

Brachial plexus injury

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The brachial plexus is a collection of peripheral nerves, the complexity of which dictates slow, unpredictable and sometimes confused recovery after injury; surgical repair should therefore form part of an integrated overall plan for repair of the limb. This may also include tendon transfers, osteotomies, arthrodesis and possibly muscle autotransplantation (see later). The structure of the brachial plexus determines the:

- pattern and pathology of injury
- clinical and diagnostic findings
- surgical options available for treatment.

These three topics form the basis for the rest of this contribution. This article should be read in conjunction with 'Peripheral nerve disorders', page 259, and 'Physiology of the peripheral nerve system', page 264a.

Pattern and pathology of injury

Traction

In most 'developed' countries, traction is the most common injuring mechanism for the brachial plexus, though penetrating injury by gunshot or blade is becoming increasingly common in some places. Motorcycle accidents account for most brachial plexus injuries to adults in the UK, and these lesions are predominantly a condition of young men, devastating the patient at the beginning of his working life. In these accidents, the head and shoulder may be forced apart, applying force to one or many roots of the plexus. Fracture of the clavicle (especially with fracture to the neck of the scapula) allows further traction separation of the arm from the torso and correspondingly greater force to be applied to the brachial plexus. Traction may:

- stretch the nerve root (leaving it in physical continuity)
- rupture the nerve root (two stumps will then be available for repair)
- avulse the nerve root from the spinal cord (no proximal stump available).

Avulsion – only one end of the root is available to the surgeon, and repair is not possible. Sometimes, the root may be torn from the spinal cord, but not from the foramen, giving the appearance of non-injury: a potentially confusing discovery for the unaware. Any of the injury types may occur to any or all of the five roots,

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providing a rich variety of possible pathology. However, in practice, certain patterns are more commonly seen.

Injury to the supraclavicular portion of the plexus is most common and, in 10–15% of these injuries, the subclavian artery is also ruptured. Avulsion from the spinal cord may be accompanied by a lesion within the cord. Avulsion of all roots (C5–T1) occurs in a large proportion of brachial plexus injuries caused by motorcycle accidents: at least 15% of patients who present initially with no limb function. Avulsion of the lower roots (C8–T1) is common and rupture usually occurs in the upper roots (C5–C6).

Some injuries do not occur in the proximal (supraclavicular) plexus but in the distal (infraclavicular) region. These injuries are often less severe and may follow anterior dislocation of the shoulder. Despite their more benign course, they may include nerve rupture or even division of a nerve by the sharp edge of the pectoralis minor tendon or, in the case of the axillary nerve the subscapularis tendon.

About 10% of supraclavicular injuries are associated with infraclavicular injuries (i.e. double-level injuries). From this we can identify some useful categories and descriptions of brachial plexus injury (Figure 1).

Stretched nerve root (lesion-in-continuity) – the essential question is 'Will this lesion-in-continuity recover better if left alone than if I excise and graft (repair) it?' In the past, many surgeons have avoided this question by adopting a cautious policy of waiting to see what spontaneous recovery occurs before embarking on repair. The authors believe that satisfactory spontaneous recovery occurs only if there is a simple physiological conduction block (rare) or an axonotmesis leaving internal neural microtopography intact. Both of these will present a reassuringly normal microscopic appearance on early exposure and inspection.

Clinical and diagnostic findings

The clinical features of a brachial plexus injury lead to a diagnosis, and so a prognosis, without which the surgeon cannot make a judgement about the advisability and strategy of surgery.

The history of the accident offers some clues. As the dissipated kinetic energy rises (proportionate to the mass and the square of the velocity), so does the likely damage. American footballers often suffer transient plexus palsy because, despite their mass, their speed is low, as is the kinetic energy dissipated. The plexus damage is therefore correspondingly benign. This is not true for a lightweight motorcyclist airborne at 30 miles per hour and facing an abrupt halt.

Types of brachial plexus injury

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| • Open | e.g. gunshot, blade, surgeon |
| • Closed | traction, occasionally crush injury, very occasionally radiation injury |
| • Total | global (in all roots) |
| • Partial | predominantly upper or lower |
| • Supraclavicular | prognostic significance |
| • Infraclavicular | prognostic significance |

Other injuries must be suspected and sought, particularly head injuries, spinal damage and thoracic and vascular damage.

Symptoms

The symptoms of patients with brachial plexus injury are usually those of paralysis, altered or absent sensibility, sometimes pain, and those associated with other injuries. It is helpful to establish any evolution of symptoms since the accident; rapid and considerable amelioration may prompt reservations about surgery.

The pain of deafferentation, a dreadful, crushing, burning, distracting and unremitting pain which is worse at night, is quite common with avulsion injuries, but may not be present in the immediate aftermath of the injury.

Signs

The signs of brachial plexus injury are those relating to adjacent structures and those attributable to plexus damage itself.

Some signs strongly suggest the avulsion of roots. These signs must be sought.

- Horner's syndrome (Figure 2) implies T1 root avulsions because the sympathetic rami leave this root as it emerges from the foramen.
- Phrenic nerve palsy suggests C4 and C5 avulsion, as does rhomboid paralysis.
- In the lower limb, altered sensitivity, upgoing plantar reflex or poor proprioception (can the patient stand on one leg, eyes closed?) implies spinal cord damage associated with avulsions. Other adjacent structures may provide clues to injury: pulses may be absent or diminished if the subclavian artery is occluded, or a large haematoma may suggest rupture of the vessel. Fracture of rib or clavicle has no specific diagnostic significance.

Examination of the plexus itself provides the most information. The absence of Tinel's sign (Figure 3) within the region of the plexus may be a gloomy finding, as it implies no proximal nerve stumps and thus indicates an avulsion injury.

The disposition of paralysis and sensory loss may fit a root, cord or nerve pattern. Careful charting, repeated several times over the first few days, will guide the surgeon to an anatomical diagnosis. It is helpful to follow the discipline of a formal muscle and sensory chart and, if one is not available, it may be constructed quickly by listing the muscle groups of the upper limb in anatomical order. The examiner then records power and sensibility (to light touch and pinprick).

Investigations

Imaging: few investigations are of real help in the severe or high-energy injury. A raised hemidiaphragm on a chest radiograph may indicate phrenic nerve palsy, but otherwise imaging is problematic.

Clinical features of Horner's syndrome

- Loss of head and neck sympathetic tone
- Miosis (constricted pupil)
- Ptosis of the upper lid (Müller's muscle)
- Anhidrosis of half face
- Enophthalmos (uncertain cause)

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Tinel's sign

- Tingling in the sensory distribution of a nerve (motor or sensory) on percussion of the nerve trunk
- A sign of irritability (often injury) at the site where the phenomenon is elicited—it also occurs at a point reached by a recovering nerve and so a 'migrating' Tinel's sign is evidence of the gradual distal progress of neural recovery—an encouraging and useful piece of information. However, beware! Tinel's sign has no quantitative value and is subjective; it does not predict the extent of the recovery.

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The current resolution of MRI does not provide the anticipated soft tissue definition needed to distinguish rupture from continuity or, in most cases, to see avulsions. CT scans are usually combined with myelography, and the results are user-dependent. Myelography is also skill-dependent, but can yield remarkable accuracy in capable hands. Each of these techniques may give false-positive and false-negative results, but the author's practice is to request these images if the patient is appropriately fit and cooperative. They allow the patient to be prepared for surgical exploration, and imaging findings can then be compared with findings at surgery. For example, a nerve root clearly seen on myelography but apparently absent at exploration, should be sought far into the root foramen.

Neurophysiology is not valuable in the first weeks after injury. It may subsequently identify denervation potentials in muscles and, more importantly, subclinical signs of innervation or recovery. It may also show preserved sensory potentials in the presence of sensory loss, implying avulsion or conduction block, which in turn are distinguished by the presence of preserved motor conduction (conduction block, i.e. neurapraxia).

Exploration is the most accurate investigation of the supraclavicular plexus. If this inspection is undertaken in the first week after injury, the effect of trauma on the anatomical structures is easily identified and understood. This premise, however, raises arguments for and against early surgery. Current opinion suggests that early surgery offers the best diagnostic and therapeutic opportunities for significant supraclavicular lesions (other factors permitting) as well as improving the overall management of brachial plexus injury (Figure 4).

The argument against early surgery usually hinges on the response of the surgeon to discovering one or more functionless roots in continuity. In practice this is a rare finding. Such surgeons will be experienced in peripheral nerve surgery and able to categorize most 'in continuity' lesions as recoverable or not recoverable. If in doubt, it is possible to leave the decision on the particular root for subsequent re-exploration. In the author's practice, this has seldom been necessary (never in an adult). Like many surgeons, the author believes that the benefits of early diagnosis, prognosis and treatment outweigh the small chance of such a dilemma. In addition, there is strong clinical evidence of the deleterious effects of presurgical delay on final outcome for motor and sensory function following nerve repair.

Early or delayed exploration of supraclavicular plexus

Arguments for early exploration

- Allows ease of diagnosis because tissue planes are open, fibrosis and scarring are absent and the nerve in continuity is assessed visually
- Surgery is not destructive and carries little morbidity or penalty
- Allows early repair, avoiding increased target organ decay
- Allows early prognosis for patient and consequent early decisions about future employment, rehabilitation and resettlement
- Surgery is easier, dissection less traumatic and anaesthesia less prolonged
- Results are inversely proportional to presurgical delay

Arguments against early exploration

- Spontaneous recovery may occur without surgery
- The lesion-in-continuity may or may not recover and the surgeon is occasionally unable to determine this at surgery

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In many patients, other injuries prevent early exploration and the surgeon must then decide when to explore the plexus. They should consider that a Tinel's sign occurs at the regenerating front of a peripheral nerve, but that it is not a quantitative phenomenon. Also, the consequence of prolonged delay in repair (more than 3 months) leads to further substantial degeneration of the target organ, and probably the anterior horn cells and dorsal column cells, which are needed for central reconnection.

Options at surgery

The anatomical diagnosis

The first aim of surgery is to establish the anatomical diagnosis. To the gross anatomical findings may be added information from sensory-evoked potentials, useful when an apparently normal functionless nerve root is encountered. In such circumstances, an intradural avulsion may have occurred, but with the root remaining in the foraminal canal. Stimulating this root will fail to produce a sensory-evoked potential on the sensory cortex. Such recording requires standard conditions of temperature and anaesthesia, and experience in interpretation. Similarly, tetanic intraoperative stimulation of the nerves encountered will detect any subclinical motor activity, which may add to diagnosis. Recording conduction across a neuroma is not useful.

Repair or reconstruction?

When the diagnosis is complete, the surgeon can address the question of repair or reconstruction. A discussion of the full range of options available is not possible here; certain general principles, however, should be understood (Figure 5).

Primary nerve repair is not possible after traction injury, and grafts will be required. Reconstruction of avulsed nerves may require axons to be donated from other nearby nerves. Examples of such 'nerve transfers' include transfer from phrenic nerve, spinal accessory nerve, intercostal nerves and, most controversially, from the contralateral C7 nerve root, which appears to be

Principles of surgical repair or reconstruction

- All ruptures require nerve grafts for repair, because there is a zone of injury and when this is excised tension-free coaptation (essential in nerve repair) is not possible
- Avulsion injuries cannot be repaired but other nerves may be transferred to re-innervate the avulsed nerve (see below)
- Nerve transfers are usually undertaken to restore motor function; the avulsed root or the distal nerve it supplies may be co-opted to a donor nerve that provides motor axons for regeneration
- Surgery aims for a stable shoulder with external rotation, elbow flexion and as much hand function as possible
- Surgery solutions must be designed by a surgeon or surgical team who will take responsibility for integrating nerve recovery with later treatments such as arthrodeses, osteotomies, or tendon transfers

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expendable with relatively slight penalty. Such nerve transfers can also come from adjacent roots of the plexus (intraplexal transfer). For example, if the C8–T1 roots are avulsed and the C5, C6, C7 roots are ruptured, one may direct most of the plexus axonal material to the shoulder and hand (by repairing C5 and transferring C6 to C8 and C7 to T1), recognizing that restoration of the simple function of elbow flexion may be achieved by other means. Alternatively, proximity of nerve stumps from different roots may make it possible to repair without grafting; this is hard to resist in terms of subsequent speed and quality of recovery. In short, the options open to the reconstructing surgeon are now greater at primary surgery because of the greater understanding of the role of nerve transfers.

Free-functioning muscle autotransplantation: in this technique, whole muscles (e.g. gracilis) are microsurgically transplanted, revascularized and re-innervated within the recipient limb. This is necessary only when the normal muscle has deteriorated beyond re-innervation by late repair. A common example is the gracilis muscle in late presentation brachial plexus injury, where the elbow flexors have suffered irrecoverable endplate degeneration. Nerve transfers may re-innervate these transplanted muscles, in this case, usually the intercostal nerves 3, 4 and 5.

Results

Some general principles dictate the extent of recovery that may be expected. Recovery is inversely related to the presurgical delay, and the longer the nerve grafts used, the poorer the result. Grafts of less than 5 cm yield the best results, but are not always possible; grafts larger than 10 cm yield poor results, which may nevertheless in some cases be obviated by successful microsurgically vascularized nerve grafting. The greater the energy dissipated at trauma, and the more nerve roots avulsed, the worse the outlook; nerve transfers seldom function as well as orthotopic repairs. Pain can prevent attention to rehabilitation regimens: a motivated and determined patient will make the most of his or her recovery, but it is difficult to remain enthusiastic and motivated when in constant, disabling pain. ♦