
EVIDENCE-BASED MEDICINE

Evidence-Based Interventional Pain Medicine
According to Clinical Diagnoses

22. Traumatic Plexus Lesion

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■ **Abstract:** Pain, motor, and sensory deficits characterize patients with a traumatic lesion of the brachial plexus. Frequently, more severe injuries co-exist that require immediate surgical attention.

Early rehabilitation and physical therapy are the cornerstones of treatment. Pharmacological management can be difficult. Surgical reconstruction is frequently advised when nerves are disrupted. The results, mostly from small historical reports, vary greatly. Neurostimulation may have an additional beneficial effect, especially if the pathophysiology of nociception and neuropathic pain becomes evident in these complex patients. ■

Key Words: evidence-based medicine, traumatic plexus lesion, spinal cord stimulation, brain stimulation, surgery

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INTRODUCTION

This review on traumatic plexus lesions is part of the series “Evidence-Based Interventional Pain Medicine according to clinical diagnoses.” Recommendations formulated in this article are based on “Grading strength of recommendations and quality of evidence in clinical guidelines” described by Guyatt et al.,¹ and adapted by van Kleef et al.² in the editorial accompanying the first article of this series (Table 1). The latest literature update was performed in March 2010.

Traumatic brachial plexus lesions are the most common form of plexus injuries. This syndrome is frequently the result of high-impact trauma in young male adults, as is often observed following a motorcycle accident or industrial injury. It can be caused by damage of the cervical vertebrae, the clavicle, and the humerus.

An injury due to trauma of the lumbosacral plexus may also result from a serious accident, but this occurs less frequently. The lumbosacral plexus can also be involved after a hemorrhage in the pelvic retroperitoneum, or as a result of tumor spread. The symptoms mainly occur in the thigh and around the knee region.

Table 1. Summary of Evidence Scores and Implications for Recommendation

Score	Description	Implication
1 A+	Effectiveness demonstrated in various RCTs of good quality. The benefits clearly outweigh risk and burdens	Positive recommendation
1 B+	One RCT or more RCTs with methodologic weaknesses, demonstrate effectiveness. The benefits clearly outweigh risk and burdens	
2 B+	One or more RCTs with methodologic weaknesses, demonstrate effectiveness. Benefits closely balanced with risk and burdens	
2 B±	Multiple RCTs, with methodologic weaknesses, yield contradictory results better or worse than the control treatment. Benefits closely balanced with risk and burdens, or uncertainty in the estimates of benefits, risk and burdens.	Considered, preferably study-related
2 C+	Effectiveness only demonstrated in observational studies. Given that there is no conclusive evidence of the effect, benefits closely balanced need with risk and burdens	
0	There is no literature or there are case reports available, but these are insufficient to suggest effectiveness and/or safety. These treatments should only be applied in relation to studies.	Only study-related
2 C-	Observational studies indicate no or too short-lived effectiveness. Given that there is no positive clinical effect, risk and burdens outweigh the benefit	Negative recommendation
2 B-	One or more RCTs with methodologic weaknesses, or large observational studies that do not indicate any superiority to the control treatment. Given that there is no positive clinical effect, risk and burdens outweigh the benefit	
2 A-	RCT of a good quality which does not exhibit any clinical effect. Given that there is no positive clinical effect, risk and burdens outweigh the benefit	

RCT randomized controlled trial.

Due to the low incidence of the latter, only brachial plexus lesions will be addressed here.

Anatomically, the brachial plexus consists of the five roots (radices) stemming from the (C5 to T1 dermatomes, which ultimately form the five major peripheral nerves of the arm (musculocutaneous nerve, axillary nerve, radial nerve, median nerve, and ulnar nerve). Therefore, clinical and anatomical distinctions exist between superior (C5 to C7) and inferior (C8 to Th1) brachial plexus lesions. Majority of brachial plexus injuries result from a combination of traction and avulsion caused by the trauma itself. Penetrating trauma and surgical interventions can also lead to a disruption of the nerves. Anatomically, brachial plexus injuries can be stratified according to location: preganglionic (ie, nerve root avulsion from the spinal cord), postganglionic, or combination lesions. Postganglionic lesions can be further classified into nerve disruption and lesions in continuity. Lesions located between the spinal cord and (proximal) ganglion can result in particularly debilitating pain complaints.^{3,4}

I. DIAGNOSIS

I.A HISTORY

In addition to the (often severe) neurologic impairment, neuropathic pain in the arm/hand area is the main complaint in 30% to 90% of people with a traumatic brachial plexus lesion (de-afferentation pain) (Figure

1). In the presence of a “preganglionic lesion,” the incidence of serious pain increases to approximately 90%.³ In one observational study involving 22 patients with signs of postconcussion syndrome, in the absence of severe brain damage secondary to whiplash injury, half had neurologic evidence of brachial plexus injury.⁵

In those patients where the radices of the nerves are disconnected from the spinal cord (root avulsion), severe pain usually occurs immediately following the injury, although in some cases it can arise after a pain-free interval.^{6,7} The initial continuous “background pain” is often described as “burning,” “shooting,” or “stabbing” in quality, but may later be exacerbated by superimposed paroxysms. The pain is usually worst in the distal parts of the arm and hand, typically in a nondermatomal distribution. The use of validated questionnaires designed to distinguish between neuropathic and nociceptive pain may be useful for diagnostic, prognostic, and therapeutic purposes.⁸ Serious sensory and/or motor loss confirms the diagnosis (MRC scale) (Medical Research Council; see Table 2). Psychosocial problems in the context of chronic pain and disability can also be influenced by cognitive disorders secondary to head trauma.

I.B PHYSICAL EXAMINATION

The results of neurologic examination depend on the location of the lesion (Table 3).^{4,9} Injury to the upper

Table 2. MRC Grading for Muscle Strength

0	Total paralysis, no contraction
1	Flicker contraction, no movement in joint
2	Muscle contraction with active motion with gravity eliminated
3	Full range of motion against gravity
4	Full range of motion against gravity with some resistance
5	Full range of motion against gravity with maximum resistance for that muscle

region of the plexus generally results in extensive loss of function in the proximal part of the arm and shoulder girdle, with motor function in the hand remaining unaltered or partially intact. By contrast, damage to the lower part of the plexus typically leads to serious loss of hand function, while the sensory losses are less extensive. Muscle strength can be determined by the MRC grading scale, with a correlation existing between the degree of motor dysfunction and the severity of injury (Table 2). Vegetative changes in the arm and hand often arise because of the trauma. A Horner's symptom on the affected side is indicative of a plexus lesion in the proximal region of roots C8 to T1. Combined damage to the plexus and spinal cord may lead to a difficult and time-consuming diagnostic process.^{10,11}

I.C ADDITIONAL TESTS

In cases of major trauma, imaging can differentiate preganglionic from postganglion lesions, a distinction that serves a vital role in guiding treatment. Conventional magnetic resonance imaging (MRI) can detect signal changes in the spinal cord (present in approximately 20% of preganglionic injuries), which may indicate edema or hemorrhage in the acute phase and myelomalacia in the chronic phase. Contrast enhancement of intradural nerve roots may signify functional impairment despite morphological continuity. Abnormal enhancement of paraspinal muscles on MRI can also serve as a sensitive indicator of root avulsion.

Computed tomographic (CT)-myelography facilitates visual separation of ventral and dorsal nerve roots, and identification of intradural neuropathology. At upper levels (eg, C5 to C6), CT-myelography may be a

more sensitive diagnostic tool than MRI for preganglionic lesions. Diffusion-weighted neurography may be a valuable tool for evaluating postganglionic plexus lesions, where neural structures can be difficult to distinguish from adjacent tissues using conventional MRI.¹²

Neurophysiological testing like EMG (electromyogram)/Nerve Conducting Studies (NCS) and somatosensory evoked potential can lead to better identification of the site of the lesion. This is especially important if surgical reconstruction is being considered.⁹ Traditionally, EMG has been performed 3 to 4 weeks after injury, although some experts advocate earlier (1 to 2 weeks) testing, arguing that a normal sensory evoked potential from an anesthetic finger suggests preganglionic pathology. One of the drawbacks in performing EMG too early is that it cannot reliably distinguish between axonotmesis (nerve lesion within an intact perineurium) and neurotmesis (complete cutting of the nerve and perineurium). A neuropsychological examination should be considered if there is concomitant brain damage caused by the accident.

I.D DIFFERENTIAL DIAGNOSIS

The medical history in trauma patients is often conclusive. If pain and weakness develop suddenly without any inciting traumatic event, one should consider neuralgic amyotrophy of the brachial plexus.

Table 4 gives an overview of potential differential diagnoses, clinical signs, and general treatment of brachial plexopathy.

II. TREATMENT OPTIONS

II.A CONSERVATIVE, SURGICAL, AND INTERVENTIONAL MANAGEMENT

Extensive physical therapy is a first-line treatment strategy for a traumatic strain or avulsion injury. This is necessary to prevent the development of contractures and secondary pain.

Due to the complexity of problems and the disability that frequently ensues, an individually tailored

Table 3. Neurologic Symptoms Which May Occur with a Brachial Plexus Lesion (Adapted from Kuks et al.⁴)

Brachial Plexus Lesion	Dermatome	Motor	Sensory
Impairment in the upper part	C5–C6–C7	Reduced abduction of the upper arm, endorotation of the elbow and pronation of the hand (policeman's tip position)	± Extensor side of the forearm
Impairment in the lower part	C8–Th1	Paralysis of the small intrinsic hand muscles, flexors of the hand and fingers	Reduced sensation in the ulnar part of the forearm

Table 4. Differential Diagnosis, Clinical Signs, and General Treatment of Brachial Plexopathy

Syndrome	Brachial Plexus Lesion	Thoracic Outlet Syndrome	Cervical Radiculopathy	Neuralgic Amyotrophy
Cause	Traumatic/surgical stretch/sharp	Spontaneous, repetitive movements	Spontaneous/herniated cervical disk/flexion-extension trauma	Spontaneous/viral?; demyelination
Symptoms	Functional loss, paresthesias, pain	Pain, paresthesias Weakness in hand, cold intolerance; vascular signs	Radicular pain, gradual onset and motor/sensory symptoms	Acute onset, severe pain in arm and shoulder region, root distribution weakness
Diagnosis	MRI/CT-myelography/EMG	Clinical neurophysiologic testing	Positive effect of segmental nerve blocks	EMG/MRI
Treatment	Physical therapy/ operative-reconstructive	Physical therapy Local infiltration	Antineuropathic drugs Physical therapy/rehabilitation Segmental nerve blocks	Pharmacological/analgesics Physical therapy

MRI, magnetic resonance imaging; EMG, electromyogram.

rehabilitation program is indicated in most patients. Besides physical support and the use of orthoses, psychosocial interventions also play an important role. Drug therapy depends primarily on the type and quality of pain (eg, nociceptive vs. neuropathic, spontaneous vs. evoked). Recent clinical trials have demonstrated synergistic effects when two different adjuvants (eg, nortriptyline and gabapentin), or an adjuvant and opioid (gabapentin and morphine) are used to treat neuropathic pain.^{13,14} Some experts advocate a mechanistic-based treatment approach based on response to a series of prognostic infusion tests.¹⁵

Most surgical therapies are initiated 3 to 6 months after injury, although some literature suggests that early intervention may improve outcomes.¹⁶ Especially in cases of sharp, lacerating injuries, urgent exploration within the first 72 hours and repair by-end-to-end anastomoses appear to be necessary.⁹ Surgical intervention is also usually considered if there has been no improvement (signs of re-innervation) in 3 to 6 months. A prolonged follow-up with delayed intervention carries the risk of severe muscular atrophy. These surgical interventions ostensibly reduce the risk of persistent serious pain, and may be particularly compelling if there is a chance to restore motor function.³ The technique employed depends on the type and extent of injury. Preganglionic injuries are generally not amenable to repair, and may be treated with nerve (eg, intercostal nerves) transfer (sometimes with free muscle transfer) to restore function in denervated muscle(s). For postganglionic injuries, nerve grafting and nerve repair can improve function in 40% to 75% of patients.¹⁷

Our knowledge of the mechanisms responsible for pain in these patients is still very limited. Not surprisingly then, neurostimulation techniques have been reported to have wide-ranging and unpredictable

effects.^{18,19} Injuries characterized by complete de-afferentation are unlikely to respond positively to spinal cord stimulation (SCS). Although there is a zone of spinal hyperactivity in the dorsal part of the spinal cord, which could theoretically be amenable to SCS, disinhibition in the spinothalamic tract with changes in the spinoreticular fibers can lead to a more centralized disinhibition. In theory, motor cortex stimulation (MCS) should have a positive effect on de-afferentation pain, which has been borne out in clinical practice.¹⁹⁻²¹

Sometimes, there are other anatomical defects that can account for pain. The co-prevalence rate of pseudomeningocele has been estimated to range between 21% and 57% in patients with nerve avulsion,²² and may be treated conservatively or surgically. A traumatic lesion due to traction can cause intramedullary bleeding with focal gliosis and the formation of microscopic cystic changes. Spinal cord herniation has also been reported following brachial plexus avulsion injury,^{22,23} and typically manifests as Brown-Séquard syndrome.

The literature does not provide sufficient data regarding the effects of various pain treatments in patients with a brachial plexus lesion. However, it is well-documented that a large percentage of patients will have persistent symptoms refractory to conventional therapy. In these individuals, surgical interruption of the afferent sensory input into the spinal cord can be considered in patients. In the DREZ (dorsal root entry zone) procedure,³ also known as MDT (microsurgical DREZotomy), a 2 mm deep, 35° incision is made ventromedially in the dorsolateral sulcus of the posterior horn. The level(s) at which this intervention takes place depend(s) on the location of the radicular avulsion.

Results following DREZ have been mostly positive. Thomas and Kitchen²⁴ reported that 79% of 44

patients with brachial plexus avulsion achieved good or fair results at the mean 63-month follow-up. Sindou et al.³ found that 66% of 55 patients with preganglionic brachial plexus injuries experienced excellent or good pain relief lasting more than a year (mean follow-up: 6 years), with 71% obtaining significant functional improvement. Chen and Tu²⁵ reported more modest results. Although 80% of 40 patients with brachial plexus avulsion reported good pain relief postoperatively after thermocoagulation DREZ lesions, success rates declined to 60% at 5-year follow-up, and 50% at 10-year follow-up. Potential complications of surgical treatment include motor weakness and ataxia. Theoretically, so-called MCS could have a positive effect on this pain.

II.B COMPLICATIONS OF INTERVENTIONAL MANAGEMENT

The complications of SCS have been previously described.²⁶

Table 5. Summary of the Evidence for Interventional Management

Technique	Evaluation
Spinal cord stimulation	0

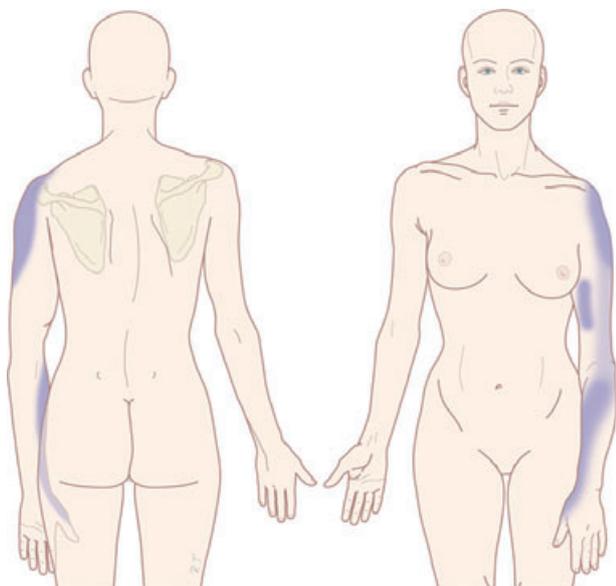


Figure 1. Clinical examination after a traumatic lesion of the plexus brachialis; changed muscle contour and changes in sensitivity.

II.C EVIDENCE FOR INTERVENTIONAL MANAGEMENT

The summary of the evidence for interventional pain management is given in Table 5.

III. RECOMMENDATIONS

In patients with pain resulting from a traumatic brachial plexus lesion who respond inadequately to drug treatment and physical therapy, SCS should only be considered in a study context after a positive trial period and in specialized centers.

III.A CLINICAL PRACTICE ALGORITHM

Figure 2 represents the treatment algorithm for traumatic plexus lesion.

III.B TECHNIQUES

The techniques for neurostimulation were described previously.²⁶

IV. SUMMARY

After high-impact trauma, damage to the brachial plexus can lead to neurologic impairment and

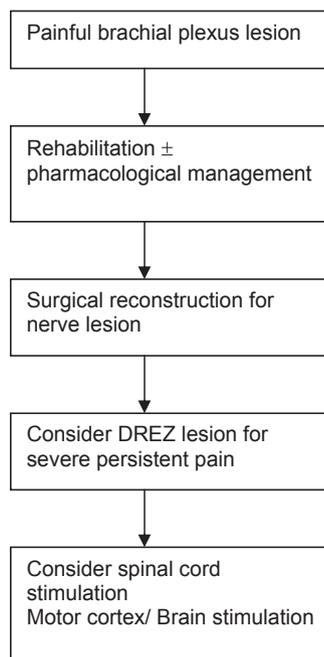


Figure 2. Practice algorithm for the treatment of painful brachial plexus lesion.

neuropathic pain. Extensive physical therapy and rehabilitation treatment are initiated first, particularly to prevent contractures, secondary pain, and atrophy. The cornerstone of treatment is pharmacotherapy, although the evidence supporting different drug regimens is typically extrapolated from clinical trials conducted for other neuropathic pain disorders. Surgical correction of brachial plexus injuries is generally attempted before SCS is used. SCS should only be considered as part of a multimodal regimen when more conventional treatment strategies have failed.

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