other vascular anomalies such as aneurysms, arteriovenous malformation.[1,2] It appears, however, that the incidence of cranial nerve dysfunction induced by PTA is quite rare.

A 62 year old woman noticed the onset of diplopia when she woke up, which was more marked on looking toward the left and associated with left orbital dull pain. The next day she developed a headache in her left temporo-occipital region. The pain gradually improved within one month, but double vision on left lateral gaze persisted. There were no bruits on auscultation of the head, orbits, or carotids. Neurologically, mild hypesthesia in the first division of the left trigeminal nerve and incomplete left abducens nerve palsy were noted. Other cranial nerves were intact and there were no pulsatile exophalmos or chemosis of the bulbar conjunctiva suggestive of a carotid-cavernous fistula.

Angiography showed that the anastomotic artery arose from the superolateral wall of the left internal carotid artery at the C4-5 junction with an infundibular-like aneurysmal dilatation at its origin, coursed posteromedially, and then joined the rostal basilar artery just below the origin of the superior cerebellar artery, filling the BA and the right SCA partially (fig). This artery was opacified on vertebral angiography only in the early arterial phase. The calibre of the BA was small and the left posterior cerebral artery was opacified through the fetal type posterior communicating artery. Using a thin section with high resolution CT cisternography, which was performed to rule out the presence of any tumour as another causative lesion, also disclosed this artery in the posterior fossa just medial to the left trigeminal nerve. The abducens nerve palsy improved gradually with conservative therapy and she was neurologically free eight weeks later. Anatomically and radiologically, we assumed that the aneurysmal dilatation at the origin of the PTA induced the abducens nerve palsy and the hypesthesia in the first division of the trigeminal nerve.

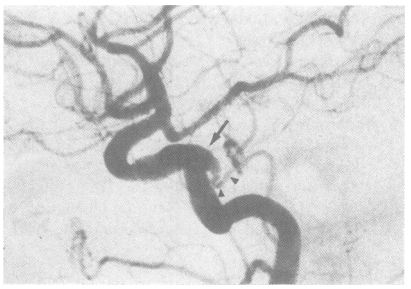

Fig *Lateral view of the left carotid angiography reveals an aneurysmal dilatation at the origin of the PTA in the cavernous portion (arrow). The PTA (arrowheads) arising from the superolateral aspect of the cavernous carotid, coursing posteriorly and then turn medially at the prepontine cistern, join the basilar artery at the superior third of it.*

Anatomical studies suggest that trigeminal neuralgia and abducens nerve palsy might result from anomalous vessels.[3,4] Among 17 PTAs which were considered as a responsible lesion for several cranial nerve dysfunctions, three cases had unruptured aneurysms or aneurysmal dilatation at the origin of PTA.[2-4] Sugar[6] suggested that the origin of the trigeminal artery, where the PTA normally become obliterated in the embryo, could be a weakened point and would be predisposed to rupture. It is, therefore, possible to consider that unruptured aneurysm or aneurysmal dilatation, or even the influence of paroxysmal change in the blood flow on the PTA, could exert pressure on cranial nerves resulting in transitory or chronic dysfunction of cranial nerves.

Persistent primitive arteries must be kept in mind as a causative agent of certain cranial nerve dysfunctions. Spontaneous recovery of cranial nerve disorder due to the vascular compression has occasionally been encountered. In such cases, cranial nerve dysfunction could be caused by the transient increase of the blood flow or the dilatation of the vessels, but the abducens nerve could tolerate the mechanical compression and recover in several weeks. There have been several reports of surgery for aneurysms of the PTA by opening the cavernous sinus[7] or by ligation of the carotid artery.[8] However, there was a risk of injury to the adjacent intact cranial nerves or, especially, of sacrificing blood supply not only to anterior but also to posterior circulation.

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Norpethidine induced myoclonus in a patient with renal failure

Sir: Pethidine (meperidine) is biotransformed by the liver to norpethidine. Accumulation of norpethidine in uraemia may produce an encephalopathy similar to uraemic encephalopathy.[2,3] We report a case of norpethidine induced generalised myoclonic status in a hemodialysis patient, resulting in severe hyperkalaemia, and successfully treated with clonazepam.

A 33 year old man with a six year history of renal failure secondary to mesangio-proliferative glomerulonephritis was admitted to the Royal Perth Hospital in August 1988. The uraemia was managed with thrice weekly home haemodialysis. Renal colic that occurred during dialysis, approximately 48 hours before admission, caused him to stop dialysing prematurely. Pethidine, 100 mg, was administered at a regional hospital and a further 200 mg was administered during the following eight hours. Morphine and prochlorperazine were used thereafter, with total doses of 60 mg and 62·5 mg respectively. Approximately 36 hours after the last dose of pethidine, he showed increasing confusion, agitation and myoclonic jerks. He was then transferred to the Royal Perth Hospital.

Clinical examination revealed a sallow, disorientated man who obeyed verbal commands inconsistently. His temperature was 36·7C, pulse was regular at 100/minute and blood pressure 230/120. There were continuous, spontaneous, arrhythmic, asyn-

chronous myoclonic jerks that affected the face and limbs. Tactile and auditory stimuli produced generalised myoclonic jerks. Fundoscopy and pupillary reactions were normal and oculocephalic, corneal and gag reflexes were present. Muscle tone was normal. Generalised myoclonic jerks were produced when deep tendon reflexes were tested. Both plantar responses were flexor. Formal assessment of muscle power, sensation and coordination was not possible. General examination revealed third and fourth heart sounds and a soft ejection systolic murmur.

Initial biochemical investigation showed: plasma potassium 7·9 mmol/l, bicarbonate 17 mmol/l, urea 47·8 mmol/l, creatinine 1504 μmol/l, glucose 7·5 mmol/l, arterial blood pH 7·24, pCO2 43 mm Hg, pO2 104 mm Hg, oxygen saturation 97%. The haemoglobin level was 97 g/l and the leucocyte count $10·8 \times 10^9$/l. Cardiomegaly was present on a chest radiograph. Three sets of blood cultures and a midstream urine specimen were sterile.

Three hours of haemodialysis did not affect the patient's neurological status. A generalised tonic/clonic convulsion occurred less than 10 minutes after administration of naloxone, 0·2 mg. Recurrent hyperkalaemia (7·5 mmol/l) was treated with oral sodium resonium. Electroencephalography revealed generalised theta and delta activity of moderate to high amplitude, intermixed with multifocal spike and sharp waves at a frequency of 1–2 per second. Intravenous injection of clonazepam, 0·5 mg, resulted in immediate cessation of myoclonic jerks and reduction of spike and sharp activity. Lower amplitude semirhythmic 3–4 Hz activity appeared. Myoclonus and hyperkalaemia did not recur. The pethidine level in the plasma stored from the time of admission was < 10 μg/l and the norpethidine level 114 μg/l. Haemodialysis did not affect the rate of fall of plasma norpethidine levels which were logarithmically related to time (correlation coefficient, $r = -0·82$; $P < 0·05$). The elimination half life of the metabolite in our patient was 25 hours.

The initial metabolism of pethidine occurs in the liver where hydrolysis to pethidinic acid and N-demethylation to norpethidine, followed by hydrolysis to norpethidinic acid, occurs. While repeated administration of pethidine in patients with cancer[1 2] or sickle cell anaemia[4] may result in norpethidine accumulation, more rapid accumulation after fewer doses occurs in renal failure. Norpethidine is a central nervous system excitant with less potent depressant effects than pethidine. The progression of excitatory effects, from nervousness and tremors to agitated delirium, multifocal myoclonus and seizures, in a group of cancer patients treated with pethidine, correlated with the plasma level of norpethidine as well as the ratio of norpethidine to pethidine in the plasma.[2]

It is likely that the myoclonus and generalised convulsions in our patient were related to norpethidine accumulation. Normal muscle tone and marked stimulus sensitivity do not usually occur in a phenothiazine (prochlorperazine) induced syndrome. In our patient, the effect of uraemia on the threshold for central excitation, resulted in lower norpethidine levels producing myoclonus and seizures compared to the levels required in cancer patients (424–1856 μg/l).[2]

In both uraemic and norpethidine related myoclonus, the involvement of not only the distal muscles of the stimulated limb, but also proximal muscles and other limbs in the myoclonic response to peripheral stimuli, suggests that the myoclonus was a reticular rather than a cortical reflex type. The former has been described in uraemia,[5] hyponatraemia and post anoxic encephalopathy.

Because of norpethidine's long half life in uraemic patients, neurological abnormalities may persist for several days after cessation of pethidine. In the presence of renal failure, repeated muscle contraction may result in life threatening hyperkalaemia and urgent control of myoclonus and seizures becomes necessary. Clonazepam was very effective in this case and is also dramatically effective in controlling post-anoxic myoclonus, where diazepam, phenytoin and phenobarbitone have been ineffective.[6] In another reported case[3] of myoclonus due to presumed norpethidine toxicity, clonazepam was ineffective but the route of administration was not recorded. Naloxone, by completely antagonising the depressant effects of pethidine and norpethidine but only partially antagonising the latter's excitatory effects,[7] may unmask seizure activity when used in norpethidine intoxication. From our data, norpethidine appears to be difficult to remove by dialysis.

In uraemic patients requiring narcotic analgesia, agents such as pethidine and dextropropoxyphene with toxic metabolites should be avoided. While morphine and codeine may result in unusually severe and prolonged respiratory depression and sedation in renal failure, their cautious use at lower dosage, with careful clinical monitoring, is to be preferred.

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Intramedullary spinal cord metastasis following a slowly progressive course

Sir: Intramedullary spinal cord metastasis is a well known but rare complication of cancer.[1] In the majority of patients it occurs in conjunction with widespread systemic disease and progresses rapidly.[2 3] We report a patient in whom the condition was the only indication of relapse and whose signs and symptoms progressed slowly.

A 54 year old woman was referred to our department because of difficulty in walking, burning sensations in the thighs and low backache, slowly progressive over the past year. At the age of 52 an adenocarcinoma of the lung had been detected and a lobectomy of the left lung had been performed.

Examination showed proximal paresis of the legs, MRC grade 3–4/5, more pronounced on the right side. Distally, the strength was nearly normal. Superficial pain and