

pontine trigeminal pathways was present in six versus six patients. Vascular compression was severe in two versus seven and steroids at onset of trigeminal neuralgia were given to one versus six patients. Preoperative immunosuppressant therapy was administered in one versus four patients (table). Variables were compared with two-sample *t* test or Fisher's exact test, as appropriate. No significant prognostic factor was found. There was no mortality or long-term morbidity.

The high incidence of vascular compression at trigeminal-root entry zone suggests a causal relation rather than a chance association, whereas the 40% of failures of microvascular decompression in our series questions the possible causative role of vascular compression in patients with MS and trigeminal neuralgia. The causal role of vascular compression is debated even in trigeminal neuralgia and a simple cause-effect mechanism has not yet been established. A more complex balance between the excitability threshold of neurons of the trigeminal nucleus, and possible pathological inputs from the compressed trigeminal root of entry zone might be considered. In patients with MS and trigeminal neuralgia, with vascular compression at operation, a dual cause could be hypothesised; the action of vascular compression might be facilitated by the underlying hyperexcitability of trigeminal pathways due to evolving demyelination. This might explain not only the higher incidence of trigeminal neuralgia in patients with MS, but also the worse results obtained by both microvascular decompression and percutaneous procedures<sup>4</sup> in patients with MS and trigeminal neuralgia than in the series of essential trigeminal neuralgia. A direct relation between post-operative sensory deficits and the cure rate<sup>3</sup> still limits percutaneous methods in patients with MS. Disease progression is likely to cause neurological deficits and recurrences of trigeminal pain; the possibility of obtaining long-term relief of trigeminal neuralgia without damaging the nerve should not be denied to patients with MS. An ad-hoc MRI protocol<sup>5</sup> should be used to select MS patients with MS and trigeminal neuralgia as surgical candidates.

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## Treatment of traumatic bleeding with recombinant factor VIIa

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Surgical intervention failed to stop life-threatening bleeding caused by injury complicated by severe coagulopathy. Administration of recombinant factor VIIa immediately corrected the coagulopathy and bleeding stopped.

A 19-year-old soldier was admitted with a high-velocity rifle injury. The bullet tore the inferior vena cava at L5 level causing extensive damage to paravertebral muscles at the exit wound. He was admitted in a critical condition with profound hypovolaemic shock, ketoacidosis, hypothermia, and disseminated intravascular coagulation. Ligation of the inferior vena cava was done immediately, yet surgical attempts to achieve haemostasis (including repeated packing) and attempts to correct the ketoacidosis and hypothermia failed to control bleeding. He received 5 L packed cells, 3 L fresh frozen plasma, 20 units of platelets, and 10 units of cryoprecipitate with tranexamic acid. He continued to bleed at a rate of 300 mL/min and a fatal outcome seemed inevitable.

In a desperate attempt to control the bleeding, 60 µg/kg of recombinant factor VIIa were given intravenously. 10 min after injection, coagulation tests improved markedly, the rate of bleeding decreased to 10-15 mL/min but slow oozing from all wound surfaces continued. After 1 h a repeat dose of recombinant factor VIIa (60 µg/kg) was given. The oozing stopped immediately with a return to normal of coagulation tests, enabling surgeons to identify and ligate some small vessel tears. Fibrin sealant was sprayed over the area to prevent rebleeding. The patient's condition remained stable, and he had no further blood loss.

Recombinant factor VIIa is an effective treatment for patients with haemophilia with inhibitors.<sup>1</sup> Its mode of action is not completely clear. Some studies suggest tissue-factor-dependent mechanisms, whereas others emphasise the role of factors Xa and IXa on the surface of activated platelets. These studies relate thrombin generation on activated platelets to the high level of recombinant factor VIIa binding to platelet surfaces.<sup>2</sup> Therapeutic doses of recombinant factor VIIa are not established; different doses have been used during surgery in patients with haemophilia and inhibitors.<sup>3</sup> The use of recombinant factor VIIa has been reported to control bleeding in patients with thrombocytopathies,<sup>4</sup> liver disease, liver transplantation, and patients undergoing cardiac surgery.<sup>5</sup> Due to its thrombogenic potential, recombinant factor VIIa has been contraindicated for use in patients with severe disseminated intravascular coagulation or crush injuries. The successful use of recombinant factor VIIa in Dengue fever with bleeding<sup>5</sup> and in the case we report suggests that this contraindication should be re-evaluated.

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