

Brachial Plexus Lesions

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ANATOMY

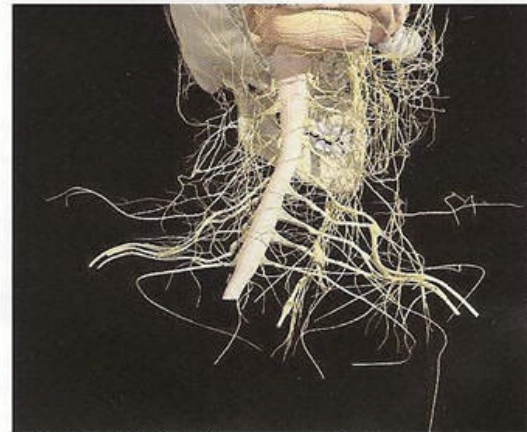
The brachial plexus is formed from the ventral rami of the lower four cervical nerve roots along with the ascending portion of the T1 nerve root although branches from C4 and T2 may also be involved. The nerves originating from the brachial plexus are responsible for the entire sensory and motor supply to the upper limbs as well as some back and chest muscles. The plexus extends from the neck to the axilla emerging from between the middle and anterior scalene muscles.

The C5 and C6 roots merge to form the upper trunk, C7 root continues as the middle trunk and C8 and T1 roots merge to form the lower trunk. These trunks divide into anterior and posterior divisions with the three posterior divisions combining to form the posterior cord, the anterior divisions of the upper middle trunks combining to form the lateral cord and the anterior division of the lower trunk continuing as the medial cord. The three cords are named by their position in relation to the middle part of the axillary artery. Each cord divides into two main branches in the region of the lateral aspect of the axillary artery with the posterior cord becoming the axillary nerve and radial nerve, the lateral cord becoming the musculo-cutaneous nerve and lateral head of the median nerve and the medial cord becoming the ulnar nerve and medial head of the median nerve.

The ventral rami of C5, 6 and 7 send off branches prior to the convergence of C5 and C6 to form the upper trunk. These branches join to form the long thoracic nerve and

supply the cervical para-spinal muscles, serratus anterior and rhomboids. Since these muscles are rarely, if ever, affected in brachial plexus lesions it is most likely that the damage occurs at the trunk level and not at the nerve root level. However, some patients with chronic "burners" were found to have fibrosed C5 and C6 nerve roots at the points where they emerged from the foramina (4). These changes may well be due to impingement rather than traction as will be discussed later.

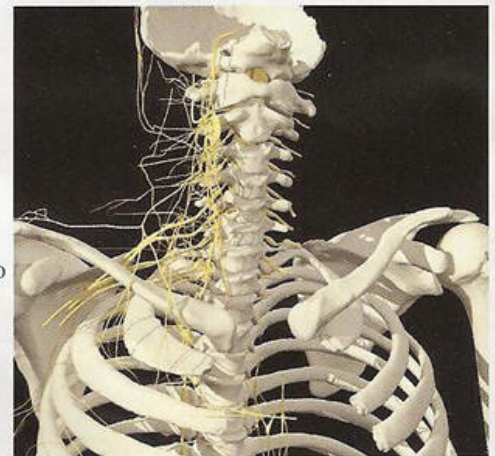
Fig 1 Cervical Neural Anatomy



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Fig 2 Cervico-Thoracic Neural Anatomy

Note: The more oblique angle of the upper trunk makes it more vulnerable to side flexion stresses



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MECHANISMS OF INJURY

True plexus injuries are as a result of traction on the plexus although the literature does include compression injuries which are the result of combined neck extension as well as ipsilateral side flexion and ipsilateral rotation. This combination of movements causes abrupt narrowing of the neural foramina resulting in pinching or nipping of the emerging nerve root and is similar to the Spurling test carried out to establish the existence of a cervical radiculopathy¹. A positive Spurling test indicates cervical foraminal stenosis due to disc herniation or osteophyte impingement into the involved neural foramen. Strictly speaking these compression injuries are nerve root rather than Plexus injuries and will not be dealt with in this article.

The single most common cause of brachial plexus lesions is the birthing process in natural, or vaginal, births and appears to be due to excessive traction being applied to the baby's head during delivery. This traction is applied because the infant's shoulders are stuck in the birth canal so the traction force on the brachial plexus is probably due to strong shoulder girdle depression. Depending on the study, 1-3 per 1,000 newborns are affected by OBPI (Obstetric Brachial Plexus Injury).

Stab wounds involving the plexus will, of course, also produce severe plexus symptoms and these are nearly always immediately explored and repaired surgically.

The usual mechanism of traumatic plexus injury involves a relatively high-speed impact, sometimes with an



opponent as in rugby and American football and sometimes with the ground or barrier as in motor-cycle racing and skiing etc. There is usually a rapid deceleration of horizontal forward momentum (diving head-on tackle, high-sided motor-bike

accident) with the impact involving simultaneous side-flexion of the head and neck away from the depressed shoulder girdle. This mechanism causes severe traction forces on the brachial plexus, especially the upper trunk and can result in one of three scenarios:

1: Neuropraxia

Mild traction which can result in a neuropraxia causing the "burners and stingers" well known and loved in rugby circles. The symptoms, as the names suggest, are characterised by a burning / stinging pain accompanied by numbness, paraesthesia and muscle weakness commonly affecting the whole arm from the upper fibres of the trapezius to the fingertips. Symptoms typically last from a few seconds to 20 minutes but some symptoms may still be present after three weeks. Histological findings show demyelination of the axon sheath but no axonal disruption.

Repeated neuropraxias at the same level can result in significant weakness affecting usually deltoid and biceps².

Although there are no figures indicating how common these symptoms are in rugby players at least 50% of college American footballers have experienced a "burner"^{3&4} on at least one occasion.

2: Axontmesis

Moderate force and traction can result in an axontmesis where there is disruption of the axon and the myelin sheath with preservation of the fibrous epineurium which acts as a conduit for the regenerating axon which, after a 7 day delay following injury, regenerates at a rate of about 1mm per day. Recovery, especially of the lower plexus lesions which have a longer distance to travel to their motor end-plates in the hand, is often incomplete as the muscles supplied by these nerves are frequently atrophied and replaced by fibrous tissue by the time axon regeneration has taken place

3: Neurotmesis

Neurotmesis is, thankfully, not very common in rugby but is seen all too often in motor-cycle racing and is sometimes associated with the significant trauma to the traversing major blood vessels, bone structure and muscles. Neurotmesis amounts to complete severance of the nerves and there would be no chance of distal regeneration without surgical repair of the nerve sheath, following which recovery should occur at a similar rate to that of an axontmesis but is seldom anything approaching a good recovery.

Although we conveniently place these three degrees of traction force and trauma in separate "boxes" it is common to find combinations of neuropraxia and

axontmesis in the same episode with some axons resisting the traction force while others surrender to the force.

SYMPTOMS OF PLEXUS LESIONS

Injuries affecting the upper part of the plexus (C5 and C6 levels) are most common due to the angle of the emerging nerve roots causing a greater stretching to be achieved by shoulder girdle forced depression associated with contra-lateral head / neck side flexion. Hence the most common symptoms consist of weakness or paralysis of deltoid, supraspinatus, infraspinatus, biceps and brachioradialis which is, of course, an Erb-Duchenne paralysis or "Erb's Palsy".

Posterior cord lesions (C5,6,7 and 8) can result in paralysis or weakness of deltoid, triceps, wrist extensors and finger extensors so the patient is unable to extend elbow, wrist or fingers.

Lower plexus lesions (C8 and T1) can result in weakness or paralysis of the lumbricals and interossei of the hand causing clawing of the hand known to us all as "Klumpke's Palsy" or Dejerine-Klumpke paralysis. There is often "Horner's Syndrome" present.

Complete brachial plexus injury results in total arm paralysis and numbness.

Most plexus injuries show maximal symptoms and deficit immediately at the time of injury. Gradual onset of deficit may point to vascular injuries.

Traumatic brachial plexus injuries must be distinguished from cervical radiculopathies and other acute cervical conditions at the earliest possible opportunity. It must be remembered that, in the majority of cases, the arm weakness in brachial plexus lesions is extra-segmental and the sensory dysfunction is not usually dermatomal⁵.

Recovering axontmesis cases are sometimes troubled by persistent neuromas at the injury site.

CASE HISTORY:

History of Injury

Mr E was a 33 year old amateur motor-cycle race rider who had suffered a high speed accident three days earlier in a club championship race. He had no recollection of the mechanics of the accident (knowing the mechanics of the trauma makes it much easier for the physiotherapist or attending physician to be able to work out what forces

may have been applied to what structures) as he had been rendered unconscious for several minutes after the accident and had associated retro-grade amnesia. The right shoulder of his leathers was damaged, as was the right side of his helmet.

He was complaining of a "funny" right shoulder which was not excessively painful but he was experiencing severe weakness of the shoulder in general. He also was complaining of a stiff, painful neck. He presented with X-rays of his right shoulder and neck along with a report from A&E indicating "no bone injury".

Examination

He needed help with undressing as he was unable to remove his shirt unaided.

There was severe ecchymosis and swelling extending over the whole of the right side of his neck and affecting the skin over the upper fibres of the trapezius and ventrally extending over the clavicular fascia.

Passive movements of his right shoulder were full and completely pain-free.

There was total loss of sensation over the dorsal aspect of the scapula above the spine of the scapula and extending to the point of the shoulder.

There was total paralysis of his right deltoid. Weak supraspinatus contractions were present.

There was severe weakness of his right biceps brachii, brachialis and brachioradialis. He was unable to flex his elbow against gravity.

The right biceps reflex was absent.

There was paraesthesia extending to the **left** thumb and index finger.

Spurling sign was positive to the left indicating a probable root compression due to vigorous pinching of the left C6 emerging nerve root during the abrupt narrowing of the C5 / 6 neural foramen on the contra-lateral side to the trauma.

There was extensive tearing of the musculature on the right side of the neck and shoulder girdle, hence the extensive ecchymosis.

All neck movements were limited especially side-flexion to the left. This limitation appeared to be mostly due to the muscle damage and caused no radiating arm symptoms.

Probable Injury Scenario and Diagnosis

The injury was probably caused by the patient's forward, horizontal momentum being suddenly stopped by his right shoulder and right side of his head impacting with the ground and producing extreme range of depression of his right shoulder girdle and, at the same time, such a degree of side-flexion to the left neck extension and rotation to the left that the left C6 nerve root was violently impinged at the neural foramen. In addition, the upper trunk of the right brachial plexus had suffered either an axontmesis or neurotmesis due to the violent traction applied on impact.

Treatment / Management

After establishing the probable diagnosis no treatment was given but a letter was written to the patient's GP indicating the history, diagnosis and recommendation for referral to either a neurologist or neuro-surgeon.

Review

The patient was reviewed three years after his injury during which time he had undergone two attempts at surgical repair of his right brachial plexus. Examination on review, however, showed there was still no sign of right deltoid or right biceps brachii contraction. The left sided thumb and index finger paraesthesia had resolved spontaneously within three to four weeks of the original trauma. Despite his deltoid and biceps paralysis the patient had returned to competitive motor-cycle race riding in a national championship.

Summary

Brachial plexus injuries are rare enough but bi-lateral brachial plexus injuries are almost unheard of in the literature. This case history is, therefore, an extremely rare case.

Although it is vital to be able to rapidly, and accurately, assess the presence of a brachial plexus injury with either axontmesis or neurotmesis, early assessment and prompt onward referral does not necessarily influence recovery in every case. Even severe brachial plexus injuries are all too often missed at A&E – I have personal experience of at least three, all of whom were racing motor-cyclists – so

one should not automatically presume that a "clean bill of health" from A&E indicates the absence of serious injury.

TREATMENT AND PROGNOSIS

The prognosis for the neuropraxias is usually excellent with recovery often taking minutes only. Patients who have never experienced the condition before need reassurance only after the physiotherapist has assessed that no more serious damage has occurred. In rugby this assessment is usually carried out on the pitch by the physios involved. Topical application of ice is often all that is required. In a team situation, such as rugby, the reassurance is often given by the experienced (older) player telling the injured player "I've had that dozens of times so don't worry". However, the player giving this helpful advice is often a battered forward and his advice may well not be taken seriously.

The prognosis for axontmesis and neurotmesis is variable with total brachial plexus neurotmesis being universally poor. Upper trunk axontmesis frequently makes an excellent recovery with deltoid and biceps strength returning to a functional level and sometimes to full strength. The assistance of a good physiotherapist can be vital in such cases with the provision of a steadily graduated exercise routine to stimulate muscle re-development. Recovery can take many, many months during which time the patient may become very frustrated with the slow improvement. The type of patient suffering brachial plexus trauma is frequently relatively young and active and patience in this age group is not a renowned characteristic.

Pain, however, is often debilitating for many years and, although numbers vary, about 50% of patients with brachial plexus injury have significant pain for more than three years^{6,7 & 8}. Pain management is an essential part of brachial plexus injury rehabilitation. The most effective means of modifying the pain are thought to be distraction by meaningful activity such as work or hobby; secondly, TENS⁸ is thought to be "significantly effective" in two out of three patients and variable pulse-width capabilities are thought to be important. An initial trial of eight hours per day for three to four weeks is recommended as the pain modifying effects were thought to be cumulative⁷. Trial with TENS may commence at any stage of the clinical course of brachial plexus injury, analgesics do not appear to help^{6 & 8}. Surgery may improve function but may not alter the level of pain^{6,8 & 9}. As in many chronic pain

syndromes, the ability of the physiotherapist to relate to the patient and vice versa is essential⁹.

PREVENTION?

Because of the nature of the mechanism of this type of injury it is very difficult to be able to prevent the trauma satisfactorily. Some ideas have been suggested in some sports where brachial plexus injuries are prevalent:

American Football: strengthen the neck muscles and elevators of the shoulder girdle to try to prevent excessive side-flexion and girdle depression. The use of thicker foam rubber rolls placed between the base of the helmet and the upper fibres of the trapezius to reduce side-flexion and extension. It is uncertain whether either of these approaches is effective.

Rugby: The rugby authorities have looked at the opposite approach, i.e. instead of increasing the padding and protection they are introducing rules to lessen the padding in shoulder pads. Although at first sight this seems a wrong approach, it is hoped that players will think twice about attempting high-speed crash tackles leading with the shoulder if it hurts to do so. Padding has been so effective that even severe blows to the upper fibres of the trapezius and deeper structures have not been too uncomfortable. There is little doubt, however, that correct tackling technique could help prevent such injuries and much emphasis is placed on tackling technique throughout all age groups of rugby players.

Motor-Cycle Racing: It would be almost impossible to prevent such injuries in motor-cycle racing since they are not usually caused by anything which can be changed by equipment modification, track layout or by legislation. The same problem exists with skiing, mountain biking etc.

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