compensation claim had been made and was considered justified'.¹¹

The other paper, by Browne *et al.*, cited by Dr Quintner was an essay in which the authors expressed their views.¹² No patient numbers were reported.

Dr Quintner may consider my emphasis on the importance of the clinical examination a 'truism', but it is nevertheless one that bears repeating if only to avoid the temptation to rationalise the absence of clinical abnormalities by resorting to untenable hypotheses of neuropathic pain to explain this phenomenon. One can only be grateful that all of that neuropathic pain which afflicted so many Australian workers up until the mid 1980s has seen fit to resolve, apparently spontaneously.

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LETTER TO THE EDITOR

Activated protein C resistance caused by factor V Arg 506→Gln mutation has no role in thrombotic manifestations of Behçet's disease

Vascular involvement is found in approximately 25% of patients with Behçet's disease and includes venous occlusions (88%), arterial aneurysms, or arterial occlusions (12%),¹ but the mechanism of thrombosis remains unexplained. Recently, a genetically determined defect in anticoagulation characterised by resistance to activated protein C (APC) was frequently found in patients with venous thromboembolism.² APC resistance is highly linked to a single factor V gene mutation, Arg 506→Gln.³

To see whether APC resistance could explain the thrombotic events observed in Behçet's disease we looked for the factor V gene mutation in 15 unrelated patients suffering from the condition. The patients

were 11 men and four women (mean age 39 years (range 15-58)). All fulfilled the International Study Group criteria for Behçet's disease.⁴ Superficial thrombophlebitis and retinal occlusions were not considered. All patients had a history of thrombosis, affecting veins in 12 and arteries in six. Recurrent vascular events were noted in nine patients: venous followed by venous in five, venous then arterial in three, and arterial-arterial in one. The factor V gene mutation was identified using the polymerase chain reaction and denaturing gradient gel electrophoresis, as described previously.⁵ The plasma APC resistance test could not be performed because the majority of patients were receiving anticoagulants at the time of the study. No patient had the Arg 506→Gln mutation.

We conclude that APC resistance linked to the Arg 506→Gln mutation is not a cause of thrombotic manifestations in Behçet's disease.

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