Correspondence

Fibrinolytic defect in chronic back pain

To the Editor:

We are very pleased to note that Hurri and colleagues (1991) have confirmed our observation of defective fibrinolytic activity in patients with chronic back pain (Jayson et al. 1984, Pountain et al. 1987). They demonstrated this by a significant reduction in tissue plasminogen activator activity (TPA.Ac). In addition, they found an increase in tissue plasminogen activator antigen (TPA.Ag), but no change was found in plasminogen activator inhibitor (PAI) when compared with their controls. The latter result is extremely surprising, because an increase in TPA.Ag with normal levels of PAI should lead to an increase in fibrinolytic activity.

In our own recent observations (Cooper et al. 1991), we similarly demonstrated reduced TPA.Ac and increased TPA.Ag as compared with our controls, but also there were elevated levels of PAI. The increase in inhibitor activity exceeds the increase in antigen, with an overall decrease in fibrinolytic activity. This is the pattern of changes found in many disorders in which fibrinolytic defects occur. We therefore question the measurements made by Hurri et al. (1991) of normal PAI levels.

It occurs to us that the venesection to obtain blood for these assessments may have been performed after myelography, as described in their paper. The process of myelography may act as a stimulus to fibrinolytic activity, and this might be the explanation for the lack of a difference in PAI levels in the patients compared with the controls, who presumably did not undergo myelography.

The authors rested the patients and controls before the venesection, but they do not state whether or not the subjects were fasted and refrained from smoking. These are essential preliminaries for accurate assessments of fibrinolytic activity; and if blood specimens were not obtained under these conditions, accurate results cannot be obtained. This also might explain the lack of a difference in PAI levels.

References

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Malcolm I. V. Jayson, M.D.
Professor of Rheumatology
University of Manchester, Rheumatic Diseases Centre, Hope Hospital
Eccles Old Road, Salford M6 8HD, England

To the Editor:

Professor Jayson is striking at a very important question concerning our previous study. In fact, we also expected an excess of PAI in the patient series; and for the lack of an expected difference, we could not find any specific explanation. The blood samples were collected before myelography so it could not act as a stimulus of fibrinolytic activity in our study. The patients were fasted and refrained from smoking 12 hours before blood sampling.

We have vigorously tried to find an explanation for the lack of difference in PAI concentrations between the two groups. Frankly, we have not succeeded; and quite obviously, new patient series must be studied for a final clarification of this very pertinent question.

Heikki Hurri, M.D.
The Invalid Foundation, Orthopedic Hospital, Tenholantie 10, SF-00280 Helsinki, Finland