Streptokinase and serum enzyme activity in myocardial infarction

In a controlled study of fibrinolytic treatment of unstable angina, Shepherd *et al*¹ observed rises in serum aspartate and alanine aminotransferase (SGOT and SGPT) activity in eight out of 20 patients. They suggested the liver as the likely source of the increases. I have compared the activity of these two enzymes in patients with myocardial infarction who had been receiving streptokinase in almost identical dosage with that used by Shepherd *et al*.

Patients, methods, and results

During a controlled study of fibrinolytic treatment of acute myocardial infarction 534 measurements of SGOT and SGPT were made in 94 patients who had been treated with streptokinase (Streptase). SGOT and SGPT were estimated with the use of Boehringer reagents (upper limit of normal for both enzymes in our laboratory 18 IU/l). The streptokinase was given intravenously in a dosage of 250 000 units initially, then 100 000 units hourly for 18 hours.

There was only a slight increase in SGPT compared with a pronounced increase in SGOT (figure). Further statistical evaluation of SGPT activity was not undertaken because of these findings. In the study by Shepherd *et al* the maximum increase in enzyme activity was definitely delayed.

SGOT and SGPT activity after myocardial infarction in patients receiving streptokinase. Top: Dashed line represents patients beginning treatment within four hours after onset of symptoms. Solid line represents patients beginning treatment 4-12 hours after onset of symptoms. Bottom: Open circles indicate mean values.

Comment

These results differ from those of Shepherd *et al*,¹ but their patients had unstable angina, and mine acute myocardial infarction.

Several workers² have described haemorrhages in areas revascularised by coronary artery surgery. Deterioration of myocardial function also almost invariably occurs after surgical revascularisation.³ Both conditions may cause the increases in SGOT and SGPT as found by Shepherd *et al*.¹ It would be of great interest if it could be confirmed by coronary angiography that the patients with an increase in enzyme activity described by Shepherd *et al* (eight out of 20)¹ were those with an effective coronary revascularisation.

Investigations using enzyme kinetic methods⁴ or mathematical statistical model calculations⁴ have shown that the enzyme method for determining infarct size is not applicable if any treatment results

in improvement or deterioration of myocardial perfusion. On the basis of our own results we have discussed the hypothesis that fibrinolytic treatment may improve myocardial perfusion, which was indicated by an earlier and steeper rise in SGOT and creatine phosphokinase (as a "wash-out" phenomenon).⁵

Another question that remains unanswered is whether streptokinase-induced alteration of peripheral vascular resistance and blood viscosity produces a characteristic pattern of serum enzyme changes in patients with unstable angina (in the absence of myocardial infarction). As Shepherd *et al*¹ have produced only an initial report and the results of their controlled trial have not yet been published, any detailed interpretation of the enzyme values (hepatic source, for example) would be premature.

¹ Shepherd, J T, *et al*, *British Medical Journal*, 1976, **1**, 562.

² Roberts, R, and Sobel, B E, *Circulation*, 1974, **50**, 867.

³ Shepherd, R L, *et al*, *Circulation*, 1974, **49**, 467.

⁴ Roe, C R, and Starmer, C F, *Circulation*, 1975, **52**, 1.

⁵ Praetorius, F, *et al*, *Klinische Wochenschrift*, 1973, **51**, 397.

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Myeloneuropathy associated with hypothyroidism

Polyneuropathy is a rare complication of hypothyroidism.¹⁻⁴ We here report a patient who presented with a myeloneuropathy that improved on treatment with thyroxine.

Case report

A 61-year-old man gave a two-year history of progressive dragging of his feet, weakness of his hands, and paraesthesiae in his fingers and toes. His skin was pale and dry. There was distal weakness and wasting of the calf, peroneal, and intrinsic hand muscles; sensation was impaired in a glove-and-stocking pattern. The deep tendon reflexes were reduced in the arms, absent at the ankles, and brisk at the knees; the plantar responses were extensor. Investigations confirmed that he was hypothyroid: serum thyroxine was 25 nmol/l (2 µg/100 ml); thyrotrophin 100 mU/l; radioiodine uptake 12% at 24 hours; creatinine phosphokinase 417 IU/l; and CSF protein 0.75 g/l. Electromyography showed denervation, and the nerve conduction velocities were reduced (ulnar nerve, 45.6 m/s; lateral popliteal nerve 26.2 m/s). No sensory potential could be evoked with 200 volts.

(a) Transverse section of sural nerve showing endoneurial and perineurial fibrosis and reduction in myelinated fibres. (Solochrome cyanin $\times 116$.)

(b) Section of gastrocnemius muscle showing group fibre atrophy and fibres with internal nuclei. (H & E $\times 116$.)