pathogenetic factors. *Muscle Nerve* 1982;5:573-92.
- Whitaker JN, Engel WK. Vascular deposits of immunoglobulin and complement in idiopathic inflammatory myopathy. *N Engl J Med* 1972;286:333-8.
- Paulson OB, Engel AG, Gomez MR. Muscle blood flow in Duchenne type muscular dystrophy, limb-girdle dystrophy, polymyositis, and in normal controls. *J Neurol Neurosurg Psychiatry* 1974;37:685-90.
- Niinikoski J, Hunt TK. Measurement of wound oxygen with implanted Silastic tube. *Surgery* 1972;71:22-6.
- Jussila EJ, Niinikoski J. Effect of vascular reconstructions on tissue gas tensions in calf muscles of patients with occlusive arterial disease. *Ann Chir Gyn* 1981;70:56-60.

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Dilated tonic pupils in neurosyphilis

Sir: In patients with central nervous system syphilis, pupillary abnormalities are a relatively common finding, and the most important early ocular sign.¹ The following case is presented as an example of a previously noted² but not widely recognised association between bilaterally dilated tonic pupils, fixed to light and accommodation, and neurosyphilis.

A 48-year-old male presented with a 4 to 5 year history of mild photophobia. There was no history of diabetes, hypertension, trauma, headaches, fever, chills, nausea, vomiting, seizures, weakness, pain, or use of medications or illicit drugs. He claimed no alcohol use for at least four months but admitted to previous heavy ethanol abuse. He stated that he had occasional diplopia and intermittent urinary incontinence. At age 26 years, the patient presented with a penile chancre and positive serum VDRL, and was treated with a series of intra-