

**Brachial Plexopathy (Traumatic)**

The brachial plexus is a network of nerves that comes from the spinal cord and controls muscle movements and sensation in the shoulder, arm and hand. Traumatic injuries to this network — most commonly caused by accidents or by childbirth (Erb’s palsy or obstetrical brachial plexus) — can leave the arm useless. Tumors, compression or irradiation can also injure the brachial plexus.

Trauma accounts for a large proportion of brachial plexopathies. The mechanism of an injury and the magnitude, rate, and direction of deforming forces ultimately determine the extent and location of a traumatic brachial plexopathy.

A lesion of the brachial plexus can result in motor, sensory, and sympathetic disturbances. Impairments can be transient, as in stinger or burner injuries in football players, or they may result in intractable palsy. Because of the changing arrangement of the brachial plexus as it progresses distally, injuries to it may result in diverse paralyses, anesthesias, and paresthesias, depending on the exact level of injury and the extent of injury to the various elements at that level.

**Anatomy**

The anterior ramification of the spinal nerves C5 to T1 combines to form the brachial plexus. C5 and C6 merge into the upper trunk, C7 forms the middle trunk, and C8 and T1 merge to form the lower trunk. Anterior divisions from the upper and middle trunks form the lateral cord. The medial cord is the anterior division of the lower trunk. Posterior divisions from all 3 trunks form the posterior cord. Terminal branches originate from the C5 root, trunks, and cords to supply the upper extremity and the shoulder girdle. The spinal nerves emerge from the vertebral foramina and pass between the anterior and middle scalenus; they then pass between the clavicle and the first rib, near the coracoid and humeral head. The plexus is relatively
tethered at the prevertebral fascia at its proximal aspect and by the auxiliary sheath in the mid arm.

**Diagnosis**

Symptoms of a traumatic brachial plexus injury are typically noticed soon after the injury, but the diagnosis of a brachial plexus injury may be delayed, often because other serious injuries need treatment more urgently. Patient evaluation should occur as early as possible (usually by three months) following injury. Read more about brachial plexus diagnosis

Brachial plexopathies may be difficult to accurately diagnose, even with a meticulous investigation. This is not only because the anatomic design of the plexus pose challenges, but also because the types of lesions and injuries that occur are frequently incomplete and complex. Even so, establishing a precise anatomic diagnosis and estimating the severity of the lesion is imperative for prognostic, surgical, and rehabilitative purposes.

**Pathophysiology**

In traumatic brachial plexopathy, nerve roots may be avulsed from the cord, or the plexus may be subject to traction or compression. Any injury that increases the distance between the relatively fixed points of the prevertebral fascia and the mid forearm may injure the brachial plexus. Traction or compression may result in ischemia, which initially damages the vasa vasorum. Severe compression injuries can result in intraneural hematomas, which can compress adjacent nerve tissue.

**Mortality/Morbidity**

Coexistent musculoskeletal or central nervous system injury, such as spinal cord injury (SCI) or traumatic brain injury (BI), is common after violent trauma and presents a diagnostic challenge.
• 80% of patients with severe traumatic brachial plexopathy had multiple traumas to the head and skeletal system.
• Root avulsion and contusions of the brachial plexus and cord, which are other frequently occurring coexistent, complicating factors, pose additional diagnostic and prognostic challenges.

Sex

In general, traumatic brachial plexopathy is more prevalent in men than in women because of an association with violent trauma and sports.

• Certain conditions, such as thoracic outlet syndrome (TOS), are statistically more common in women than in men.
• Other regional differences influence sex- and cause-related statistics.

Age

Because of an association with violent trauma and sports-related injuries, traumatic brachial plexopathy is most prevalent in males in their mid teens and in men in their early 30s.

Clinical

History taking should include inquiry into the mechanism of injury, as well as a description of patient symptoms. Common mechanisms of injury involve cervical extension, rotation, lateral bending, and depression or hyper abduction of the shoulder.

Patients should be queried about weakness, sensory loss, paresthesias and dysesthesias, and the location of symptoms in the arm.

Physical

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The physician should examine the cervical spine, shoulder, clavicle, scapula, and related joints for range of motion (ROM), alignment, and tender points. A thorough neurologic examination of the upper extremity should include manual muscle testing, sensory examination, and an evaluation of deep tendon reflexes.

- The site of injury can be accurately localized with a precise neurologic examination by using the correlative neuro anatomy.
- A sensory examination should include testing for light-touch sensation, pinprick sensation, 2-point discrimination, vibration sensation, and proprioception.
- In an anterior dislocation of the shoulder, the sensory distribution of the auxiliary and musculocutaneous nerves are tested to detect nerve injury in the early stages.
- Associated problems that require prompt attention can be identified with the following:
  - Evaluation of joint instability and scapular winging
  - Auscultation to detect hemidiaphragmatic paralysis
  - Observation of patterns of muscle weakness and/or atrophy, in which the injured side is compared with the uninvolved side
  - Testing for SCI and BI

**Causes**

As previously noted, a large proportion of brachial plexopathies are caused by trauma. The mechanism of traumatic injuries and the magnitude, rate, and direction of deforming forces ultimately determine the extent and location of the injury. Mechanisms include traction, penetrating injury, and crushing or compression.

Closed injuries, such as those caused by motor vehicle accidents, industrial accidents, and sports-related trauma, are more common in civilian life than in military life. Violent torsion of the upper limb, either upward or downward, may damage the plexus. Shrapnel...
injuries and blast injuries, as well as gunshot wounds and knife injuries to the neck or axilla, can cause lesions in the brachial plexus.

Iatrogenic injuries occur during surgery, particularly in procedures involving the following:

(1) Neck or shoulder
(2) Opening of the chest
(3) Regional anesthetic blocks
(4) Placement of cannulas.

Injuries to the brachial plexus of neonates may occur during birth, as a result of the strain placed on the plexus by a wide separation of the head and shoulder or by forced adduction of the shoulder joint during a difficult delivery.

Other Problems to Be Considered

- Traumatic root avulsion
- Anterior horn cell disorders
- Cerebrovascular accident (CVA)
- Peripheral neuropathy
- Entrapment syndromes of the upper extremity
- Iatrogenic injury - Injection and/or block, thoracotomy, tourniquet paralysis
- Sports injury - Stingers, burners
- Psychogenic paralysis
- Intraspinal and brachial plexus neoplasm
- Myopathy
- Neurodegenerative process
- Toxic process - Exposure to heavy metals, synthetic hydrocarbons, alcohol
- Infiltrative process
- Vasculitic process - Polyarteritis nodosa (PAN), systemic lupus
erythematous (SLE), diabetes
Hemorrhagic process in the spinal cord or nerve sheath
Immunogenic process - Human immunodeficiency virus (HIV) infection, transverse myelitis
Shoulder and scapulothoracic dislocation, fracture, tendinitis, or capsulitis.

**Lab Studies**

- Electro diagnosis has become a mainstay in the diagnostic evaluation of brachial plexopathies. Electro diagnostic tests provide physiologic data about the continuity of pathways and of lesion type and severity. Serial testing is helpful to determine prognosis.
- While positive waves and fibrillations (which indicate axonal injury) do not appear for several weeks after injury, sensory nerve action potentials (SNAPs) can be useful within days of injury to distinguish a presynaptic lesion from a postsynaptic lesion. With postsynaptic lesions, SNAPs are absent, whereas they are present with presynaptic ganglionic lesions.
- Somatosensory evoked potentials (SSEPs) are also useful to assess proximal lesions, such as root avulsions.

**Imaging Studies**

- Many peripheral nerve injuries can be associated with other soft-tissue or bone injuries that can be detected at radiography.
  - Radiographs of the injury site help to identify fractures or foreign bodies. For example, fractures of the cervical spine are frequently associated with brachial plexus injuries.
  - In cases of phrenic nerve paralysis, chest radiographs demonstrate unilateral elevation of the diaphragm.
  - Midhumeral fractures are associated with radial nerve injuries, and midforearm fractures of the ulna or radius.
are associated with median or ulnar nerve injuries, respectively.
- To rule out bony and ligamentous injuries, all patients with auxiliary nerve injury should initially undergo radiography of the shoulder and cervical spine.
- The resolution of the fine anatomic detail of soft tissue is better with magnetic resonance imaging (MRI) than with computed tomography (CT) scanning.
  - Conventional MRI is used to visualize normal and abnormal peripheral nerve structures.
  - Moreover, in a study by West and colleagues, MRI depicted signal intensity changes in denervated muscle as early as 4 days after clinical symptoms developed. With short-tau inversion recovery (STIR) techniques, signal intensity changes in the thenar muscles were demonstrated on MRI scans of 100% of the patients with clinically advanced carpal tunnel syndrome.
  - With neurapraxic nerve injuries, the signal intensity in the innervated muscles remains normal on STIR or T2-weighted images. Therefore, after a peripheral nerve injury, early MRI of the muscle can be useful in distinguishing a neurapraxic injury from more severe axonotmesis or neurotmesis.
- Because CT scanning and traditional MRI techniques have inherent limitations in their resolution and distinction of peripheral nerves from the surrounding structures, magnetic resonance neurography (MRN) has been developed.
  - MRN can depict normal and abnormal peripheral nerves in various regions of the body.
  - The injured peripheral nerve can be assessed by orienting the images along the course of the damaged nerve. For example, the loss of signal intensity on T2-weighted images indicates damage to the myelin sheath.
  - In addition, loss of water content in denervated nerves of the deep muscles can be assessed with MRN when needle electromyography (EMG) is difficult to perform.

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The predictive value of MRN in the diagnosis of peripheral nerve trauma has not yet been reliably established.

- CT scanning can be used in the investigation of occult fractures that are not depicted on plain radiographs.
- With myelography, CT scanning can be used to demonstrate root avulsion.

Other Tests

Clinical threshold testing can be used to evaluate sensory function in peripheral nerves. These tests can be used to determine the level of stimulus necessary to elicit a response.

Semmes-Weinstein monofilaments are fine filaments that exert a discrete amount of pressure on the fingertips. They are used to perform threshold testing. Vibratory senses can be assessed by means of clinical threshold testing with low (30 Hz) to high (256 Hz) frequencies.

Histologic Findings

At light microscopy, nerves injured with epineurectomy or crush mechanism have widespread fiber degeneration and myelin debris in the subperineurial region. The centrofascicular areas are relatively preserved compared with the subperineurial regions. The central vessels are preserved mostly within the centrofascicular area of the injured nerve. The thickness of myelin in the axons is decreased after injury, and the inter nodal length becomes more variable compared with its length before injury. A loss of cross-sectional area without a loss in the muscle fiber count begins within 1 week of denervation.

Treatment
The brachial plexus is a network of nerves that comes from the spinal cord and controls muscle movements and sensation in the shoulder, arm and hand. Brachial plexus injuries are caused by damage to those nerves. There are multiple types of brachial plexus injuries and treatment is determined by the type of injury.

Depending on local expertise, the rehabilitation program may be undertaken with a physical therapist and/or an occupational therapist. The goals are to preserve ROM, improve strength, and manage pain.

Patients should undergo physical therapy to maintain ROM and to optimize the recovery of motor function as muscle reinnervation occurs.

The goal of treatment is to return function to the structures supplied by the damaged nerves and to improve the patient’s quality of life. The injured nerve and the exogenous sources of nerve injury are treated.

At the onset of injury, early mobilization and icing are used. In the sub acute phase, therapy gradually progresses from passive to active motion and from assisted to active ROM, as tolerated.

Heat, ultrasonography, transcutaneous electrical nerve stimulation (TENS), interferential current stimulation, and/or electrical stimulation are used, depending on the predominant symptoms.

Cervical muscle strengthening and the correction of upper extremity muscle imbalances are included in the protocol as well.

The use of appropriate slings, the protection of extremities and joints, and the prevention of subluxation must be considered.

Cervical pillows or collars may be required for patients with combined lesions of the roots and plexus.

**Occupational Therapy**

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During occupational therapy efforts are concentrated on maintaining ROM in the shoulder; fabricating appropriate orthoses to support the function of the hand, elbow, and arm; and addressing edema control and sensory deficits, with testing and therapy.

Occupational therapy may address issues related to the patient’s ability to write, type, and find alternate ways of communicating.

Additionally, occupational therapy provides help with retraining for activities of daily living (ADLs), including the use of 1-arm techniques, adaptive equipment, and self-ranging and strengthening exercises.

**Recreational Therapy**

Recreational therapy should address compensatory strategies and activities that can substitute for altered or lost function in extremities that were required for recreation prior to injury.

**Medical Issues/Complications**

- Complications may include intractable pain syndromes, such as persistent neuropathy and complex regional pain syndrome type 2 (CRPS II or causalgia), skin damage and infection, significant muscle atrophy, contractures and capsulitis, subluxations, sensory loss, osteopenia, heterotopic ossification, myofascial pain, and depression and anxiety.
- Bone dislocation with neurologic deficit requires prompt anatomic reduction to prevent irreversible nerve damage.
- The use of analgesics can help patients control pain from nerve injuries. Steroids may help to decrease endoneurial edema associated with nerve injury.
- Hyperbaric oxygen decreases vascular compromise of the vasa nervorum, as well as endoneurial edema and pressure. Hyperbaric oxygen is an approved adjunctive treatment for acute traumatic ischemic reperfusion injury.
- Ciliary neurotrophic factor (CNTF), which enhances motor neuron survival in vivo and in vitro, is in the investigational stage.

**Surgical Intervention**

Surgery is reserved for patients in whom symptoms persist despite appropriate conservative treatment. Two important issues to consider before surgery are as follows:

(1) Whether function can be obtained after the nerve is repaired

(2) Whether the potential benefit to the patient outweighs the surgical risks, costs, and loss of productivity.

The timing of surgery is important as well.

*Other factors to consider are as follows:*

- In clean lacerating injuries in which the nerve ends are visible in the wound or when clinical examination reveals obvious motor and sensory deficits from the laceration, immediate primary repair may be indicated.
- In blunt transections resulting from lacerations, delayed repair has a better surgical result.
- Injuries without evidence of early spontaneous recovery, such as those caused by bullets, crushing blows, traction, fractures, or injections, are explored several months after the injury.
- Brachial plexus stretches or contusions are observed for 4 months. If no evidence of recovery is present, the plexus is explored.
- Nerve or tendon transfers may be necessary if nerve repair is unsuccessful.
  - Brachial plexus injuries are not always reparable. In such cases, neurotizations or nerve transfers may offer a better functional outcome.
Two criteria that must be present before fascicular repair or interfascicular grafting is considered are:

- The fascicular bundle must be large enough for suturing.
- The bundle must be sharply localized or sufficiently well defined so that it can be identified and mobilized for repair.

The spinal accessory or long thoracic nerve can be grafted onto distal arm nerve trunks, with some improvement in elbow flexion.

Intravascular care with proper axial orientation of the fascicles, hemostasis, suture material, and suture line tension leads to better outcomes. Tension of the suture line and inadequate preparation of the nerve stumps are two leading causes of regenerative failure across the suture site (resulting in poor recovery of nerve function).

Surgical repairs are most effective within 3 months of the injury. Surgical delays in excess of 5 months dramatically decrease the rate of functional return.

When repair does not provide adequate results, planned tendon transfers can increase extremity function.

Rarely, in cases of a complete multilevel injury (eg, flail injury, anesthetic arm), amputation may result in a better functional outcome, because the patient can use the extremity with an appropriate prosthesis. However, the result may be less cosmetically pleasing than would that obtained with other approaches.

A pain management strategy is of great importance in improving the patient's ability to cope and function and in improving his/her quality of life.

Other Treatment
• In cases of CRPS II, sympathetic (ie, stellate) blockade may be required, along with the appropriate combination of neuropathic and narcotic medications.
• For incomplete, painful injuries, and especially in cases of CRPS II, the use of a spinal cord stimulator on a trial basis may be beneficial. If this trial is successful, the stimulator may be implanted.
• Implantable peripheral nerve stimulators have also been successfully used in some centers.
• The use of an implantable intrathecal device (eg, pump) may be considered in cases in which the employment of oral medications, therapy, and a spinal cord stimulator fail.

Medication

Nonsteroidal anti-inflammatory drugs (NSAIDs) and neuropathic pain medications are most commonly used in the treatment of traumatic brachial plexopathy, depending on the symptoms and the length of time since the injury’s occurrence. During the acute phase, narcotic analgesics may also be necessary, but they should not be used for long-term pain management. Narcotic medications are also indicated in the acute postoperative period.

Neuropathic pain medications are useful for the relief of dysesthetic pain in the acute and chronic phases. There is no drug of choice, and medications often must be tried in serial fashion to find one that provides optimal relief for the patient.

Drug Category: Nonsteroidal anti-inflammatory drugs

After acute injury, NSAIDs are particularly helpful in relieving pain related to the injury, including injuries involving soft tissues, such as muscles and ligaments.

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Celecoxib (Celebrex)</th>
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<tbody>
<tr>
<td>Description</td>
<td>Inhibits primarily COX-2. COX-2 is</td>
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considered an inducible isoenzyme, induced during pain and inflammatory stimuli. Inhibition of COX-1 may contribute to NSAID GI toxicity. At therapeutic concentrations, COX-1 isoenzyme is not inhibited; thus, GI toxicity may be decreased. Seek the lowest dose for each patient.

**Adult Dose**
200 mg/d PO qd; alternatively, 100 mg PO bid

**Pediatric Dose**
Not established

**Contraindications**
Documented hypersensitivity

**Interactions**
Coadministration with fluconazole may increase celecoxib plasma concentrations because of inhibition of celecoxib metabolism; coadministration with rifampin may decrease celecoxib plasma concentrations

**Pregnancy**
B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals
D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus

**Precautions**
May cause fluid retention and peripheral edema; caution in compromised cardiac function, hypertension, conditions predisposing patient to fluid retention; caution in severe heart failure and hyponatremia (may cause deterioration in circulatory hemodynamics); NSAIDs may mask usual signs of infection; caution in existing controlled infections; evaluate symptoms and signs suggesting liver dysfunction or abnormal liver laboratory
<table>
<thead>
<tr>
<th><strong>Drug Name</strong></th>
<th>Naproxen (Naprosyn, Aleve)</th>
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<tbody>
<tr>
<td><strong>Description</strong></td>
<td>For relief of mild to moderate pain; naproxen inhibits inflammatory reactions and pain by reducing the activity of cyclooxygenase, which decreases prostaglandin synthesis.</td>
</tr>
<tr>
<td><strong>Adult Dose</strong></td>
<td>500 mg PO followed by 250 mg PO q6-8h; not to exceed 1.25 g/d</td>
</tr>
<tr>
<td><strong>Pediatric Dose</strong></td>
<td>&lt;2 years: Not established &gt;2 years: 2.5 mg/kg/dose PO; not to exceed 10 mg/kg/d</td>
</tr>
<tr>
<td><strong>Contraindications</strong></td>
<td>Documented hypersensitivity; peptic ulcer disease; recent GI bleeding or perforation; renal insufficiency</td>
</tr>
<tr>
<td><strong>Interactions</strong></td>
<td>Coadministration with aspirin increases risk of serious NSAID-related adverse effects; probenecid may increase concentrations and, possibly, toxicity of NSAIDs; may decrease effect of hydralazine, captopril, and beta blockers; may decrease diuretic effects of furosemide and thiazides; may increase PT when patient is taking anticoagulants (instruct patients to watch for signs of bleeding); may increase risk of methotrexate toxicity; phenytoin levels may be increased when administered concurrently</td>
</tr>
<tr>
<td><strong>Pregnancy</strong></td>
<td>B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus</td>
</tr>
<tr>
<td><strong>Precautions</strong></td>
<td>Acute renal insufficiency, interstitial</td>
</tr>
</tbody>
</table>
nephritis, hyperkalemia, hyponatremia, and renal papillary necrosis may occur; patients with preexisting renal disease or compromised renal perfusion risk acute renal failure; leukopenia occurs rarely, is transient, and usually returns to normal during therapy; persistent leukopenia, granulocytopenia, or thrombocytopenia warrants further evaluation and may require discontinuation of drug

**Drug Category: Anticonvulsants**

The use of certain antiepileptic drugs, such as the GABA analogue gabapentin (Neurontin), has proven helpful in some cases of neuropathic pain. Anticonvulsants have central and peripheral anticholinergic effects, as well as sedative effects, and block the active reuptake of norepinephrine and serotonin. The multifactorial mechanism of analgesia could include improved sleep, an altered perception of pain, and an increased pain threshold. The efficacy of these drugs can be potentiated with the concomitant use of opiates and NSAIDS. Rarely should these drugs be used in the treatment of acute pain, because they may require a few weeks to become effective.

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Gabapentin (Neurontin)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Description</strong></td>
<td>Has anticonvulsant properties and antineuralgic effects; however, the exact mechanism of action is unknown. Gabapentin is structurally related to GABA, but it does not interact with GABA receptors. Titration to effect can take place over several days (300 mg on day 1, 300 mg bid on day 2, and 300 mg tid on day 3).</td>
</tr>
<tr>
<td><strong>Adult Dose</strong></td>
<td>Day 1: 100 mg PO tid or 300 mg qhs</td>
</tr>
</tbody>
</table>
Day 2: 400 mg PO tid over 3 d and titrate prn; not to exceed 1200 mg PO qid

**Pediatric Dose**

<table>
<thead>
<tr>
<th>Age</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;12 years</td>
<td>Not established</td>
</tr>
<tr>
<td>&gt;12 years</td>
<td>Administer as in adults</td>
</tr>
</tbody>
</table>

**Contraindications**

Documented hypersensitivity

**Interactions**

Antacids may significantly reduce bioavailability (administer at least 2 h following antacids); may significantly increase norethindrone levels

**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

**Precautions**

Caution in severe renal disease

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**Drug Category: Tricyclic antidepressants**

This is a complex group of drugs that have central and peripheral anticholinergic effects, as well as sedative effects. They have central effects on pain transmission. Tricyclic antidepressants block the active reuptake of norepinephrine and serotonin.

**Drug Name**

Nortriptyline (Pamelor)

**Description**

Has demonstrated effectiveness in the treatment of chronic pain. By inhibiting the reuptake of serotonin and/or norepinephrine by the presynaptic neuronal membrane, this drug increases the synaptic concentration of these neurotransmitters in the central nervous system. Pharmacodynamic effects, such as the desensitization of adenyl cyclase and the down-regulation of beta-adrenergic receptors and serotonin receptors, also appear to play a
<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Doxepin (Sinequan, Adapin)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Description</strong></td>
<td>Inhibits histamine and acetylcholine activity; doxepin has proven useful in the treatment of various forms of depression associated with chronic and neuropathic pain.</td>
</tr>
<tr>
<td><strong>Adult Dose</strong></td>
<td>10-150 mg/d PO qhs or divided bid/tid</td>
</tr>
</tbody>
</table>
| **Pediatric Dose**| <12 years: Not recommended  
>12 years: 25-50 mg/d PO qhs or bid/tid; increase gradually to 100 mg/d |
| **Contraindications** | Documented hypersensitivity; urinary retention; acute recovery phase following myocardial infarction; glaucoma |
| **Interactions**  | Decreases antihypertensive effects of |

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Nortriptyline</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Role</strong></td>
<td>Inhibits histamine and acetylcholine activity; doxepin has proven useful in the treatment of various forms of depression associated with chronic and neuropathic pain.</td>
</tr>
<tr>
<td><strong>Adult Dose</strong></td>
<td>25 mg PO tid/qid; not to exceed 150 mg/d</td>
</tr>
</tbody>
</table>
| **Pediatric Dose**| <25 kg: Not established  
25-35 kg: 10-20 mg/d PO  
35-54 kg: 25-35 mg/d PO  
>54 kg: Administer as in adults |
| **Contraindications** | Documented hypersensitivity; narrow-angle glaucoma; do not administer to patients that have taken MAOIs in past 14 days |
| **Interactions**  | Cimetidine may increase Nortriptyline levels when used concurrently; may increase prothrombin time in patients taking warfarin |
| **Pregnancy**     | D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus |
| **Precautions**   | Caution in cardiac conduction disturbances and history of hyperthyroidism, renal or hepatic impairment; because of pronounced effects in cardiovascular system, best to avoid in elderly |

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clonidine but increases effects of sympathomimetics and benzodiazepines; effects of desipramine increase with phenytoin, carbamazepine, and barbiturates

<table>
<thead>
<tr>
<th>Pregnancy</th>
<th>C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Precautions</td>
<td>Caution in cardiovascular disease, conduction disturbances, seizure disorders, urinary retention, hyperthyroidism, and patients receiving thyroid replacement</td>
</tr>
</tbody>
</table>

**Drug Category: Analgesics**

Narcotics are indicated in the acute injury period and in the postoperative period should reconstructive surgery be required. In rare cases in which patients require long-term opioid use, these patients should use scheduled, longer-acting medications, such as methadone.

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Methadone (Dolophine)</th>
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<tbody>
<tr>
<td><strong>Description</strong></td>
<td>Used in the management of severe pain. Methadone inhibits ascending pain pathways, diminishing the perception of and response to pain.</td>
</tr>
<tr>
<td><strong>Adult Dose</strong></td>
<td>2.5-10 mg PO/IM/SC q3-8h prn; increase to a maintenance dose of 5-20 mg q6-8h</td>
</tr>
<tr>
<td><strong>Pediatric Dose</strong></td>
<td>0.7 mg/kg/d PO/IM/SC divided q4-6h prn, not to exceed 10 mg/dose</td>
</tr>
<tr>
<td><strong>Contraindications</strong></td>
<td>Documented hypersensitivity; bronchial asthma or increased intracranial pressure</td>
</tr>
<tr>
<td><strong>Interactions</strong></td>
<td>Phenytoin, rifampin, and pentazocine may decrease blood levels; phenothiazines, tricyclic antidepressants, MAOIs, and CNS</td>
</tr>
<tr>
<td><strong>Drug Name</strong></td>
<td>Oxycodone (OxyContin, Roxicodone, OxyIR)</td>
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<tr>
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</tr>
<tr>
<td><strong>Description</strong></td>
<td>Indicated for the relief of moderate to severe pain.</td>
</tr>
</tbody>
</table>
| **Adult Dose** | Immediate release: 5 mg PO q6h prn  
Controlled release: 10 mg PO bid |
| **Pediatric Dose** | Immediate release:  
<6 years: Not established  
6-12 years: 1.25 mg PO q6h prn  
>12 years: 2.5 mg PO q6h prn  
Controlled release: Not established |
| **Contraindications** | Documented hypersensitivity |
| **Interactions** | Phenothiazines may antagonize analgesic effects; MAOIs, general anesthesia, CNS depressants, and tricyclic antidepressants may increase toxicity |
| **Pregnancy** | B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals  
D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus |
<p>| <strong>Precautions</strong> | Caution in COPD, emphysema, and renal insufficiency |
| <strong>Drug Name</strong> | Oxycodone and acetaminophen (Percocet) |</p>
<table>
<thead>
<tr>
<th><strong>Description</strong></th>
<th>Drug combination indicated for the relief of moderate to severe pain.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adult Dose</strong></td>
<td>1-2 tab or cap PO q4-6h prn pain</td>
</tr>
<tr>
<td><strong>Pediatric Dose</strong></td>
<td>0.05-0.15 mg/kg/dose oxycodone PO; not to exceed 5 mg/dose of oxycodone PO q4-6h prn</td>
</tr>
<tr>
<td><strong>Contraindications</strong></td>
<td>Documented hypersensitivity</td>
</tr>
<tr>
<td><strong>Interactions</strong></td>
<td>Phenothiazines may decrease analgesic effects of this medication; toxicity increases with coadministration of either CNS depressants or tricyclic antidepressants</td>
</tr>
<tr>
<td><strong>Pregnancy</strong></td>
<td>C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus</td>
</tr>
<tr>
<td><strong>Precautions</strong></td>
<td>Duration of action may increase in the elderly; be aware of total daily dose of acetaminophen patient is receiving; not to exceed 4000 mg of acetaminophen per 24 h; higher doses may cause liver toxicity</td>
</tr>
<tr>
<td><strong>Drug Name</strong></td>
<td>Fentanyl citrate (Duragesic)</td>
</tr>
<tr>
<td><strong>Description</strong></td>
<td>Potent narcotic analgesic with much shorter half-life than morphine sulfate. Fentanyl citrate is the DOC for conscious sedation analgesia. It is ideal for analgesic action of short duration during anesthesia and for the immediate postoperative period. Fentanyl citrate is excellent for pain management and sedation with short duration (30-60 min); it is easy to titrate. The drug is easily and quickly reversed with naloxone. After the initial dose, subsequent doses</td>
</tr>
</tbody>
</table>
should not be titrated more frequently than q3h or q6h thereafter. When the transdermal dosage form used, controlled with 72-h dosing intervals effective in most patients. However, some patients require 48-h dosing intervals.

<table>
<thead>
<tr>
<th>Adult Dose</th>
<th>Emergency: 0.5-2 mcg/kg/dose IV/IM Analgesia: 0.5-1 mcg/kg/dose IV/IM q30-60min Transdermal: Apply a 25 mcg/h system q48-72h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pediatric Dose</td>
<td>&lt;2 years: 2-3 mcg/kg/dose IV/IM q30-60min 2-12 years: 1-2 mcg/kg/dose IV/IM q60min &gt;12 years: Administer as in adults</td>
</tr>
<tr>
<td>Contraindications</td>
<td>Documented hypersensitivity; hypotension or potentially compromised airway where it would be difficult to establish rapid airway control</td>
</tr>
<tr>
<td>Interactions</td>
<td>Phenothiazines may antagonize analgesic effects of opiate agonists; TCAs may potentiate adverse effects of fentanyl when both drugs used concurrently</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus D - Fetal risk shown in humans; use only if benefits outweigh risk to fetus</td>
</tr>
<tr>
<td>Precautions</td>
<td>Caution in hypotension, respiratory depression, constipation, nausea, emesis, and urinary retention; idiosyncratic reaction, known as chest wall rigidity syndrome, may require neuromuscular blockade to increase ventilation</td>
</tr>
<tr>
<td><strong>Drug Name</strong></td>
<td>Hydrocodone and acetaminophen (Lorcet)</td>
</tr>
<tr>
<td><strong>Description</strong></td>
<td>Drug combination indicated for moderate to severe pain.</td>
</tr>
<tr>
<td><strong>Adult Dose</strong></td>
<td>1-2 tab or cap PO q4-6h prn pain</td>
</tr>
<tr>
<td><strong>Pediatric Dose</strong></td>
<td>&lt;12 years: 10-15 mg/kg/dose based on acetaminophen PO q4-6h prn; not to exceed 2.6 g/d acetaminophen; &gt;12 years: 750 mg acetaminophen PO q4h; not to exceed 10 mg hydrocodone bitartrate per dose or 5 doses/24 h</td>
</tr>
<tr>
<td><strong>Contraindications</strong></td>
<td>Documented hypersensitivity; high altitude cerebral edema (HACE) or elevated intracranial pressure (ICP)</td>
</tr>
<tr>
<td><strong>Interactions</strong></td>
<td>Coadministration with phenothiazines may decrease analgesic effects; toxicity increases with CNS depressants or tricyclic antidepressants</td>
</tr>
<tr>
<td><strong>Pregnancy</strong></td>
<td>C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus</td>
</tr>
<tr>
<td><strong>Precautions</strong></td>
<td>Tablets contain metabisulfite, which may cause hypersensitivity; caution in patients dependent on opiates, because substitution may result in acute opiate-withdrawal symptoms; caution in severe renal or hepatic dysfunction</td>
</tr>
</tbody>
</table>

<p>| <strong>Drug Name</strong> | Tramadol (Ultram) |
| <strong>Description</strong> | Inhibits ascending pain pathways, altering perception of and response to pain. Tramadol also inhibits the reuptake of norepinephrine and serotonin. |
| <strong>Adult Dose</strong> | 50-100 mg PO q4-6h; not to exceed 400 |</p>
<table>
<thead>
<tr>
<th>mg/d</th>
</tr>
</thead>
</table>

**Pediatric Dose**

Not established

**Contraindications**

Documented hypersensitivity; opioid-dependent patients; concurrent use of MAOI or within 14 days; use of SSRIs, TCAs, opioids, acute alcohol intoxication

**Interactions**

Significantly decreases effects of carbamazepine, cimetidine increases toxicity, risk of serotonin syndrome with coadministration of antidepressants

**Pregnancy**

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

**Precautions**

Can cause dizziness, nausea, constipation, sweating, pruritus; additive sedation with alcohol and TCAs; abrupt discontinuation can precipitate opioid withdrawal symptoms; adjust dose in liver disease, myxedema, hypothyroidism, hypoadrenalism; pregnancy, breast-feeding; seizure; development of tolerance or dependency with extended use

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**Further Inpatient Care**

- If physical therapy is not initiated promptly after surgery, denervation can occur and can result in muscle atrophy and fibrosis, joint stiffness, motor endplate atrophy, and trophic skin changes.
- Traditional treatment, which involves several weeks of immobilization is not advocated. Instead, the use of a short period to allow healing and adequate strengthening of the repair site is advised.
• Repairs (nerve transfer/neurotization, as well as tendon transfer) are protected by means of relaxed joint posturing for about 3 weeks.
• To prevent disruption of the sutures at the repair site, the patient should avoid strenuous physical activity.
• In nerve transfers, the extremity is immobilized for 4 weeks after surgery, at which time physical therapy is initiated.
• Postoperative clinical examinations are performed every 3 months for the first 2 years after surgery and every 6 months after that.
• At each postoperative visit, the ROM, strength, and sensation in the treated area should be tested, and the results should be documented.

Further Outpatient Care

• Continuation of physical therapy and/or occupational therapy and follow-up with a surgeon and/or orthotist may be needed.
• Vocational rehabilitation and modifications at home and/or work are also assessed.
• In some cases, repeated electro diagnostic evaluations may be required for prognostication and further treatment planning. These tests can be used to detect early signs of muscle reinnervation several months before clinically evident muscle contractions appear.
• A variety of medications may be required, mainly for the management of associated painful states.

Deterrence

Measures that the patient can use to prevent setbacks and further damage include the following:

  o Protecting the damaged limb from repeat injury and extremes of motion
o Maintaining the functional ROM
o Strengthening muscles in the cervical region and limbs
o Making appropriate modifications in the workplace and/or at home

Complications

Late complications may include the following:

o Pain syndromes, such as persistent neuropathy, neuroma, and CRPS II
o Skin damage and infection
o Significant muscle atrophy
o Contracture and capsulitis
o Subluxation
o Sensory loss
o Osteopenia
o Heterotopic ossification
o Myofascial pain
o Depression and anxiety

Prognosis

The outcome and prognosis of acute injury varies widely, depending on the type and etiology of injury and the timing of therapy.

o The extent of injury to neural tissue and the age and medical status of the injured patient are important factors that influence the outcome.
o Patient compliance and motivation for recovery can also have an important effect on the overall success of therapy.
  • With mild neurapraxic lesions, spontaneous recovery may occur only days or weeks after the trauma has occurred.
  • Following a gunshot wound, spontaneous recover may occur as late as 11 months later.

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• Recovery from axonometric injuries usually occurs over months.
  o In axonotmesis, although axons regenerate, functional recovery depends on the associated injuries, the amount of healthy proximal axon that remains after injury, and the age of the patient.
  o Recovery is usually complete unless the injury is so proximal that atrophy of the motor endplate or sensory receptor occurs before the axon can grow back to these organs.
  o In cases of a coexisting root avulsion, the above scenario of a very proximal lesion, resulting in atrophy of the motor endplate or sensory receptor, may be possible. Therefore, healing may be greatly delayed or incomplete.
• In neurotmesis, regeneration occurs, but function rarely returns to its pre-injury level.
• Generally, the rate of spontaneous recovery after shotgun wounds is lower than it is with other mechanisms.
• Neural injuries associated with fractures have a greater incidence of spontaneous resolution; generally, recovery is less common with neural injuries secondary to dislocations.
• Lesions resulting from shoulder dislocations heal within 12-45 weeks, depending on severity of the dislocation and, consequently, the type and extent of the associated neural injury or injuries.

Patient Education

Educating the patient, family, and rehabilitation team, as well as medical practitioners involved in the patient’s post discharge care, may have several benefits.

  o It facilitates the coordination and planning of services.
  o It hastens the implementation of appropriate interventions.
• It results in a better recovery.

Of equal importance is addressing the associated psychological factors, with the aim of improving the following:

• The patient’s mood stability
• The patient’s coping skills
• Family functioning
• Pain management
• Patient motivation
• Patient participation in therapy
• Overall outcome

Medical Pitfalls

• Failing to consider an injury of the cervical spine or spinal cord or a traumatic BI can delay early intervention and result in unwanted residual, long-term sequelae.
• In the initial assessment after a sports-related injury, the sideline personnel and physician should maintain a healthy degree of suspicion with regard to underlying spine injury or concussion. For example, with persistent symptoms of a burner injury, or post-concussive state, a complete assessment may be needed to prevent premature return to play.
• Overlooking a brachial plexus injury can lead to further damage that may persist.
• The precise localization of the lesion on electro diagnostic studies and the determination of the appropriate prognosis can be challenging in the initial post injury period or when multiple structures and/or levels are involved.
• Repeat study or the use of an additional diagnostic investigation (imaging or other) may be considered in cases in which there is poor functional recovery.