Instructional Course Lecture: Assessment and Early Management of Traumatic Brachial Plexus Injury
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Abstract
Closed traumatic brachial plexus injury is usually associated with a high energy injury mechanism and is usually caused by traction on the cervical spine, upper limb or both. There are frequently other potentially life-threatening injuries which require prompt early intervention and for this reason the brachial plexus injury is often initially missed. The aim of this review is to describe the patterns of plexus injury and the important features to help in early diagnosis, assessment and management.

INTRODUCTION
The brachial plexus is formed from the ventral and dorsal rootlets from C5 to T1 which leave the cervical spinal cord and combine to form the spinal roots. The dural covering extends as far as the intervertebral foramina where the spinal roots emerge between the anterior and middle scalene muscles and enter the neck. The roots are intimately related to the higher cervical roots which form the cervical plexus and they receive sympathetic outflow from the stellate ganglion at the cervicothoracic junction.

The C5 and C6 roots merge to form the upper trunk. The C7 continues as the middle trunk and the C8 and T1 roots combine to form the lower trunk within the posterior triangle of the neck. Each trunk has anterior and posterior divisions at the level of the clavicle and further branching results in the lateral, posterior and medial cords which are named relative to their relationship with the axillary artery below the clavicle.

The upper plexus has branches and is relatively well fixed at the level of the scalene muscles. The lower trunk is more mobile. Traction to the upper trunk may produce nerve ruptures which are distal to the dorsal root ganglion and are therefore known as postganglionic injuries. These injuries are frequently amenable to surgical repair or reconstruction.

The lower trunk is more prone to direct avulsion from the spinal cord and the injury is often proximal to the dorsal root ganglion and therefore is called a preganglionic injury. The dorsal root ganglion contains the cell body of the sensory axons and as because this remains in continuity with the peripheral nerve in a preganglionic injury, sensory conduction is still present, although not perceived at cortical level due to the lack of afferent connections.

Plexus injuries are anatomically divided into supraclavicular and infraclavicular injuries. Knowledge of plexus anatomy helps to define the level of the brachial plexus injury which is important for subsequent management. In the supraclavicular region, the important branches include the phrenic nerve arising from C3-5, the dorsal scapular nerve from the upper C5 root, the long thoracic nerve from C5-7 and the suprascapular nerve from the upper trunk C5-6. In the infraclavicular plexus the important branches are the axillary and radial nerves from the posterior cord, the musculocutaneous nerve from the lateral cord, the median nerve from both the lateral and medial cords and the ulnar nerve from the medial cord.

CLASSIFICATION
The majority of plexus injuries in the adult are closed avulsion or traction injuries and they may involve predominantly the upper (70%), lower (20%) or whole (10%) of the plexus. The incidence of stabbings, gunshots or blast injuries is on the increase and these mechanisms may result in open plexus injury. There is a rise in the number of interventional vascular procedures performed by physicians and this has been mirrored by a rise in iatrogenic brachial plexus injury. The Leffert classification is a commonly used system to describe brachial plexus lesions. The Power modification is more comprehensive and provides useful prognostic information regarding the injury. The Type
defines the mechanism and the subtype defines the severity of the injury. Subtype “C” injuries are severe and most likely to have associated vascular involvement.

**Figure 1**

<table>
<thead>
<tr>
<th>Power modification of Leffert Classification:</th>
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<tbody>
<tr>
<td><strong>Type I</strong> Open (supraclavicular or infraclavicular lesion)</td>
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<tr>
<td>A Stabbing</td>
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<tr>
<td>B Low velocity gunshot</td>
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<tr>
<td>C High velocity gunshot or blast</td>
</tr>
<tr>
<td><strong>Type II</strong> Closed (supraclavicular or infraclavicular lesion)</td>
</tr>
<tr>
<td>A Upper plexus (C5-6)</td>
</tr>
<tr>
<td>B Lower plexus (C8-T1)</td>
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<tr>
<td>C Pan plexus (C5-T1)</td>
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<tr>
<td><strong>Type III</strong> Pathological and iatrogenic</td>
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<tr>
<td>A Iatrogenic</td>
</tr>
<tr>
<td>B Post radiation</td>
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<tr>
<td>C Malignancy</td>
</tr>
<tr>
<td><strong>Type IV</strong> Obstetric</td>
</tr>
<tr>
<td>A Erb's (C5-6)</td>
</tr>
<tr>
<td>B Klumpke (C8-T1)</td>
</tr>
<tr>
<td>C Mixed (C5-T1)</td>
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**MECHANISM**

Type II closed brachial plexus injuries are the commonest seen in our practice. They are effectively traction injuries that have occurred during high energy transfer. Common mechanisms are motorcycle accidents, pedestrians hit by motor vehicles and in falls. Upper trunk injury may occur with a fall onto the top of the shoulder depressing the shoulder girdle whilst the head also hits the ground and the neck is laterally flexed to the contralateral side. Lower trunk and root injury may occur when the arm is forcibly abducted. The situation is not as simple as this and most mechanisms involve a complex range of upper limb and spinal motion as the flails about the torso.

Type I open injuries are less common and may range from minor penetrating wounds in the supraclavicular fossa or axilla to complex major blast wounds with near amputation of the upper limb.

**INITIAL ASSESSMENT**

All trauma patients should be assessed using the ATLS principle. Life threatening injuries must be rapidly identified and treated. A systematic secondary survey will identify the remaining injuries. The mechanism of the injury should be reported to the trauma team by the pre-hospital care team and this information will help to predict expected injury patterns.

Brachial plexus injury is often found in association with major head injuries and the depressed level of consciousness may make neurological examination of the limbs difficult.

Cervical spine fractures are common including body and lateral mass fractures. Transverse element avulsions are frequently seen. Root avulsions from the spinal cord may result in bleeding into the cord and upper motor neurone long tract signs in the legs.

Fractures and dislocations of the shoulder girdle and upper limb are common. The sternoclavicular joint may be dislocated, the clavicle or scapula fractured. There may be a floating shoulder with ipsilateral humeral shaft fracture. The shoulder may be dislocated or unstable, having dislocated at the time of the injury. The periscapular musculature is often injured and severe cases may present with scapulothoracic dissociation. This is often a fatal injury but shorter times on scene and quicker helicopter transfers may mean that some of these patients will make it to hospital and early surgical intervention for the associated vascular injury may be life saving.

The torso is frequently injured with haemopneumothoraces, rib fractures (including the first), abdominal visceral injuries and other limb injuries. The phrenic nerve may be injured resulting in decreased expansion of the ipsilateral chest.

The patient may present with a Horner's Sign from the damage to the head and neck sympathetic outflow through the cervicothoracic, mid and upper cervical sympathetic ganglia. This is most often present with preganglionic root avulsions at the C8-T1 level. The other cutaneous nerves within the cervical plexus may be injured with commonly numbness in the C4 territory to the apex of the shoulder and the supraclavicular nerves across to the upper chest and as far as the nipple line.

Pulses may be reduced in the upper limb due to damage to the subclavian or axillary vessels. There may be an expanding haematoma within the neck.

**ASSESSMENT OF THE PLEXUS INJURY**

The examination of the injured arm should be complete, but key peripheral nerves should be examined to try and identify the location and extent of the injury within the plexus. The dorsal scapular supplies the rhomboids and if paralysed the scapulae will not be retractable and the shoulder may tilt inferiorly and forwards. The long thoracic nerve supplies serratus anterior and if paralysed the scapula will wing at its
medial border. These three nerves arise proximally in the plexus from the roots or upper trunk and they may be intact in a distal injury at the infraclavicular level. When paralysed they signify a high and proximal injury and further assessment will be necessary to establish whether the injury is pre or post ganglionic.

The suprascapular nerve aids shoulder elevation and external rotation and when paralysed the shoulder will be held in internal rotation. This nerve arises from the upper trunk but is more commonly injured distally as it winds around the spine of the scapula or as it crosses through the suprascapular notch. Therefore it is less reliable as a marker of level of plexus injury.

Biceps function should be assessed as the musculocutaneous nerve arises from the lateral cord and is a key upper limb muscle function and failure of recovery of biceps function is often used to gauge timing of operative exploration in closed traction injuries.

Hand function should be assessed thoroughly because this provides the most accurate guide to functional outcome. Lower root involvement at C8 and T1 will result in poor hand function.

Lower limb neurological examination may reveal brisk reflexes when long tract injury has occurred due to root avulsions from the cervical spinal cord.

**RADIOGRAPHIC ASSESSMENT**

The trauma chest radiograph may show an elevated hemidiaphragm due to the phrenic nerve palsy associated with upper trunk and pan plexus injury. There may be rib fractures, haemothorax or a pleural cap from the haemorrhage around the plexus or from an associated major vessel injury.

Radiographs of the upper limb should be taken as fractures may not readily be apparent and the clinical findings are often unreliable due to the associated nerve injury.

**SPECIAL INVESTIGATIONS**

Further investigations are often necessary to exclude associated injuries to the spinal cord or to the main vessels. They may also help to establish the type of injury to the nerve root and whether distal rupture is present or the more severe preganglionic root avulsion from the spinal cord.

**ANGIOGRAPHY**

Invasive angiography is the gold standard investigation to establish a major vessel injury. It requires specialised expertise from an interventional radiologist and takes time to perform. There are significant complications associated with this investigation.

Indirect angiography may be performed more readily with intravenous injection of the contrast followed by CT of the chest and extremity.

**MYELOGRAPHY**

This was the gold standard investigation for many years and was used to look for torn dural sacs around damaged nerve roots that are called pseudomeningoceles. It was unreliable in the acute setting due to clotted blood obscuring the imaging of the avulsed root within the neural foramina. It was useful preoperatively because CSF leaks can complicate neck exploration after plexus injury.

**COMPUTED TOMOGRAPHY**

CT is used to establish extent of neck and chest injuries associated with the brachial plexus injury. CT myelography replaced myelograms for assessing root avulsion injury from the spinal cord but may overestimate root avulsion injury as pseudomeningoceles may be present with intact roots within them.

**MAGNETIC RESONANCE IMAGING**

This is not useful in the acute setting due to other complex and potentially life-threatening injuries that take priority. Later it is used to assess cervical cord injury and presence of pseudomeningoceles. Modern scanners and expert reporting provide reliable information on nerve root injury that approaches the sensitivity and specificity of traditional myelographic examinations. MRI is of less use in imaging the plexus in the acute phase because there is intense signal change around the whole plexus, even with minor injuries. Later it may be of use in preoperative planning and may even show large neuromata in the chronic plexus injury.

**NEUROPHYSIOLOGICAL TESTS**

It is impossible to distinguish between neuropraxia, axontomesis and neurontomesis in the acute phase. After 6 weeks neuropraxia may resolve and electromyography at this stage may identify muscle denervation. This is particularly used in the paraspinal musculature supplied by the posterior primary rami to establish root integrity.

Sensory nerve action potentials may be recorded in preganglionic injury due to the intact sensory axon and cell body in the dorsal root ganglion. Motor action potentials will
be absent in these cases as there is no connection between the motor cell body in the anterior horn of the spinal cord and the axon and end plate.

Somatosensory evoked potentials with peripheral stimulations and cortical recording can help to identify intact central connections with intact nerve roots.

THE HISTAMINE TEST
This test to distinguish between pre and postganglionic injury is of historical interest only. It relies on the cutaneous axon reflexes. A drop of histamine on the skin along the distribution of a peripheral nerve should produce a sequential triple response with cutaneous vasodilatation, wheal formation, and a flare response. When a nerve is interrupted proximal to the ganglion a normal axon response will be seen due to the intact sensory fibres and the sympathetic outflow from the stellate ganglion. If the injury is distal to the ganglion there is local vasodilatation due to the mechanical stimulus and the histamine increases the oedema to produce the wheal, however the flare response is absent.

SURGICAL INICATIONS
When the condition of the patient permits exploration, open plexus injury should be treated with early assessment and primary repair with or without interpositional cable grafts from the sural nerves. Injuries to adjacent vessels must be treated first to restore perfusion to the upper extremity followed by repair of the plexus injury.

When the patient is transferred to a specialist unit and there is a delay, the surgery should wait until there is healing of the wound and stabilization of any other injuries. During this period any neurological deficits should be documented and recovery monitored to determine the injury level and extent. Electromyography may be used at approximately 4 weeks after the injury to assist in determining the level of the injury and any potential for spontaneous recovery. Exploration of the plexus is indicated for residual defects 6 weeks after the injury.

Closed injury due to traction should be monitored with serial clinical assessments. Infraclavicular brachial plexus injuries are less common than supraclavicular injuries and the prognosis is better. Neurophysiological testing can be undertaken at 4 weeks and physiotherapy used to maintain passive joint range of motion. Delayed surgical exploration may be undertaken after approximately 12 weeks when adequate time for spontaneous recovery of a neuropraxia has elapsed and there is a residual significant neurological defect. If function has not returned, if any return has ceased, or if the patient demonstrates non-anatomical return of function with isolated lack of proximal function in the presence of good distal nerve recovery and if there is evidence that the lesions are at the postganglionic level, then exploration is justified at 3 to 6 months after injury.

SURGICAL GOALS
In order of priority:

- restoration of elbow flexion
- restoration of shoulder abduction
- restoration of sensation to the medial border of the forearm and hand

After brachial plexus repair and reconstruction, 12 to 18 months are required to determine the extent of neural regeneration.

SUMMARY
Traumatic brachial plexus injury is often associated with other life-threatening injuries that should be treated first. Associated vascular injuries should be identified. Delayed exploration is warranted in closed injuries after a period of expectant observation. Open injuries should be explored early and primary repair undertaken where possible.

References
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