We describe three patients with pre-ganglionic (avulsion) injuries of the brachial plexus which caused a partial Brown-Séquard syndrome.

A partial Brown-Séquard syndrome occurs in between 2% and 5% of patients who sustain pre-ganglionic (avulsion) injuries to the brachial plexus.1 We report three cases which illustrate its causation, presentation and progression.

Case reports

Case 1. A 23-year-old man sustained multiple injuries including a right-sided injury to the brachial plexus in a motorcycle accident in October 1993. His other injuries prevented exploration of the brachial plexus for six months. At operation, post-ganglionic ruptures of C5 and C6 and avulsion of the roots of C7, C8 and T1 were identified. Grafts from the superficial radial nerve were placed between the stump of C5 and the suprascapular nerve and lateral cord.

After two years he had returned to full-time employment, having retrained as an engineer. He returned six years after his accident complaining of severe intermittent disabling pain in the upper limb which had started 18 months earlier. He had also become aware of some loss of temperature sense in his left lower limb four years after his accident. On examination at that time there was some recovery of function with grade 4 power in pectoralis major and biceps on the Medical Research Council (MRC) scale.2 After seven years he noted weakness of his right lower limb which progressed from a reduced running distance to difficulty walking and cycling. The sensory changes in his left lower limb also progressed and after eight years he was aware that the sensation of light touch in the left lower limb had diminished.

After 13 years he had developed upper motor neurone weakness in his right lower limb with sustained clonus and an upgoing plantar response. Sensation to pinprick was reduced in his left leg as was temperature sensation. The threshold for cooling was 10°C on the sole of the left foot, compared with 1.4°C on the right. The sensation of pinprick and temperature was diminished below T4 on the left side. Vibration sense was diminished in both lower limbs, with a threshold of 24 units on the right and 18 units on the left. Joint position sense was normal. The sensory test methods which we used, and their normal values, have been described by Quraishi et al3 (Fig. 1).

MRI of the cervical spine after 13 years showed cavitation of the spinal cord at C7 (Fig. 2). The cord was displaced to the right and was tethered in the foramen of C7 which could be seen on the MRI.

CT angiogram showed intact carotid and vertebral arteries. A dynamic flexion-extension MRI of the cervical spine revealed no instability. The patient declined surgery to release his spinal cord, but remains under review.

Case 2. A 27-year-old shop manager sustained an isolated injury to his left upper limb in a motorcycle accident in April 1995. He was found to have a severe brachial plexus lesion, a Bernard-Horner sign and a fractured scapula. A CT myelogram indicated avulsion of the roots of C6 to T1 with a pseudomeningocele at T1. The brachial plexus was explored two months after the accident and these findings were confirmed. In addition, there was a post-ganglionic rupture of C5. An attempt was made to attach a small stump of C5 to the root of C6, but since the area was severely scarred further grafting was thought to be futile.

Two years later he underwent neurotisation of the fourth and fifth intercostal nerves to the radial nerve in an attempt to relieve severe pain on the back of his left hand. This improved his condition and he was able to work as a gardener and harman.

In 1999, four years after his accident he presented with a one-year history of severe shooting pains in his left arm. The pain was worsened...
by cold and by light touch. He graded it as 9 of 10 on a visual analogue scale. There had been some recovery of sensation in the fifth cervical dermatome, but no return of muscle function. An area of hypersensitivity was found just above and behind the elbow which caused pain to radiate up the arm and down to the hand. Medication for neuropathic pain was begun. MRI in 2002 showed irregularity of the spinal cord with evidence of deposits of haemosiderin.

Three years later, ten years after his accident, he noticed weakness of his left leg and decreased sensation in his right lower leg, particularly the sense of temperature. He had noticed wasting of the left leg and was unable to run. MRI suggested Wallerian degeneration of the dorsal columns of the cervical cord. On examination, there was generalised weakness (MRC grade 2) in the hamstrings (MRC grade 3) dorsiflexors of the ankle (MRC grade 3). He was found to have brisk reflexes, but normal sensation and temperature sense. The power and reflexes in his right (contralateral) lower limb were normal but, with altered sensation, loss of pinprick sensation and temperature sense. Vibration sense was normal in both lower limbs. Thus he had a partial Brown-Séquard syndrome with sparing of the dorsal columns.

MRI of the cervical spine carried out the following day showed an extensive pre-vertebral haematoma and oedema of the cord from C4 to C7. He underwent exploration of the brachial plexus three weeks after his initial injury. He was found to have a rupture of C5 and a complete avulsion of C6; C7 and C8 were undisplaced, but with no good sensory action potential and were therefore thought to have been injured within the spinal cord. T1 was not exposed. C5 was grafted using the superficial branch of the radial nerve, and the spinal accessory nerve was transferred onto the ventral root of C6.

Six weeks after the injury he developed neuropathic pain, describing it as burning and throbbing with occasional shooting from elbow to hand. He was given pregabalin (initially 150 mg bd increased to 300 mg bd. Two weeks later the power in the left lower limb was found to have improved with MRC grade-4/5 power in the hip flexors. Sensation to pinprick remained diminished in his right leg.
He returned to part-time work five months after the accident. Four months after this when his pain was less severe he returned to driving an adapted automatic car. The Brown-Séquard-syndrome was present but not progressing. MRI showed abnormal linear hyperintensity in the T2-weighted images at the C4 and C5 level but without any definite epidural collection.

Discussion
In 1949, Penfield\(^4\) described the case of a boy who sustained an injury of the brachial plexus and underwent amputation of his right arm at the age of 14 years. After 37 years he presented with increasing weakness and spasticity of both lower limbs, more marked on the right side. Nine years later his symptoms were so incapacitating that surgery was indicated. A lower cervical laminectomy was carried out and the spinal cord was found to be displaced to the right at the level of the first thoracic segment where it passed in a curve out into the intervertebral foramen and back again. Penfield\(^4\) stated: “When it (the dura) was passed in a curve out into the intervertebral foramen and the right at the level of the first thoracic nerve was well exposed. This was released and the cord then returned to the midline. Initially, the patient was weaker but the strength started to improve after three weeks. He died from cardiac disease five years later, but remained well until a few months before his death when the weakness returned and progressed.

A complete hemisection of the cord resulting in Brown-Séquard syndrome is rare. Its causes include blunt and penetrating trauma, tumour, degenerative disease, infection and haemorrhage. Pre-ganglionic lesions of the brachial plexus can be classified into two groups. Either the roots are ruptured in their intradural course, leaving central stumps of variable length (peripheral intradural pre-ganglionic rupture) or they are torn directly from the cord and central nervous tissue is attached to the avulsed root leaving a defect in the cord (central pre-ganglionic avulsion). Both lead to the death of motor neurones in the anterior horn, but since central avulsion is an injury to the central nervous system it results in scarring within the spinal cord.\(^1,5\)

A true central avulsion causes a partial Brown-Séquard syndrome at the time of injury,\(^6\) and is estimated to occur in between 2% and 5% of patients with brachial plexus injuries.\(^1\) In our first case there was no vascular injury and no evidence of an ischaemic lesion of the cord. We believe that the deepening partial Brown-Séquard syndrome was caused by progressive tethering and distortion of the cord by scar tissue. In our second case there was a sudden onset of new symptoms in a patient with no signs of Brown-Séquard syndrome at the time of injury, but in this case the symptoms were not present at all times. In the presence of deposits of haemosiderin we have attributed this to an ischaemic injury. In our third case the early onset of symptoms from the cord lesion were directly attributable to the avulsion of the spinal roots. Domisse\(^7,8\) has shown that the radicular vessels enter the spinal cord with the spinal nerves, in particular at C7 and C8, and that these vessels are injured if the roots are avulsed. Therefore an acute Brown-Séquard syndrome is caused by ischaemia but similar symptoms which arise later are usually the result of fibrosis or distortion of the cord.

The pathway from the dorsal column to the medial lemniscus contains the gracile and cuneate fascicles which cross over in the medulla oblongata. They convey the senses of fine touch, vibration and proprioception. The lower limb is represented medially in the gracile fascicle and the upper limb laterally in the cuneate. The sense of vibration is located more posteriorly than that of position.\(^9\)

The reduced sense of vibration in both lower limbs seen in our first case may be explained by the extension of the cord lesion past the posterior median septum into the left gracile fascicle, but sparing the cuneate. It may also account for the preserved position sense in both lower limbs. In our third case the preservation of vibration sense in both lower limbs indicated a lesion which had spared the ipsilateral gracile fascicle. The level at which the different pathways in the spinothalamic tract cross to the other side of the cord varies. Pain and temperature pathways cross predictably but those for other modalities may ascend before crossing. The late onset of pain or weakness of the lower limbs in patients who have sustained a significant brachial plexus injury calls for prompt, thorough investigation to exclude a new lesion of the cervical spinal cord.

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