dura mater occur even less commonly and when described, have usually been reported as incidental findings at necropsy,

Ligumski et al9 reviewed a series of seven post-mortem examinations of patients in whom deposits of extramedullary haemopoiesis were present in the central nervous system, and who had developed neurological manifestations prior to death. Four of these showed tumour masses in the dura mater which varied in size up to 5 cm in diameter, two showed involvement of the leptomeninges only and the remaining case showed a single focus in the right frontoparietal lobe. The clinical manifestations in these patients were diverse and included headaches, vasomotor reactions, episodes of unconsciousness and coma. Lund et al9 have reported the CT findings in a case of symptomatic intracranial haemopoiesis occurring secondarily to myelofibrosis. CT was performed in our patient 2 weeks prior to death, but no dural tumours were seen. Presumably the deposits of haemopoietic tissue present at that time were too tiny to be seen, but subsequently, like the nodules in the skin, grew very rapidly, reflecting the accelerated deterioration of the patient’s disease.

The reasons for the exacerbation of his myelofibrosis, with the development of widespread foci of extramedullary haemopoiesis in many unusual sites, are not clear. Splenectomy may be implicated, though most studies have shown splenectomy to result in a modest improvement in haematological status, complications being related mostly to the immediate post-operative period. Whatever the reason, this patient showed sudden rapid deterioration of his bone marrow disease, with the subsequent deposition of extramedullary haemopoietic tissue in the brain and cranial dura mater, resulting in seizures, status epilepticus and death.

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Neuralgic amyotrophy due to parvovirus infection

Sir: Neuralgic amyotrophy is commonly associated with unspecified viral illnesses. We describe here an association with parvovirus infection, the first such case reported.

A 26-year old previously fit policeman presented in the winter months five days after the onset of a febrile illness. It was characterised by myalgia, arthralgia, sore throat and mild headache. A fever of 38-5°C had been recorded once. On the fifth day of illness he noticed a fine non-titchy rash on his body. On the same day severe pains in the arms and shoulder girdle developed suddenly. The pain was present at rest and worse on movement. Paracetamol had no effect.

Examination disclosed no fever or abnormalities in the respiratory or cardiovascular systems or abdomen. The faeces were slightly red and there was palpable cervical lymphadenopathy. Over the face and trunk there was a fine maculo-papular rash which extended to the shoulders, upper arms and upper thighs but spared the distal extremities. In places the rash coalesced. There was visible fasciulation of the right deltoid muscle and movement at both shoulders was limited by pain. Over the next two days the rash faded and he was left pain free but weak. Neurological examination on the right revealed very severe weakness of the supra and infraspinatus and deltoid muscles. On the left there was severe weakness of biceps and brachioradialis together with some weakness of infraspinatus. Dorsally there was severe weakness of the left wrist, fingers and thumb extension. No upper limb reflexes were elicitable. There were two areas of sensory deficit coincident with the distribution of the axillary nerve over the right and radial nerve on the left. A clinical diagnosis of neuralgic amyotrophy was made. An EMG showed severe but patchy denervation predominantly in the C5 area and C6 innervated muscles on the right and right arm extensor on the left. The left radial sensory action potential was absent. On the fifth day a parvoviral serology revealed recent infection with parvovirus. The IgM (by RIA) being 4.8 units eleven days after the onset of the febrile illness then falling, and the IgG (also by RIA) rising to more than 100 units twenty nine days after onset.

This patient presents a typical picture of neu ogic amyotrophy, otherwise known as brachial neuritis. The EMG study is consistent with this diagnosis. Neuralgic amyotrophy is an axonal disorder of unknown aetiology. It may follow trauma, vigorous exercise, heroin abuse, immunisation and viral infection, usually unspecified. There have been 15 reports of it occurring after Epstein-Barr virus infection. It has also been reported after herpes zoster infection. This is the first report of neuralgic amyotrophy occurring after parvovirus infection.

Erythema infectiosum or fifth disease, recognised for decades, has recently been linked to a human parvovirus. Our patient manifested the typical clinical features of a low grade fever, rash, myalgia and arthralgia. Asymptomatic infection occurs and 61% of United States blood donors have antibody to the virus. Complications reported are aplastic crises in those with haematological abnormalities, such as sickle cell disease or hereditary spherocytosis and possibly synovitis after
chronic arthritis. The virus divides preferentially in rapidly dividing cells particularly erythrocyte precursors, but by the time the rash appears, viraemia is no longer detectable. This suggests that the neuralgic amyotrophy seen in this patient is due to an idiotyncratic immune response.

We recommend serological testing for parvovirus IgM in cases of neuralgic amyotrophy to ascertain the relative causal contribution to this painful and disabling condition.

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Failure to detect plasma neuropeptide release during trigeminal thermocoagulation

Sir: Calcitonin gene related peptide (CGRP) is a novel 37 amino acid residue peptide which is widely distributed in the human nervous system and has potent pharmacological effects. CGRP-immunoreactive neurons are found in spinal and trigeminal ganglia as well as in their terminations in the spinal cord and brainstem. The peptide is released from rat trigeminal ganglion neurons in tissue culture, it causes vasodilatation of human pial arteries in vitro and when injected into human skin results in a flare-type reaction. Recently CGRP has been detected in plasma. It was therefore of interest to see whether CGRP was released during trigeminal ganglion thermocoagulation which was being carried out for intractable trigeminal neuralgia.

Vasoactive intestinal polypeptide levels were also measured. This peptide is contained in parasympathetic nerve fibres and it has been suggested that trigeminal ganglion stimulation results in the indirect activation of facial nerve parasympathetic fibres. Plasma samples (10 ml) were taken before the onset of thermocoagulation, towards the end of 3 minutes of thermocoagulation and 20 minutes after the end of the procedure. This series of samples was taken from five patients. Each plasma specimen contained 5000 units of the protease inhibitor Trasylol and was cooled in ice prior to radioimmunoassay as previously described. No CGRP was detected in any of the samples assayed. VPP levels were all within the normal plasma range and showed no rise during the procedure (less than 5 pmol/l).

During trigeminal thermocoagulation some patients develop ipsilateral facial flushing which can persist for up to 30 minutes after the procedure. This is thought to be due to the antidromic release of neurotransmitter substances. A similar release of a peptide transmitter, such as substance P, from trigeminal fibres innervating the major cerebral arteries has been proposed as the cause of headache in migraine.

The failure to detect the release of CGRP or VIP into the plasma during trigeminal thermocoagulation may have various explanations. Even if these peptides are released at sensory and parasympathetic terminals respectively, it is possible that they are not released into the circulation in significant amounts. Alternatively plasma degredation may be very rapid. It is known that VIP has a plasma half life of under one minute. However this is unlikely to explain the failure to detect CGRP as this peptide has a plasma half life of 7 minutes.

If neuropeptides really do have a physiological role in man and, in particular, are involved in the mediation of certain pain states, the demonstration of their release in vivo is of great importance. It may be that cerebrospinal fluid or jugular venous blood samples would be more likely to show changes although to obtain such specimens would be harder to justify ethically.

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