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# Brachial Plexopathies: Update on Treatment

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## Abstract

*Purpose of review* Brachial plexopathies (BPs) are a heterogeneous group of diseases which can profoundly affect person's function and quality of life. This review targets current approaches to treatment of different BP types.
*Recent findings* Although there are multiple BP etiologies, non-traumatic causes are particularly unrecognized by clinicians, leading to misdiagnoses and delay in appropriate therapies. Recent data suggests that idiopathic neuralgic amyotrophy may be 50-fold more common than previously thought, with poorer outcomes than reported unless rapid diagnosis is followed by a multidisciplinary approach to treatment, including surgery for refractory cases. Advances in diagnostic imaging reduce time to treatment, if proper recognition by primary providers leads to rapid referral to neurologists. Due to rarity, heterogeneity, and overlap of different BP syndromes, development of treatment strategies is still based on case series and retrospective studies. Despite advances in surgical therapies, there are no randomized trials to guide optimal approaches to treatment in either traumatic or non-traumatic cases.
*Summary* Recent advances in imaging and surgical therapies may significantly improve clinical outcomes for patients with BPs, if rapid diagnosis of these often under-recognized conditions can be substantially improved to optimize approach to management. Controlled trials are still needed to optimize therapeutic strategies.

## Introduction

Brachial plexopathies (BPs) are a heterogeneous group of rare and potentially disabling diseases. Based on lesion etiology, severity, and stage/duration, different treatments have been developed independently over time. However, treatment goals for these disorders are the same, including management of pain and improvement of motor and sensory dysfunction.

Historically, BPs have been classified as traumatic, non-traumatic, and iatrogenic. Depending on predominant site(s) of damage, they have also been described as supraclavicular (involving roots, and trunks) or infraclavicular (affecting cords and terminal branches). By severity of involvement, they have been described as complete or partial [1].

Non-traumatic plexopathies include idiopathic neuralgic amyotrophy (INA), hereditary neuralgic amyotrophy (HNA), infectious, inflammatory, ischemic, metabolic, neoplastic, and post-radiation plexopathies (Table 1). Traumatic BPs include neonatal palsies, "stinger and burner" type sports injuries, traction injuries, root avulsion, and direct penetrating trauma, largely from motor vehicle accidents [2]. Iatrogenic plexopathies result from nerve blocks and other injections, post-thoracotomy traction, malpositioning of the arm or shoulder during surgery, and direct intraoperative nerve trauma [1–3]. While non-surgeons are typically unfamiliar with surgical therapies for BPs, these techniques are increasingly needed to treat refractory non-traumatic plexopathies.

# Traumatic and iatrogenic plexopathies and their treatments

Trauma is the most common cause of BPs, accounting for 1.2–5% of polytrauma and sports injury cases [1, 4]. Most common among traumatic

**Table 1. Causes of brachial plexopathies by mechanism of nerve injury**

| Traumatic etiologies | Non-traumatic etiologies |
|---|---|
| Open BP injuries:<br>GSW, lacerations, stab wounds, animal bites | Inflammation:<br>INA, diabetic cervical radiculoplexus neuropathy, post-surgical inflammatory plexopathy |
| Traction injuries, compression (neuropraxia):<br>rucksack palsy, "stinger," and "burner" | Ischemia:<br>Polyarteritis nodosa, Behçet's disease, giant cell arteritis, hypersensitivity vasculitis, microscopic polyangitis, Henoch-Schönlein purpura, diabetic radiculoplexus neuropathy |
| Severe traction injury (axonotmesis) | Neoplasia:<br>Primary tumors, metastatic tumors, pancoast tumors, paraneoplastic syndromes |
| Root avulsion (neurotmesis) | Radiation:<br>Radiation induced plexopathy |
| Birth palsies | Entrapment:<br>Neurogenic thoracic outlet syndrome |
| Iatrogenic causes:<br>nerve blocks and other injections, post-thoracotomy traction, malpositioning of limbs during surgery, direct intraoperative nerve trauma | Infection:<br>Viruses: EBV, VZV, parvovirus B19, CMV, Mumps, HIV, HSV, Dengue, St Louis encephalitis, Japanese B, WNV, HEV<br>Bacteria and fungi: Leptospira sp., *Mycobacterium tuberculosis*, Yersinia sp., Salmonella typhii, *Coccidioides immitis*, Borrelia burgdorferi |

lesions are closed traction injuries. Treatment depends on nerve injury severity, with mild demyelinating neuropraxic lesions (e.g., rucksack syndrome, "stinger/burner" lesions) improving rapidly within days to months [5]. Following axonal loss (axonotmesis injury), reinnervation potential is reduced, while avulsion neurotmesis injuries never improve spontaneously. Open brachial plexus injuries often coexist with vascular injuries, which may develop later secondary to hematoma, pseudoaneurysm, or AV fistula formation. Fractures, especially clavicular fractures, can cause infraclavicular plexopathy, commonly affecting medial cord. In iatrogenic BPs, both immediate and delayed injury and secondary inflammatory process can occur following surgical procedures and injections [3].

Over the past several decades, many surgical options have been developed to improve functional outcomes in traumatic BPs. Currently available options include neurolysis, nerve repair, nerve transfer or grafting, tendon transfer, free functioning muscle transfer, and arthrodesis [6•]. Timing of surgical intervention is key to achieving maximal improvement. Indications for immediate intervention include penetrating and vascular injuries. Avulsion injuries need early recognition (Fig. 1) and treatment. Otherwise, the most common timing for nerve transfer is 3–6 months. Electrodiagnosis plays a major role in prognostication, identifying patients where good functional recovery without surgery is unlikely [4].

Distance between injury site and target muscle for reinnervation also plays an important prognostic role, since denervated muscle fibers may remain viable for reinnervation for only 12–18 months after insult. Therefore, lower trunk lesions (bearing the longest nerve pathways) have worse prognosis for motor recovery than upper trunk lesions [4]. When grafting is employed, short grafts of less than 10 cm offer superior results.

At the time of surgery, brachial plexus exploration is often accompanied by nerve grafting or nerve transfer. Because advanced imaging and preoperative electrophysiology can identify preganglionic lesions early, surgical treatment can be planned in advance, defining functional objectives, and choosing appropriate donor and recipient nerves for transfers and grafts prior to surgery [7]. Fascicles from multiple nerves have been successfully used for transfers [6•, 8], and although patients require muscle "reeducation," this technique offers faster results than nerve grafting. Further, in patients with complete upper trunk palsy without signs of clinical or electrodiagnostic recovery after 3–6 months, nerve transfer is superior to nerve grafting for restoration of elbow flexion and shoulder function [4].

Brachial plexus reimplantation is an emerging surgical technique for management of complete brachial plexus avulsion injury [9•], with significant clinical and electrophysiological improvements achieved when performed early. Other technique improvements, including adjunct use of neuroprotective agents and reparative cell therapies, are currently under investigation [9•]. When surgical intervention is delayed beyond 12 months, functioning free muscle transfer for restoration of elbow flexion has achieved some success [10]. Central adaptation and central plasticity also play roles in recovery, especially in muscle reeducation and sensory symptom control [11, 12].

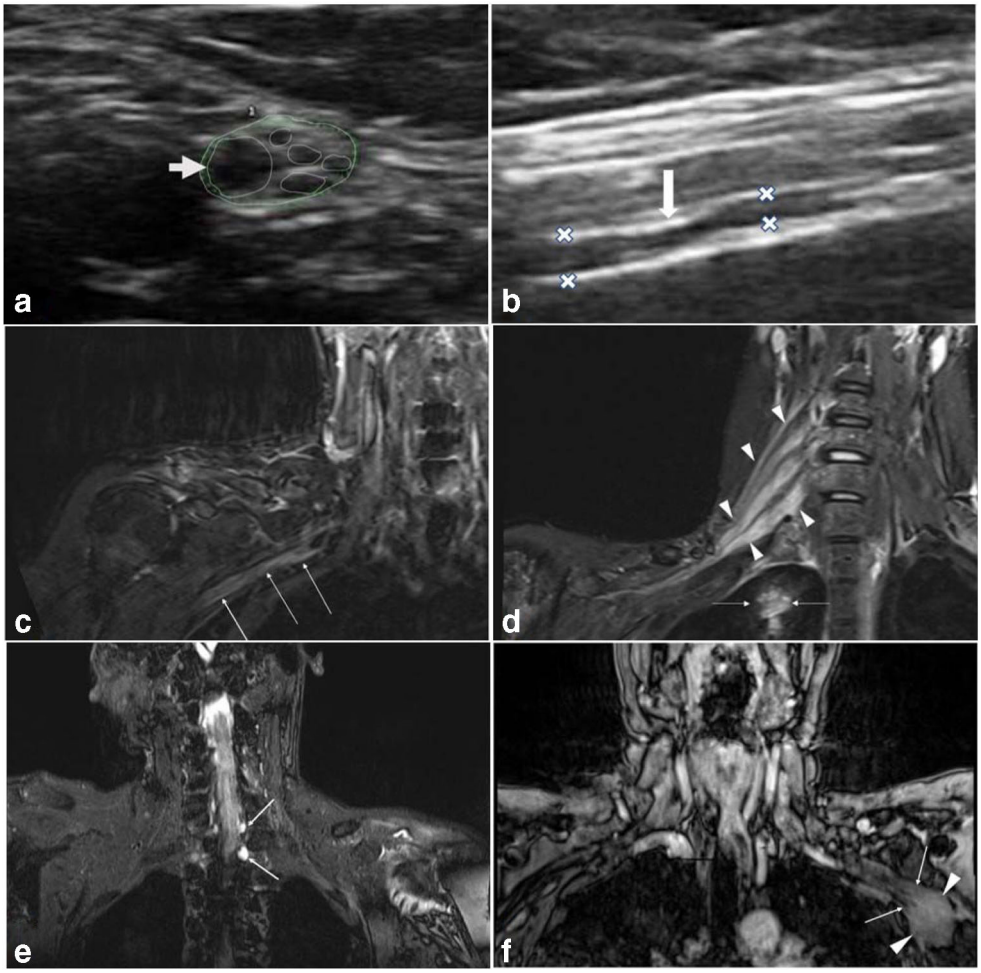


**Fig. 1.** Imaging findings in brachial plexopathies. **a** High-resolution ultrasound, transverse view of the median nerve in the arm in a patient with idiopathic neuralgic amyotrophy. Enlargement of one fascicule (cross-sectional area of 5 $mm^2$), compared to other normal fascicules (1–2 $mm^2$). **b** High-resolution ultrasound, longitudinal vew of the median nerve, caliber change with partial hourglass-like constriction of one fascicule (white arrow). **c** 3T MRI, Coronal STIR sequence showing mild patchy hyperintensity involving right brachial plexus in patient with idiopathic neuralgic amyotrophy. **d** 1.5T MRI, T2 STIR sequence, coronal view of the brachial plexus of patient with coccidiomycosis of the right lung apex (arrow), and brachial plexopathy as initial presentation. All trunks, divisions, and cords of the right brachial plexus are thickened and hyperintense (arrowheads). **e** 1.5T MRI T2 coronal view of left brachial plexus in a patient after motorcycle accident, images depicting traumatic meningoceles (arrows) consistent with root avulsion. **f** 3T MRI, T1 VIBE images, coronal view of brachial plexus in patient with breast cancer. Distal cords (arrows) are enveloped by metastatic axillary adenopathy (arrowheads).

## Brachial plexus birth palsy

Brachial plexus birth palsy (BPBP) is a special subset of traumatic BPs occurring during delivery. The two main risk factors for this rare disorder (1.15% incidence in the USA) are shoulder dystocia and macrosomia, with trauma produced by traction on the head or upper limb during delivery [13–15]. Proximal (Erb-Duchenne) palsy is most common, affecting C5-C6 in 55–60%, C5–7 in 30%, and C5-T1 in 15–20% of cases. Distal (Klumpke-Dejerine) palsies account for less than 2% of cases. Although authors previously described BPBP as

transitory, with full recovery in most cases, recent studies provide variable prognosis, with lifelong functional limb impairment in 35% of affected individuals [13]. Total paralysis and Horner signs are poor prognostic factors, and infants who do not improve within the first 2 months should be managed by a multidisciplinary team in a tertiary care facility [14].

During the first 3 months post-injury, conservative treatment consists of 10–15 days of elbow immobilization to the body to relieve pain and to foster healing of neuropraxic lesions, followed by functional rehabilitation for maintenance of joint range of motion, prevention of retractions, and muscular reinforcement. From 3 to 6 months, depending on the degree of motor recovery, imaging, functional testing, and surgical planning prepare the patient and family for surgical intervention. Multiple surgical techniques have been utilized to improve outcomes, most common of which are nerve transfers and grafting. Extraplexus neurotizations are reserved for total paralysis with avulsion [14].

As for other traumatic BPs, physical therapy and rehabilitation after surgical repairs have both peripheral and central mechanisms of action [11]. Constraint-induced movement, sensory re-education, electrical stimulation, and transcranial stimulation are important effectors of central plasticity [11], included with standard rehabilitation strategies. Patients with BPBP can adapt and participate in most activities, and even with residual deficits, function psychologically and cognitively within the normal range [16].

# Non-traumatic brachial plexopathies

Although less frequent than traumatic BPs, non-traumatic plexopathies require sophisticated diagnostic skills from clinicians, who must understand both pathophysiology and therapies for these disorders. INA accounts for > 80% of cases, while HNA (5%), neoplastic and paraneoplastic BP (0.4% and 0.5–5% respectively), radiation induced plexopathy (1–7%), infectious BP (1–2%), and neurogenic thoracic outlet syndrome (0.001%) compose the remainder. However, there is still confusion regarding what comprises active infection versus post-infectious inflammation, or neoplasm-linked BPs versus paraneoplastic processes. Further, clinical similarities exist among different categories of damage, with painful onset linked to nearly all cases of traumatic plexopathy and most nontraumatic etiologies except for some hereditary and radiation-induced plexopathies. Regarding treatment, previously unused surgical therapies are becoming increasingly important in unresponsive non-traumatic BP cases.

## Idiopathic neuralgic amyotrophy

First described in 1887, with the largest early confirmatory report by Parsonage and Turner in 1948, this condition has been known by many different names, including Parsonage-Turner syndrome, neuralgic amyotrophy, acute brachial neuropathy, acute brachial plexitis, idiopathic brachial plexopathy, idiopathic brachial neuritis, paralytic brachial neuritis, and brachial radiculitis. As currently described, INAs are distinct peripheral nerve disorders with core features of severe pain, paresis in the brachial plexus distribution, and subsequent muscle atrophy and paresthesias [17, 18]. The severity and distribution of nerve involvement vary, but most commonly the upper trunk, long thoracic,

suprascapular, and anterior interosseous nerves are damaged. However, any portion of the plexus can be affected [17].

Although INA etiology is unclear, it appears to involve genetic predisposition, susceptibility to injury, and immune triggers [17, 19]. An immune-linked hypothesis is supported by > 50% incidence of preceding infections or immunizations, response to steroids within the first 4 weeks, and pathological reports describing nerve inflammation [19]. In up to 20% of cases surgery, trauma, or strenuous exercise precede onset within 4 weeks [20].

Infectious and immunization triggers are many, including seasonal and endemic infections, as summarized in recent reviews [18, 19, 21]. While some of these infectious agents are directly neurotrophic, it is unclear why others trigger indirect inflammatory processes leading to INA. The predilection of the upper plexus and certain nerves to INA has been hypothesized to result from selective weakening of the blood-nerve barrier due to increased mobility, leading to greater exposure to immune targeting [17].

Diagnosis remains predominantly clinical, based on chronological development of signs and symptoms [22]. Although diagnostic criteria were established in 2000 and updated in 2015 [23•], underdiagnosis of INA remains common among non-neurologists, with cases often described as "unusual shoulder pain" by ED physicians [24] and PCPs [23•]. This is only partly explained by atypical presentation [25], since after training of primary practitioners to recognize this entity, INA incidence in the primary care setting increased from 2 to 3 cases per 100,000 per year to 1 in 1–2000 individuals [23•]. Unfortunately, severe pain at onset and association with strenuous physical activity causes patients to visit emergency rooms, where they are usually referred to orthopedic specialists or other care providers. Misdiagnosis with inappropriate studies and procedures slow accurate diagnosis, with delays of greater than 6 months before patients are referred to neurologists.

There are three main phases of "classic" INA presentation:

1. Pain—90% of cases have initial pain, described as severe and relentless, involving shoulder girdle muscles. Symptoms are primarily unilateral [22], with latency from inciting trigger to presentation of 1–6 weeks. Duration varies from 1 day to 2 months, though 10% have severe protracted pain.
2. Weakness, and muscle atrophy—observed universally, with amyotrophy usually occurring within 2–6 weeks of symptom onset, reflecting axonal loss. [16, 23•].
3. Recovery—degree and duration depends on lesion severity and extent. Overall, 75% of untreated cases show some recovery between 6 months and 3 years. [18, 26, 27].

This classic presentation occurs in 71% of INA cases, similarly in both adults and children [28, 29]. Extended presentation, found in 17% [8, 15], involves additional damage to remote nerves, including the lumbosacral plexus, lower cranial nerves, phrenic nerve, and recurrent laryngeal nerve [25, 30]. Again, pain is the presenting symptom, usually unrelenting and poorly responsive to pain medications. Motor weakness follows with muscle atrophy. Later sensory symptoms, present in 78% of cases, include paresthesias and hypoesthesia; autonomic symptoms are rare [25]. Atypical presentations, observed in 12% of individuals with INA, include both recurrent phenotype (5–26%), non-painful NA (less than 10% of cases), and limited nerve targeting [18]. However, a series of 281 patients with suspected INA documented single nerve involvement in 174 (46%), while 205 (54%) patient had multifocal lesions [31]. Trapezius

atrophy [32] and phrenic nerve involvement [30] were common single-nerve targeted lesions. Again, initial presentation of pain, followed by focal atrophy and slow recovery suggested that these cases be included as INA. Since atypical presentations constitute a diagnosis of exclusion, however, such affected patients should be screened for MMN, entrapment neuropathy, CIDP, Lewis-Sumner syndrome, and HNPP. FSHD may be considered, based on its predilection to focal motor neuropathies [18].

Laboratory investigations are usually unhelpful in INA diagnosis, ordered for exclusion of disease mimics. Studies are also used to identify recent viral infections, and to exclude other autoimmune conditions. CSF studies are usually normal, though abnormal CSF profiles have been found in 29% of INA cases, mostly documenting blood-brain barrier disruption. Anti-ganglioside IgM antibodies were detected in 36% of sera from patients with INA, compared only 2% of controls, but their pathological significance is unclear [33]. Genetic investigation for *SEPT9* mutation is performed when HNA is suspected because of recurrent disease or positive family history.

Electromyography and nerve conduction testing has been an important supplement to clinical findings, documenting active denervation changes 2–4 weeks after onset of symptoms, with positive sharp waves and fibrillations identified in nerve, trunk, or cord distributions.

Pathology data in INA are limited, though reports describe early non-necrotizing mononuclear inflammatory cell infiltrates surrounding epineurial and endoneurial vessels [34]. More common are later stage histological studies obtained from surgical cases, documenting macroscopically visible fascicular hourglass-like constrictions, with microscopic perineurial thickening with CD8-positive T lymphocyte infiltrates [35]. The first intraoperative descriptions of hourglass-like constrictions or torsions were reported in small monofascicular nerves and isolated median nerve fascicules during explorations of nerve compressions performed in the 1960s. Review of these case reports strongly suggests that patients had INA, and neurolysis of affected regions often produced reasonable recovery. One of the best recent case series of surgically identified hourglass-like constrictions in refractory classical INA, diagnosed by clinical and electromyographic criteria and without recovery after 2–11 months, documented hourglass-like constrictions in all affected nerves, which were successfully treated by neurolysis or nerve resection. Resected nerve histology revealed inflammatory infiltrates. [35, 36].

Yet, few patients benefitted from surgical interventions until improvement in imaging quality and availability of 3Tesla MRI made possible non-surgical detection of hourglass-like constrictions, pre and post-lesion dilations, and "bull eye" changes in clinically affected nerves [37•, 38]. Somatotopic plexus organization also can be seen on 3T imaging with MR neurography, and regions of increased T2 signal correlate with axonal loss on nerve conduction test [38]. During subsequent surgeries, nerve resection was often performed, with pathology demonstrating severe axonal loss and inflammation [27]. MRI imaging has also detected edema in affected muscles shortly after denervation [39]. However, MRI does not differentiate between sensory and motor fibers [40].

Ultrasound also identifies hourglass-like constrictions in INA (Fig. 1). Ultrasound-detected AIN and PIN hourglass-like constrictions have been confirmed intraoperatively [41], with ultrasound accurately predicting observed constriction severity. Ultrasound more easily detected affected nerves and

fascicules outside the brachial plexus than MRI, tracking nerves along their course to identify exact surgical sites [41].

Sonographic attempts to find early diagnostic markers for INA also detected swelling independent of nerve constriction. The suprascapular nerve, commonly involved in INA, was one such nerve enlarged early in INA [42]. Ultrasound better documented early extra-plexus peripheral nerve enlargement [43] than MRI, with high resolution (15+ MHz) ultrasound probes detecting morphological changes in nerves < 1 mm diameter [44]. Thus, advantages of ultrasound over MRI in identifying peripheral nerve lesions included better spatial resolution, easier and less expensive side-to-side comparison, and real-time assessment. However, ultrasound is less effective in identifying brachial plexus lesions [45•].

Overall, four types of sonographic abnormalities were described in INA: (1) swelling without constriction, (2) swelling with incomplete constriction, (3) swelling with complete constriction, and (4) rotational phenomena [46]. Such torsion or entwinement was previously described in the surgical literature, but was only later linked to INA [35]. In the proper clinical setting, prognosis depended on sonographic findings; in patients with swelling but no constriction, conservative therapies were adequate to allow clinical improvement. When swelling was accompanied by complete constriction, no reinnervation occurred [45•], requiring nerve grafting. Operative specimens revealed inflammation with severe fibrosis, causing loss of nerve continuity at sites of constriction, preventing spontaneous reinnervation. Following grafting to restore open paths for nerve regrowth, reinnervation occurred in 2.5–4 months. But timing for surgery was also important, as patients undergoing surgery 4 years after symptom onset showed minimal improvement, with persistent sonographic abnormalities 6 months following surgery [45•].

Proposed mechanisms for these morphological changes in INA include early inflammation, producing nerve enlargement, with formation of adhesions and local fascicle fixation as edema subsides. Resulting structural changes produce focal hourglass-like constrictions, increasing the likelihood of nerve torsion [46]. The timing of these changes is defined, with cases assessed in less than 6 months from clinical presentation documenting no focal constrictions at sites of edema [44], while later studies revealed partial or complete nerve constriction [45•]. Based on these data, it is reasonable to postulate that depending on the phase of the disease, the patient might benefit from different treatment approaches: near the time of clinical onset, starting steroids and/or other immunomodulatory agents might prevent severe nerve damage, while in later stage disease, nerve constriction requires reopening nerve paths by neurolysis or grafting to regain function.

## Ina treatment update

Corticosteroids shorten the time of intense pain and hasten motor recovery, if administered within 1 month of symptom onset [20, 21, 25, 47]. However, there is debate regarding superiority of intravenous or oral steroids [27] in mitigating stenotic, fibrous changes, and preventing axonal damage [18]. Locally injected steroids could actually worsen or induce INA [48, 49].

However, unlike previously reported complete recovery of 80–90% of patients within 2–3 years of therapy, more recent data suggest that 4% achieve full

**Table 2. Treatment of idiopathic neuralgic amyotrophy**

| Disease phase | Treatment |
|---|---|
| Acute phase | Corticosteroids in first month:<br>Prednisone 60 mg PO with taper<br>Methylprednisolone 1 g IV for 5 days<br>IVIG (Single dose or induction series of 4–5 treatments, one report of 12–16 months therapy) |
| Management of neuropathic pain | NSAIDs<br>Opioids<br>AEDs: gabapentin, carbamazepine<br>TCAs: amitriptyline, nortriptyline<br>Transcutaneous and direct nerve stimulation<br>Spinal cord stimulation<br>Acupuncture |
| Recovering phase | Rehabilitation: range of motion, stretching, strengthening exercise<br>Motor and sensory re-education therapy |
| Late phase (if no or minimal recovery after 6 months) | Surgical treatment (when hourglass-like constrictions and torsions are detected):<br>Neurolysis<br>Nerve grafts<br>Nerve transfers |

recovery and 16% have partial recovery after 1 year [26, 50, 51]. Further, a survey of 246 patients revealed that 60% had residual pain and motor impairment 3 years after onset [25], despite physical therapy, massage, electrical stimulation, and/or exercise. While stretching exercise may have been effective in reducing pain, strength and resistance exercise were not associated with benefits [50]. Acute phase pain was inadequately treated with NSAIDs and opioids, and antiepileptic medications and tricyclic antidepressants provided only limited mitigation of lingering neuropathic pain [50, 51]. Thus, there is a strong need for new, effective treatments for INA.

Among pharmacotherapies, effective corticosteroid dosage varied from 60 mg of oral prednisone for 1 week [25] to pulsed intravenous 1 g methylprednisolone for 5 days [49]. Efficacy of pulsed steroids with cyclophosphamide has been reported in isolated cases, but most steroid sparing immunomodulators cannot be considered as acute therapy. IVIG was evaluated in small case series [52–57] with early treatment being more effective [57] in shortening disease course than delayed treatments [58].

Pain limits physical rehabilitation in the acute phase, but it has proven beneficial in the recovery phase. Generally, treatment goals are maintenance of range of motion and prevention of function loss [59], but such therapy does not appear to speed recovery [47]. The mainstays of physical therapy are stretching, range-of-motion, and cautious use of therapeutic exercise, to avoid overload of weak muscles. However, failure to initiate PT results in adhesive capsulitis, shoulder subluxation, and dislocation due to weakness, chronic pain, and loss of function [59].

Transcutaneous nerve stimulation can be helpful for pain control, but not in promoting recovery, despite popularity of percutaneous and minimally invasive approaches to nerve stimulation [60]. Direct nerve or spinal cord stimulation has also been tested in brachial plexus localized neuropathic pain [61], with 68% reporting pain reduction following US-guided nerve or root stimulation [53, 61, 62]. There may be role for acupuncture on pain control, but not in recovery.

Most patients with INA are currently treated with a multidisciplinary approach, combining pharmacological treatment, physical modalities for pain control, and physical therapy, with surgical treatments reserved for refractory and severe cases when conservative treatments prove ineffective (Table 2), [47, 49]. Surgical procedures include neurolysis, nerve grafts, and nerve transfers, usually after 6 months from onset, but some authors suggest that delay in treatment beyond 2–6 months may result in less favorable outcomes. Typical response rates of 60% have been reported following surgical interventions, with surgical neurolysis especially effective in affected peripheral nerves [63].

## Hereditary neuralgic amyotrophy

Hereditary neuralgic amyotrophy (HNA), 10–100 times less common than INA, is an autosomal dominant disorder with high but incomplete penetrance and genetic heterogeneity [17]. Fifty-five percent have point mutations or duplication in the *SEPT9* gene on chromosome 17q25 [64]. Recurrent episodes of sensory and motor nerve damage are common, associated with sudden onset of severe, non-abating pain in over 80% of cases (> 95% of *SEPT9* mutant HNA), and subsequent muscle atrophy and weakness [54, 65]. Lesions are more widespread than clinically evident, with 56% of HNA cases having extended involvement [65]. Treatment is typically conservative—steroids may speed improvement in pain though they do not affect motor recovery [47]. However, residual pain is common, requiring symptomatic treatment.

## Vasculitic brachial plexopathy

BP has been reported in polyarteritis nodosa, Behçet's disease, giant cell arteritis, hypersensitivity vasculitides, microscopic polyangitis, and Henoch-Schönlein purpura [66, 67]. Diabetic cervical radiculoplexus neuropathy is a distinct syndrome [68], with vessel wall inflammation containing perivascular inflammatory collections of hemosiderin-laden macrophages, suggesting microvasculitis. Subsequent ischemic injury produces secondary axonal degeneration [68]. Treatment includes oral or intravenous steroids, intravenous immunoglobulin, or plasmapheresis.

## Infectious brachial plexopathy

Although infectious agents can produce NA, debate exists over whether association of NA with endemic viruses, bacteria, and fungi results from direct plexus injury, or more likely triggers secondary immune targeting of the brachial plexus [69–71]. For example, neuroborreliosis signs, including NA, appear to result from inflammatory response to the organism in susceptible patients [72], similar to post-immunization plexitis linked to tetanus, hepatitis B, and influenza antigens [18, 49].

Direct infection has been suggested for seasonal zoonoses, including Dengue and WNV [73], even though these diseases may also induce post-infectious

inflammatory disease. The best evidence for infection-associated INA is observed with hepatitis E [74–77, 78•], where IgM production and symptoms of primary infection coincide with onset of INA.

## True neurogenic thoracic outlet syndrome

The rarest of nontraumatic supraclavicular plexopathies is caused by first cervical rib, band, or scalenus anterior muscle compression. Surgical treatments target removal of suspected compression sites, with neurolysis if needed [79]; recently, non-surgical therapy has been successful in selecting cases for subsequent surgery [80]. Because of its rarity, it was previously considered a controversial entity [79].

## Neoplastic and paraneoplastic plexopathy

The frequency of neoplastic BP is 0.43% among cancer patients [81]. Solid tumors, leukemia, and lymphoma infiltrate or metastasize to the brachial plexus [82], with lung and breast metastases most commonly producing BP [83, (Fig. 1). Primary brachial plexus tumors include schwannoma (83%), neurofibroma (8%), and malignant peripheral nerve sheath tumors (9%) [83].

Although BP is rarely a first manifestation of neoplasm [82], pain is an almost universal presenting symptom. Seventy-five percent of patients will have pain, weakness, and atrophy in a lower plexus distribution, with pan-plexus involvement in the remaining 25% [82]. Local mass may be palpable, with localized tenderness [84].

Complete or subtotal surgical tumor resection is the treatment of choice for solid tumors [84], with neurolysis and nerve grafting used to aid recovery. For metastatic brachial plexus involvement, treatment also includes radiation therapy, chemotherapy, or hormonal therapy, depending on cancer type [83]. Treatment of neurolymphomatosis, a rare and often missed manifestation of lymphoma and leukemia thought to be in part of paraneoplastic origin [87, 90], includes systemic chemotherapy, sometimes combined with intrathecal chemotherapy and external-beam radiotherapy. Prognosis is generally poor, unless paraneoplastic symptoms predominate. Pain management includes analgesics and adjunct procedures including neurolytic blocks in [85] and radiofrequency ablation.

## Radiation induced plexopathy

Radiation-induced plexopathy (RIP) is usually infraclavicular, producing progressive motor and painless sensory deficits from direct axon injury and fibrosis of the vasa nervorum, with secondary nerve microinfarctions. Latent period before symptom recognition ranges from a few months to over 30 years. Overall RIP frequency in radiotherapy-treated patients is 1.8–4.9% [84, 86]. RIP is especially apparent in patients receiving high-dose boost radiotherapy, intraoperative radiation therapy, or salvage radiotherapy.

Unfortunately, conservative measures do not ameliorate RIP [87]. Surgical therapies are more helpful, with microsurgical neurolysis of the brachial plexus to release extensive radiation fibrosis, use of vascularized flap coverage to improve blood supply, and restore some degree of limb function [87] and nerve grafting and muscle and tendon transfers to restore function in paralyzed states.

It is important to immediately differentiate RIP from neoplastic BP. Diagnostic differences include positive tumor enhancement on PET or MRI in neoplastic BP and rare myokymia on EMG in radiation plexopathy.

## Future directions

Although improvements in imaging and surgical techniques have created the potential for improved BP treatment outcomes, controlled trials are desperately needed to define optimal management plans for these disorders. Potential trials, while possible for traumatic BPs, cannot be performed for non-traumatic BPs until early recognition of these disorders becomes the norm. As previously documented, this requires training of emergency physicians and PCPs in BP recognition, to avoid initial misdiagnosis and unnecessary delays to appropriate treatment, currently plaguing many centers. This also requires rapid evaluation by neurologists, to initiate and document response to initial conservative therapies, and to triage patients needing further imaging, electrodiagnostic studies, secondary surgical procedures, or focused rehabilitation modalities, potentially performed at regional multidisciplinary centers, as already exist for BPBP.

Thus, system wide changes must occur before recent advances in BP diagnosis and treatment can truly benefit affected individuals. Recognition that these changes must occur is the first step to achieving the goal of early, optimal treatment for patients afflicted with BP.

## Compliance with Ethical Standards

**Conflict of Interest**
The authors declare that they have no conflict of interest.

**Human and Animal Rights and Informed Consent**
This article does not contain any studies with human or animal subjects performed by any of the authors.

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