Galen, describing the anatomy of the brachial plexus, was fascinated with the way its nerves display a complex anatomic matrix of anastomoses (Figure 1). Those are further hidden between the upper chest, neck, and shoulder, making their surgical exposure difficult. William Smellie is credited with the first description of brachial plexus palsy in the 18th century, which he noticed in a newborn. There is evidence however of a much earlier clinical diagnosis in the Syriac Book of Medicines from the 12th century. Traumatic brachial plexus injury was studied by Flaubert, Duplay, and Reclus in the 1800s. Erb and Klumpke described upper and lower injuries, respectively, and idiopathic brachial plexopathy was defined by Parsonage and Turner in 1948. Thorburn performed the first brachial plexus anastomosis in the 20th century. Nerve transfers were reported in the late 19th century (facial nerve, radial to median nerve), but successful brachial plexus transfers with good long-term follow-up functions were not reported until the mid-20th century, with the efforts of Lurje, Seddon, Kotani, and many others.

Anatomy and Variations

Each spinal nerve has a motor ventral root and a dorsal sensory root. These meet at the dorsal root ganglion, from which exit a ventral ramus and a dorsal ramus (Figure 2). The brachial plexus derives from the ventral rami of the C5-T1 spinal nerves, which combine to form the classic anastomotic progression: 5 roots, 3 trunks, 6 divisions, 3 cords, and 5 branches (Figure 1). C5 and C6 form the superior trunk, C7 the middle trunk, and C8-T1 the inferior trunk. Each trunk then bifurcates into an anterior and a posterior division (6 total). All posterior divisions fuse to form the posterior cord (posterior to axillary artery), from which emanate the axillary and radial nerves. The anterior divisions of the superior and middle trunks form the lateral cord, whereas that of the inferior trunk becomes the medial cord. The lateral cord leads to the musculocutaneous nerve and the medial cord to the ulnar nerve, and they both contribute to the median nerve. It is important to keep in mind that over half of cadaveric plexuses show significant variations of this classic model, with at least 38 alternative schemes. Some plexuses even demonstrate innervations from the C4 (11%) and T2 (1%) nerve roots. Variability from the classic model was noted more frequently among trunks than cords.

Early nerve branches at the root level include the dorsal scapular nerve (C5, to rhomboids and levator scapulae) and the long thoracic nerve (C5-7, to serratus anterior). Injury to these nerves and to paraspinous branches (arising from dorsal rami of spinal nerves) points to a more proximal insult

Learning Objectives: After participating in this CME activity, the neurosurgeon should be better able to:
1. Describe the basic anatomic organization of the brachial plexus.
2. Differentiate the major clinical syndromes of the brachial plexus, including traumatic injuries.
The posterior cord typically sends off the subscapular medial antebrachial cutaneous nerves, which are sensory. Other branches of the lateral cord, the medial pectoral nerve (C8-T1) originates from the superior trunk. The pectoral nerves may also be used in neurotization procedures. Other branches of the medial cord include the medial brachial cutaneous and medial antebrachial cutaneous nerves, which are sensory. The posterior cord typically sends off the subscapular nerves (C5-6, to teres major and subscapularis) and the thoracodorsal nerve (C7-8, to latissimus dorsi).

Beyond the dorsal root ganglion, the gray ramus and white ramus communicans form a circuit with the sympathetic ganglion and chain. A preganglionic injury (proximal) interrupts all sympathetic connections and is more likely to cause sympathetic symptoms such as early neuropathic pain and Horner syndrome (Figure 2). Such an injury is usually caused by root avulsion and is irreparable. Electrographically, and because the sensory axon reaching its body in the ganglion is not disrupted, the sensory nerve action potentials are paradoxically preserved that usually carries a worse prognosis. The supraclavicular nerve (C5-6, to supra- and infraspinatus muscles) and subclavus branch (C5-6) arise from the superior trunk. The supraclavicular nerve is an important recipient in nerve transfer surgery to restore shoulder abduction.

Although the lateral pectoral nerve (C5-7) takes off from the lateral cord, the medial pectoral nerve (C8-T1) originates from the medial cord. The pectoral nerves may also be used in neurotization procedures. Other branches of the medial cord include the medial brachial cutaneous and medial antebrachial cutaneous nerves, which are sensory. The posterior cord typically sends off the subscapular nerves (C5-6, to teres major and subscapularis) and the thoracodorsal nerve (C7-8, to latissimus dorsi).

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Despite profound clinical loss of sensation. Differentiating between pre- and postganglionic injuries is important for prognostic evaluation and to plan surgical intervention (Table 1). Neuropathic pain medications (such as gabapentin), sympathetic blocks, or even dorsal root entry zone lesional procedures may be considered in patients with intractable causalgia.

Clinical Syndromes

Discerning upper brachial plexus injury (Duchenne-Erb palsy) from lower brachial plexus injury (Klumpke palsy) is a crucial step in the clinical assessment (Table 2). Not only does this help to point toward the mechanism of injury or diagnostic etiology; it can also predict the prognosis and available surgical options for intervention. Erb palsy typically involves the upper roots (C5, C6, and occasionally C7), and causes weakness of the muscles innervated by those roots (deltoid, biceps, rhomboids, brachioradialis, supraspinatus, infraspinatus, supinator, and occasionally wrist extensors). Weakness of those muscles results in the chronic sign of waiter tip (shoulder adducted and internally rotated, elbow extended, and wrist flexed). A downward pull on the shoulder that widens the angle between the neck and shoulder stretches the upper roots of the brachial plexus. This can be seen in humeral head subluxation, parturition, or motor vehicle accidents with forceful downward displacement of the shoulder (Figure 3).

Klumpke palsy results from stretching of the lower roots (C8, T1, occasionally C7), which is caused by a forceful upward pull of the arm widening the angle of the axilla. This may occur in traumatic injuries causing traction of an abducted arm, parturition, or Pancoast tumors (apical lung masses compressing the thoracic outlet). Lower brachial plexus injuries cause hand weakness, resulting in chronic claw deformity. Horner syndrome may occur if T1 is involved. Klumpke palsy usually has a worse prognosis than upper injuries, and successful options for surgical treatment and neurotization are less available for lower brachial plexus palsies.

Thoracic outlet syndrome is caused by compression of the lower trunk or medial cord of the brachial plexus (C8-T1 distribution) in the region between the clavicle and the first rib. It can be caused by a cervical rib, elongated transverse process of C7, or repetitive movements and compression by the pectoralis minor muscle against the coracoid process. Most commonly, this syndrome also includes arterial

Table 1. Preganglionic Versus Postganglionic Injury

<table>
<thead>
<tr>
<th></th>
<th>Preganglionic</th>
<th>Postganglionic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prognosis</td>
<td>Poor</td>
<td>Better</td>
</tr>
<tr>
<td>Common mechanism</td>
<td>Root avulsion (pseudomeningocele on MRI)</td>
<td>Stretch, ballistic, laceration</td>
</tr>
<tr>
<td>Sympathetic syndrome</td>
<td>Early neuropathic pain, Horner</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Sensory nerve action potential</td>
<td>Preserved (despite hypesthesia)</td>
<td>Absent</td>
</tr>
<tr>
<td>Paraspinal muscles</td>
<td>Denervation potentials</td>
<td>Normal</td>
</tr>
<tr>
<td>Rhomboids</td>
<td>Affected (winging of scapula)</td>
<td>Normal</td>
</tr>
<tr>
<td>Serratus anterior muscle</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Intervention</td>
<td>Irreparable, requires nerve transfers</td>
<td>Repairable (reanastomosis, grafting)</td>
</tr>
</tbody>
</table>
(pallor, ischemia) and venous (swelling, edema) symptoms from compression of the subclavian vessels. Patients usually improve with physical therapy, and surgical decompression with scalenectomy and first rib resection are reserved for intractable cases.

Acute idiopathic brachial neuropathy, or Parsonage-Turner syndrome, involves the upper brachial plexus. It is believed to be caused by an autoimmune reaction to a viral insult or vaccination, but its etiology remains not well elucidated. Typically, the patient reports severe acute shoulder and upper arm pain, followed by atrophy of the supraspinatus, infraspinatus, and deltoid muscles, with winging of the scapula. Treatment is usually conservative with immobilization and physical therapy, and recovery is expected within 6 to 24 months.

Birth brachial plexus injury most commonly involves the upper plexus (C5-C6, 50%), which luckily has a good prognosis (90% chance of spontaneous recovery). Pure lower plexus injuries (C7-T1) are rare (2%) and have a poor prognosis. Birth injuries are usually observed for 3 to 9 months and then explored surgically if no antigravity function ensues.

Traumatic Injury and Treatment Strategy

It is important to discern the mechanism of traumatic injury to decide on the treatment strategy. Penetrating or lacerating injuries are ideally explored in 24 to 48 hours if they are sharp, to be reapproximated with tension-free end-to-end anastomosis using epineurial sutures. Nonsharp injuries are usually left alone until the wound is healed. Ballistic injuries are initially treated conservatively, and delayed intervention (around 2–5 months, allowing the edges of injuries to settle) is considered if the deficits do not improve and if they involve the upper brachial plexus, which has a better prognosis than the lower plexus.

Surgical options may include grafting or nerve transfers (neurotization procedures). Stretch (traction) injury is more likely to affect the posterior and lateral cords. If the stretch injury is incomplete, improvement is expected, and follow-up with repeat electromyography (EMG) is recommended. If 9 months and then explored surgically if no antigravity function ensues.

<table>
<thead>
<tr>
<th>Erb Palsy (Upper BPI)</th>
<th>Klumpke Palsy (Lower BPI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Level</strong></td>
<td>C5-C6 ± C7</td>
</tr>
<tr>
<td><strong>Mechanism</strong></td>
<td>Downward shoulder pull widening angle between neck/shoulder</td>
</tr>
<tr>
<td><strong>Paralyzed muscles</strong></td>
<td>Deltoid, biceps, rhomboids, brachioradialis, supraspinatus, infraspinatus, ± supinator</td>
</tr>
<tr>
<td><strong>Clinical sign</strong></td>
<td>“Waiter tip”</td>
</tr>
<tr>
<td><strong>Horner syndrome</strong></td>
<td>Uncommon</td>
</tr>
<tr>
<td><strong>Prognosis</strong></td>
<td>More favorable</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Possible neurotization</td>
</tr>
</tbody>
</table>

**Figure 3.** Sagittal (A), axial (B), and coronal (C) T2-weighted MR images of the cervical spine and brachial plexus of a 29-year-old man performed 2 days after a motor vehicle accident. He had a complete C5 and C6 and partial C7 brachial plexus palsies. His follow-up EMG and nerve conduction studies showed denervation potentials in the paraspinal muscles and involvement of the rhomboid muscles. He had severe neuropathic pain but no Horner syndrome because the injury was above T1. His injury was consistent with severe traction (B, small arrow) probably causing nerve avulsion and a preganglionic injury. There is increased fluid signal around the roots on the left side (C, large arrow). (Courtesy of Semmes-Murphey Clinic.)
complete transection is suspected, it is advised to wait at least 3 weeks so the injured edges will be better defined. Sometimes a neuroma in continuity is encountered, and depending on its length, neurolysis and/or grafting may be considered. Other traumatic mechanisms include a displaced first rib fracture or compression by a hematoma. Such causes of mechanical compression can be reversible with early surgical intervention.

Readings


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To activate your online access, click “Register” at the top right corner of the website.
1. Parsonage-Turner syndrome usually involves the lower brachial plexus.
   
   True or False?

2. The brachial plexus roots always originate from C5-T1.
   
   True or False?

3. The suprascapular nerve is a branch of the superior trunk of the brachial plexus.
   
   True or False?

4. The paraspinal muscles are denervated in preganglionic injuries.
   
   True or False?

5. Upper brachial plexus injuries have a better prognosis than lower brachial plexus injuries.
   
   True or False?

6. Horner syndrome can occur in upper brachial plexus injuries (C5-6).
   
   True or False?

7. The posterior cord of the brachial plexus gives off the median nerve branch.
   
   True or False?

8. It is advised to wait a few weeks until the nerve edges are defined in sharp brachial plexus injuries.
   
   True or False?

9. The thoracic outlet syndrome typically involves the lower plexus, combined with symptoms of venous and arterial compression.
   
   True or False?

10. Chronic Erb palsy causes the waiter tip sign, where the shoulder is pathologically adducted and internally rotated and the elbow is extended.

   True or False?