Iatrogenic Nerve Injuries

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KEYWORDS

• Iatrogenic • Nerve • Injury • Repair • Management

It has been noted that the discretion of the protagonists generally draws a veil over the early proceedings in these cases.[1]

Injury inflicted by a treating physician has long been termed iatrogenic. In its strictest sense, however, this term is already a misnomer because it would imply "physician generating" (compare with cancerogenic). For this reason, Bonney and Birch proposed using iatropathic or iatrogenous (pathology inflicted by a physician).[1–3] Regardless of one's term preference, as long as doctors treat, patients will always be at risk for inadvertent maltreatment. Patients who undergo surgery are at risk for sustaining injury to a peripheral nerve in or outside the field.

IS IT RELEVANT TO DISCUSS THIS ENTITY SEPARATELY FROM TRAUMATIC NERVE INJURIES?

Our first evaluation of iatrogenic injuries revealed that 17.4% of the traumatic nerve injuries we operated on at our institution had been iatrogenic in nature.[4] Other nerve referral centers estimate similar rates among the traumatic injuries they operate on. Twenty-five percent of the largest series of operated sciatic nerve lesions (89/353),[5] 50% of femoral nerve lesions seen (47/94),[6] and 93% of operated accessory nerves of the same investigators were iatrogenic (103/111).[7]

Frequently, iatrogenic nerve lesions are not detected at all or not identified as such, or are approached with a mixture of therapeutic nihilism and negligence, which is why it should prove helpful to outline some of the more frequent lesions and to recapitulate the rationale for surgical intervention. Some nerves are more prone to injury than others because of their susceptibility or vicinity to the target structure. Sometimes, the particular technique applied puts the nerves at an increased risk for damage (eg, minor surgery for removal of lymph nodes at the posterior triangle of the neck, excessive use of retraction for a prolonged time (hip surgery), transaxillary approaches for the ones unfamiliar with brachial plexus pathology, or simply unawareness that the distinct pathology operated on arises from nerve [nerve sheath tumors mistaken for a "soft tissue growth"]). Although inadvertent nerve damage is often caused by poor surgical technique, some lesions might even be unavoidable with the best of preventive care.

If, as it is to be hoped, a peripheral nerve surgeon is consulted for advice, the pending questions always revolve around the need for surgical exploration and repair and the potential of recovery after such an intervention, compared with a conservative approach. For neurosurgeons, it is important to be able to give helpful advice and readily point out appropriate treatment approaches. The choice of approach will depend mainly on the lesion mechanism (direct intraoperative damage versus positional lesion or injection injury), the nerve involved, and the symptoms one is attempting to treat (pain, motor sensory, or cosmetic deficit). The intention of this article is not to give an all-inclusive overview but rather, to raise awareness of the more frequent injuries that will benefit from microsurgical intervention.

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INJURY MECHANISMS

Iatrogenic nerve injury can result from many nonoperative and operative causes: direct damage in the operative field, pressure or traction during anesthesia, injection of noxious material, needle puncture, pressure by external hemorrhage, ischemia, anticoagulation therapy, orthotics and casts, thermal injury, radiation therapy, and more.[1,8,9]

Common nonoperative causes include injection and needle injury, and external compression from orthotics and casts. Secondary sequelae of anticoagulation therapy affecting peripheral nerves are less frequent (eg, retroperitoneal hemorrhage and femoral palsy). Operative causes may be differentiated into intraoperative positioning damage and direct intraoperative damage. Direct intraoperative damage accounts for most of the lesions that nerve surgeons will have to explore microsurgically.[10,11] Of all the iatrogenic nerve lesions operated on at our institution, 94% were caused directly within the operative field.[4,12] The damage usually occurs at a distinct point of time because of a specific maneuver by the surgeon. A whole host of injury mechanisms to the nerves are possible, including being squeezed, drilled or wound up by screws; grabbed and squeezed together with the bone that needs to be repositioned; compressed by plates or retractors; torn with instruments; stretched or pinched with repositioning instruments; pierced by Kirschner wires or screws; contused by way of sudden repositioning maneuvers or simply because the forces applied were too strong (eg, clavicle repositioning with pliers for fracture plating, resulting in sudden injury and pressure on the brachial plexus); coagulated with Bovie cautery or bipolar forceps; transected because the nerves were not visualized; transected because they were not correctly identified or were mistaken for vessels; burnt; cemented or cement burned (hip arthroplasty); excised altogether with the target pathology (unrecognized nerve sheath tumors); or injured directly or indirectly by wire cerclage (eg, sciatic at buttock level, ulnar nerve at elbow level). Nerves are also inadvertently sutured and ligated (herniorrhaphy, varicose vein ligation), torn (vein stripping), or simply stretched to nonfunctionality. Sometimes, nerves cannot be visualized because the operative field is extended beyond the field of view. At the more extreme end are cases where nerves have been used as tendon grafts.[13–16] Deep hypothermia during cardiac standstill was once reported to result potentially in phrenic nerve dysfunction.[17] Open cardiac surgery (combination of sternotomy and hypothermia) has an inherent risk for brachial plexus palsy (stretch injury due to sternotomy and hypothermia).[8] Although, in retrospect, some of the reported cases of perioperative iatrogenic brachial plexus palsy might, in fact, have been due to neuralgic amyotrophy, many of the injuries described could happen despite the best of preventive measures and awareness of approach-specific risk to nerve. It is commendable if the surgeon who inflicted an injury consults a nerve surgeon for advice to initiate the appropriate treatment. Unfortunately, not only in our experience, this is still the exception.[2–4,18]

Direct and Intraoperative Damage

Procedures prone to causing nerve injury

Among the common procedures that cause nerve lesions are osteosynthesis and osteotomy, arthrodesis, lymph node biopsies at the posterior triangle of the neck, carpal tunnel release (CTR), operative varicose vein treatment, Baker cyst removal, and inguinal hernia repair. **Table 1** lists and groups causative procedures of 178 of 210 iatrogenic injuries operated on at the Neurosurgical Department of the University of Ulm from January 1990 to March 2005. In terms of procedure categories, orthopedic procedures account for most of the injuries (26%), followed by ''minor surgery'' (24%) and hand surgery (18%). Inguinal hernia repair (9%) and varicose vein operations (7%) contributed similar case rates to the nonoperative treatment category (7%), since hernia repair and varicose vein operations are operative treatment categories.

REGIONS WHERE NERVES ARE AT PARTICULAR RISK

Even before we observed a current drastic rise in lesions after CTR, the carpal tunnel and distal wrist (19%, or 36/191 operated iatrogenic lesions), the posterior triangle of the neck (15% or 29/191), and the knee region including the popliteal fossa (21%, or 40/191) accounted for more than half of the iatrogenic lesions (55%) we operated on until 2002.[12] In these regions, nerves lie superficial, close together, or in the immediate vicinity of the target structure. The groin remains a region at risk because of hernia repair. Surgeons treating inguinal hernias are well aware of the inherent risk for ilioinguinal or genitofemoral nerve damage. Despite awareness and preventive measures, these nerves remain at a particularly high risk because of their small caliber in conjunction with their course over or under fascia, which impedes their identification; minimally invasive procedures with mesh application might not particularly help in the recognition of these nerves.

Table 1
Case numbers of procedures that caused iatrogenic injury, grouped into categories

Category	No. of Cases	Procedures (No. of Cases)
Orthopedic procedures (26%)	55	Osteosynthesis (18) Osteotomy (7) Repair of ruptured ligament (7) Removal of plates (5) Removal of exostoses (4) Kirschner wire placement (2) Release of gastrocnemius muscle (2) Endoscopic meniscectomy (2) Myotomy (2) Open meniscectomy (1) Lengthening of Achilles tendon (1) Wire cerclage (1) Muscle fixation (1)
Minor surgery (20%)	43	Lymph node biopsy (22) Ganglion cyst removal (6) Lipoma removal (4) Hematoma evacuation (3) Abscess drainage (3) Cyst removal (2) Foreign body removal (2) Excision sebaceous cyst (1)
Hand surgery (15%)	32	Endoscopic (15) Open (6) CTR, tenolysis (5) Index finger release (3) Dupuytren release (3)
Other surgical procedures (9%)	19	Varicose vein operations (14) Removal of Baker cyst (5)
Inguinal hernia repair (8%)	16	Open (15) Endoscopic (1)
Nonsurgical treatment (6%)	13	Veni- or arteriopuncture (5) Cast application (5) Malpositioning (1) Injection injury (1) Removal of suction drain (1)
Remainder (15%)	32	Various others, not within one category
Total (100%)	210	

Depicted are 178 cases of 210 that were operated on at the Neurosurgical Department of the University of Ulm between January 1990 and March 2005.

A well-appreciated classic is injury of the common peroneal nerve at the fibular head (eg, casts, positioning). Here, the common peroneal nerve courses over and around the fibular head and neck in a superficial plane between bone and soft tissue before it branches and is therefore vulnerable to compression. The ulnar sulcus at elbow level is always of particular concern during positioning of patients.

NERVES FREQUENTLY INJURED

A full description of all nerves at risk is far beyond the scope and intention of this article. We prefer to describe a selection of nerves that will consistently have to be repaired. Our own series of iatrogenic nerve injuries operated on is regularly updated. From January 1990 to January 2008, we operated on 263 iatrogenic nerve injuries. The top 10 nerves affected have seen no major changes throughout the years. Among them are the median (41/263, 16%), spinal accessory (33/263, 13%), peroneal (30/263, 11% [19 common, 9 superficial, 2 deep]), radial (25/263, 10% [13 main branch, 6 posterior interosseous, 6 superficial sensory), genitofemoral (13/263, 5%), ilioinguinal (11/263, 4%), femoral (11/263, 4%), and ulnar (10/263, 4%). We did,

Fig. 1. Partial median nerve transection due to open CTR. Wrist level is toward the right and palm level on the left side. The nerve is depicted after neuroma resection and interfascicular dissection in preparation for placement of interpositional sural autografts of different lengths.

however, recognize an increase in referrals for median nerve injury in general and specifically after CTR. Thirty-one of the 41 median lesions were due to CTR (open: 14/31, 45%; endoscopic: 17/31, 55%), so that the median now is the most frequently injured nerve. Our rate of operated iatrogenic nerve lesions since 2000 ranges from 10 to 22 per year. In 2007 alone, of 22 operated iatrogenic nerve injuries, 5 were median lesions (23%).

Median Nerve at Wrist and Palm Level

Most of the time, this nerve is damaged from attempted CTR. The injury happens from open (**Fig. 1**) and endoscopic procedures (**Fig. 2**). For damage during endoscopic procedures, several factors can be made accountable. They are discussed separately under ''Avoidance of Iatrogenic Nerve Injury During Endoscopic Carpal Tunnel Release'' in this issue. In open procedures, a correlation with faulty or ''suboptimal skin incisions'' can often be observed.[1,19] Certainly, to be secure with the more minimalized open types of CTR, expertise in regional anatomy is paramount because the surgeon also has to be able to work within tunnels to achieve complete release (eg, lifting the distal and proximal skin edges). Knowledge of skin level landmarks is of great importance for endoscopic and open release techniques. These landmarks help to project the main nerve trunk, the confines of the retinaculum, the recurrent motor branch, and the course of the ulnar nerve and superficial vascular arch on skin level before incision (see Fig. 2 in article by Kretschmer and colleagues in this issue). Because of false trajectories, severe lesions of the ulnar nerve as a result of attempted median nerve CTR are also encountered.[20] Knowledge of pertinent landmarks and variations is also important when evaluating and exploring such lesions. Passage of the recurrent motor branch through the retinaculum on the radial side is one example. Numerous variations of the median and ulnar nerve branching pattern and sensory and muscular supply have been described.[21–23] Examples include overlap of sensory supply from the ulnar side[23] (eg, communicans or Berrettini branch), or motor supply to the abductor pollicis brevis from the distal deep ulnar branch. Even after complete median nerve transection at wrist level, the possibility exists of maintained ''median nerve function'' due to anomalous innervation. In other words, complete transection can result in incomplete functional deficit because of cross-over supply from the ulnar nerve (and vice versa).[24,25] Therefore, this finding should not be mistaken for spontaneous recovery preventing an attempt of exploration and reconstruction. Various types of communications (so-called anastomoses) exist between the ulnar and median nerves. The Martin-Gruber varieties at the proximal forearm level can be found in 20% to 40%.[26–29] Median fascicles cross over to the ulnar nerve.[27] (The reverse cross-over in an ulnar-to-median direction at the forearm level is termed Marinacci anastomosis.[30–32]) At hand level, the so-called ''Riche-Cannieu'' anastomoses with exchange of fascicles between ulnar and median nerves (both directions possible) are less well defined.[24,25,33,34] After a median nerve injury, new symptoms occur immediately; at surgery, an acute, electric, shooting type of pain is noted to arise from the lesion distally to the supplied fingers, depending on the type of anesthesia applied (nerve block versus more superficial tissue infiltration). As a hallmark, a new deficit occurs after surgery and the symptoms occur

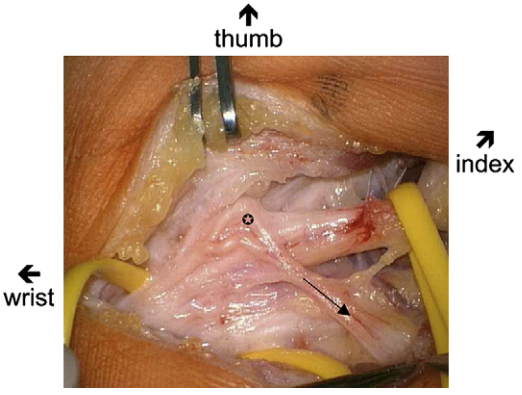

Fig. 2. Partial injury of left median nerve at the branching point of main trunk into common digital nerves due to attempted endoscopic CTR. Reexploration before dissection and microsurgical repair. Interfascicular trauma with openly displayed fascicles and scar formation can be appreciated (*star*). One fascicle group that naturally courses toward the thumb is reversed to the contralateral side (*arrow*).

acutely. Signs can be additional hypesthesia; more severe pain, which is changed in type and character (lancinating, shooting); additional or worsened numbness; and a new motor deficit. Acutely worsened and new symptoms should therefore raise the awareness for injury. As with other traumatic nerve injuries, a Hoffmann-Tinel's sign can be elicited when tapping on the skin overlying the nerve injury. Incomplete injury may especially present with a severe pain syndrome. Early exploration and reconstruction before development of a chronic pain syndrome is recommended. Incomplete or unsuccessful CTR should be differentiated from iatrogenic median nerve damage: in contrast to acute injury, symptoms do not change but preoperative complaints do not improve, and gradually become worse.

Spinal Accessory Nerve

Most traumatic lesions to the spinal accessory nerve are iatrogenic in origin.[7,35] By far the most frequent causative procedure is lymph node biopsy; Bostrom and Dahlin[36] recently described a 3% to 10% incidence. The injury is located in the posterior triangle of the neck (trigonum colli laterale). The nerve emerges underneath the lateral border of (or sometimes through) the sternocleidomastoid muscle at a distance of 7 to 9 cm from the clavicle[37] to enter the posterior triangle of the neck, which it traverses to pass underneath the trapezius muscle that it finally innervates.[38] Within its course, the nerve frequently passes in direct vicinity to a lymph node. When a lymph node biopsy is done with a minimal skin incision centered over the node and the readily palpable node is resected without a more careful circumferential dissection, it is all too easy to damage the nerve. The accessory nerve is frequently transected (**Fig. 3**), caused by tearing, cutting, or monopolar cautery around the node's fat pad. Despite being a "motor nerve," patients often describe a sharp, electric shock–like pain during the procedure (nociceptive fibers). The true extent of the damage usually becomes evident with delay. Initial complaints of shoulder pain are misinterpreted as "wound pain" or "omarthrosis." The patients experience pain and a "pulling" sensation in the neck. Attempts to elevate and rotate the arm internally and externally ("hair-combing movement") produce progressive discomfort. Unfortunately, the correct diagnosis is often not made until the pathognomonic pattern of muscle wasting and the alteration of the scapula's position and fixation on the thorax becomes obvious. Shoulder abduction still is possible but impaired, especially above the horizontal. The shoulder girdle is wasted and a groove develops between the neck and shoulder because of the lack of the trapezius bulk. The shoulder sags down because of the lack of pull from the upper trapezius portion that inserts at the scapular spine (**Fig. 4**). At the same time, the unopposed pull of the serratus anterior muscle at the inferior angle and anterior surface of the scapula pulls the scapula down and lateral in a direction "around the thorax" (see **Fig. 4**). In addition, the upper posterior trapezial coverage of the scapula is severely diminished (see **Fig. 4**C). In conjunction, these result in a variation of scapular winging also referred to as a "swing-position" (see **Fig. 4**A); the scapula is rotated outward, away from the spine, and pulled caudally (see **Fig. 4**B). It needs to be differentiated from the "classic" scapula alata that is attributed to long thoracic nerve damage and serratus anterior palsy. Usually, the correct diagnosis is made after considerable delay. In contrast to many other nerves, the spinal accessory still seems to fare well even after delayed microsurgical reconstruction. Useful functional results can still be expected after 9 months (partly because the distance to the target muscle is short).[7,35,39] During microsurgical exploration, it is at times difficult to detect the distal stump because it has retracted considerably; in some cases, the distal stump cannot be detected at all. The value of direct muscular neurotization by use of a coapted autologous nerve graft in such cases has not been validated on a larger scale. Detection of the proximal stump and differentiation from the sensory nerves at Erb's point can be aided by direct and even transcutaneous stimulation before skin incision; the proximal stump will conduct a retrograde potential to the more proximal accessory branch supplying the sternocleidomastoid.

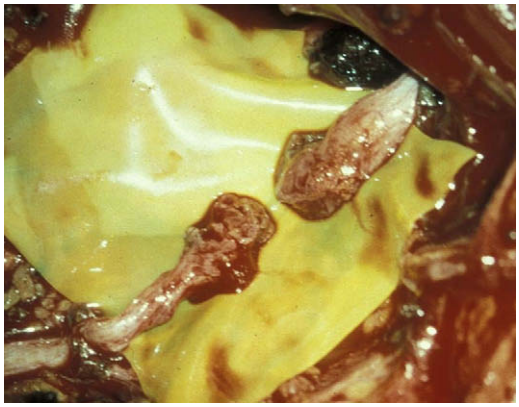

Fig. 3. Right spinal accessory nerve in the posterior triangle of the neck, found transected after lymph node biopsy.

In cases where the functional postoperative result has been disappointing, transfer of the levator scapulae and rhomboids might be an alternative

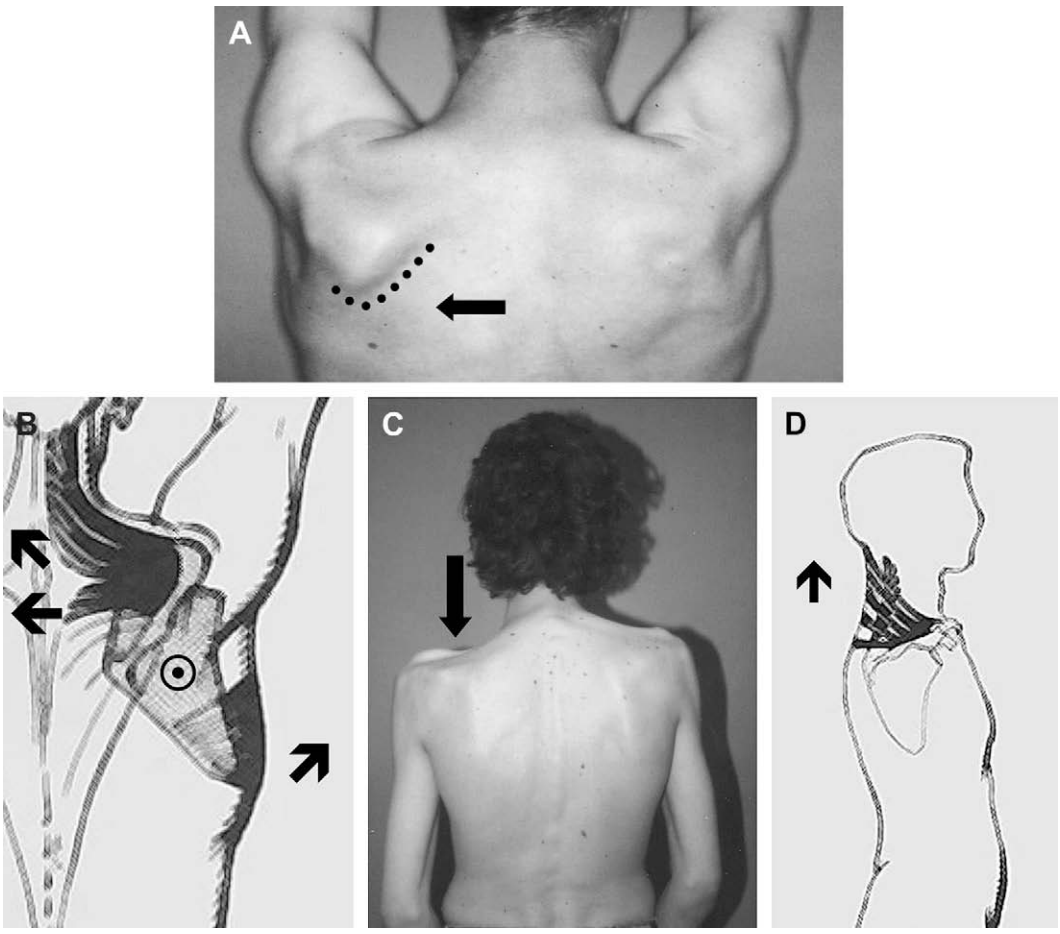

Fig. 4. Pathognomonic findings after spinal accessory nerve injury (*A*, *C*) in contraposition to physiologic trapezius function for fixation and movement of scapula (*B*, *D*). (*A*) Elevation of arms above 90° pronounces lack of trapezius covering scapula on left shoulder of this athlete. The dotted line marks the inferior angle of the scapula, which is now prominent in contrast to the right unaffected shoulder. Apart from lack of covering muscle, this is also the result of unopposed serratus anterior pull. The scapula is brought into a "swing-position" as it moves away from the spine and around the thorax in an anterolateral direction (*arrow*). (*B*) Simplified depiction of upper trapezius insertion on upper lateral aspect of scapula, and serratus anterior insertion on inferior angle and anterior scapular surface, resulting in counterclockwise turn of scapula around a central axis (⊙) on synergistic contraction, enabling elevation/abduction. Right-pointing arrow indicates the direction of pull of serratus anterior muscle lateral around the thorax. Left-pointing arrow indicates the pull of trapezius medially towards the spine. Upper-left-pointing arrow indicates the pull of upper trapezius medially and upwards. (*C*) Complete atrophy of the upper trapezius portion resulted in marked groove of left shoulder girdle and sagging of shoulder (*arrow*). The scapula also moved around the thorax in an anterolateral direction (swing-position). (*D*) The main function of the upper trapezius portion is to stabilize and lift the scapula in a cranial direction (*arrow*).

(Eden-Lange procedure).[40] Results apparently are not as favorable as the ones for scapular winging because of serratus anterior loss.

Radial Nerve

The radial nerve is most susceptible to injury where it spirals around the humerus, at its proximal forearm course, and at the distal wrist, where its sensory cutaneous branch emerges underneath the brachioradialis fascia. Not only does a dislocated humeral fracture put the radial nerve at risk, but so do subsequent open reduction and fixation. The radial nerve has been found plated to the humeral bone in all larger series (**Fig. 5**).[1,3,41] If the nerve was plated but is in discontinuity in conjunction with a grossly displaced fracture, it seems more likely that the nerve was initially torn by the fracture impact. At wrist level, Kirschner wire placement for radial fractures can pierce the superficial sensory branch. Apparently, mini incisions have been found safer than pure

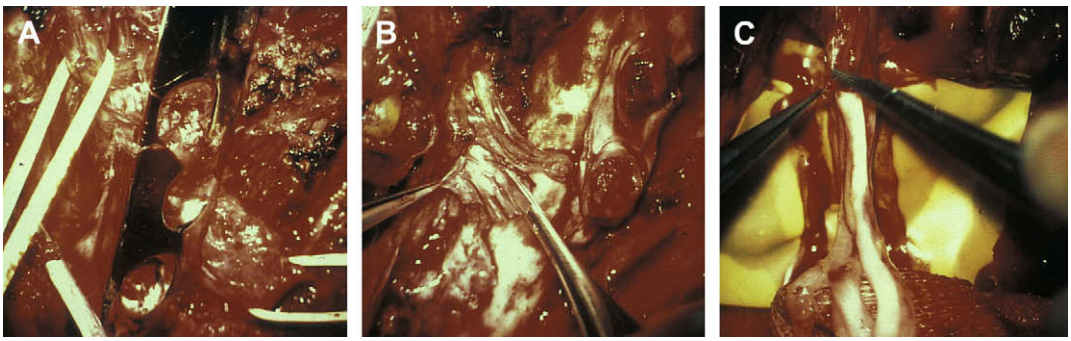

Fig. 5. Plating of radial nerve. (*A*) Plated midhumeral fracture. Radial nerve compressed under plate. Vessel loops around nerve lateral and medial to plate. (*B*) After plate removal, neuroma resection, and an interfascicular dissection prepared for graft coapted. (*C*) Sural nerve grafts coapted to proximal nerve stump with 10-0 suture.

percutaneous placement.[42] The radial nerve has an excellent prognosis for functionally useful recovery after reconstruction, with frequent full recovery of strong wrist extension after grafting, and functionally useful finger extension. In the rare case where nerve reconstruction is not an option, tendon transfers should be considered. The good motor outcome after radial nerve reconstruction is in sharp contrast to treatment results after injury to its cutaneous branch at wrist level, the superficial sensory radial nerve. Transections and partial injuries can result in painful neuropathies that are hard to treat. Among the numerous treatment approaches described, none has proved to be decisively more effective than proximal neuroma resection that allows for stump retraction underneath the brachioradialis muscle or decompression and external neurolysis of a microscopically intact nerve.[43]

Cutaneous and Digital Nerves

Damage to cutaneous and digital nerves happens far more often than comes to attention. If all cutaneous and digital nerves were grouped together, they would comprise a sizeable portion of all iatrogenic nerve injuries. When a sensory nerve is cut, dysesthesias occur and painful neuromas can develop. The symptoms can be out of proportion to the nerve's size, supplied area, and importance. Examples of frequently cut cutaneous branches prone to developing ailing symptoms are the superficial radial nerve at wrist level after Kirschner wiring of radioulnar fractures or needle stick injury (**Fig. 6**), the medial antebrachial cutaneous branch at elbow level (following ulnar nerve/cubital tunnel decompression),[44] digital nerves of the hand[45] (**Fig. 7**, after trigger finger release), the infrapatellar branch of the saphenous

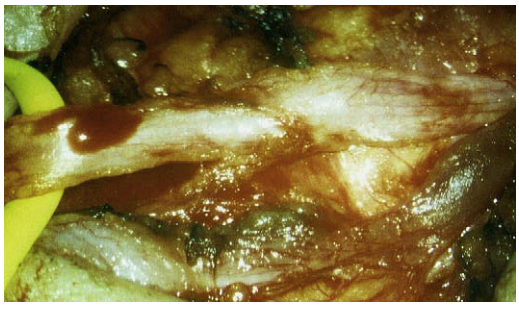

Fig. 6. Right superficial sensory radial nerve at wrist level after needle stick injury due to attempted vessel puncture, resulting in pain and paresthesia. Photograph depicts scar and neuromatous enlargement of the nerve from the midportion to the right. Interfascicular neurolysis revealed a monofascicular neuroma that was resected.

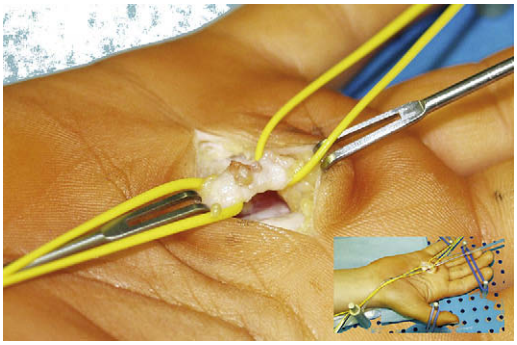

Fig. 7. Partial laceration of common digital nerve due to trigger finger release of the ring finger. Release aims at transection of the A1 flexor pulley at the metacarpal head level. Most commonly affected are the ring and middle finger and, to a lesser degree, the thumb. This patient complained of hypersensitivity and dysesthesia to the opposing sides of D IV and III and electrifying distally shooting pain on touch after such a release. The Hoffman-Tinel's sign was positive over the lesion, reproducing the pain. In this case, the previous incision at the distal palmar flexor crease was used to expose the nerve for microsurgical, split end-to-end repair.

nerve at knee level after arthroscopic and open knee procedures, and various cutaneous branches at the dorsum of the foot. Frequently, no rationale exists for reconstruction because it would imply sacrifice of another sensory nerve as an autograft. The usual options are neuroma resection and burial of the proximal stump in deeper tissue layers or decompression/neurolysis. Insertion of artificial guiding tubes might be an alternative when proximal and distal stumps can be located and sensation is functionally important (eg, digital branches to inner aspect of thumb and index). The guiding matrix might at least prevent formation of yet another end-bulb neuroma because axons will be provided with a target structure. By way of nerve conduits, distances of close to 3 cm can be overcome by sprouting axons until "reconnection to the distal stump."[46–50] It is our opinion that the high material expenditure for this indication has so far prevented its broader use. Interposition of a freeze-thawed muscle graft as a guiding bridge that provides the sprouting axons with a basal lamina substitute is not commonly performed.[49]

Trunk: Inguinal Nerves

The ilioinguinal, genitofemoral, and sometimes iliohypogastric nerves are cut, coagulated, sutured, or incorporated into a mesh at open and endoscopic hernia repairs.[51] Incidences of inguinal nerve damage after open and laparoscopic hernia repair are quoted as 0.5% to 2%.[52,53] If exploration is attempted from anterior by way of the old incision, it is often difficult to identify clearly the damaged nerve within the scar and incised fascia, unless a suture or clip can be identified around the nerve. So, often, only scar tissue and possibly a contained but not visualized neuroma can be resected.[54,55] Some investigators advocate neurolysis if a nerve can be found in scar or suture but is otherwise intact. At the same time, reexploration should not leave the abdominal wall weakened with potential for recurrent herniation. Others have recommended a retroperitoneal approach to allow for identification of the affected nerve proximal to healthy tissue.[52,53] Such an approach aims at neuroma resection and deep burial of the proximal stump to treat severe neuropathic pain. The neuroma that will form again will then be protected deep in tissue. The importance of retroperitoneal placement has been pointed out for the genital branch of the genitofemoral nerve.[56] Exact preoperative diagnosis of the affected inguinal nerve is then, however, mandatory. Despite theoretic differences in innervation pattern and sophisticated, diagnostic nerve blocks,[57] it often is not possible to clearly differentiate between genitofemoral and ilioinguinal damage. If a chronic pain syndrome has already developed ("central sensitization"), it is frequently difficult to obtain long-term relief with neuroma resection. Conservative therapy with serial infiltrations of local anesthetics and corticoids has its role before repeated surgical attempts.

Lower Extremity Nerves

Frequently, the common peroneal and the superficial and deep branch, the saphenous, its infrapatellar branch, and, less frequently, the tibial nerve are injured during orthopedic procedures and varicose vein treatment. Varicose vein surgery threatens all of the above-mentioned nerves (eg, ligation, stripping, and tearing out). The main femoral and sciatic trunk can be damaged during orthopedic procedures (**Figs. 8** and **9**) involving the femur and hip joint. Hip arthroplasty is a typical example (see **Fig. 8**).[58–61] Traction on the nerve during sustained extreme positional maneuvers, cement for artificial hip joints (thermal and mechanical damage), and the instrumentation as such, and cerclage for reinforcement of femoral fracture instrumentation are reported mechanisms. For injury to the sensory cutaneous nerves of the ankle and dorsum of the foot, Kirschner wiring, exostosectomy, and instrumentation for fracture treatment are frequent lesion mechanisms. Another procedure with high potential for peroneal and tibial nerve damage is Baker cyst removal at the popliteal fossa.

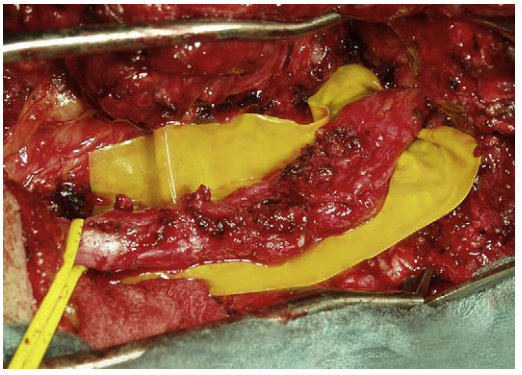

Fig. 8. Iatrogenic lesion of right sciatic nerve 6 months after cemented hip arthroplasty in a 60-year-old patient. The nerve was explored at an infrapiriformis level and found to be in partial continuity only. Cement fragments were additionally found around the nerve. Evaluation with nerve action potentials confirmed a complete injury with no conduction across the lesion, necessitating graft repair.

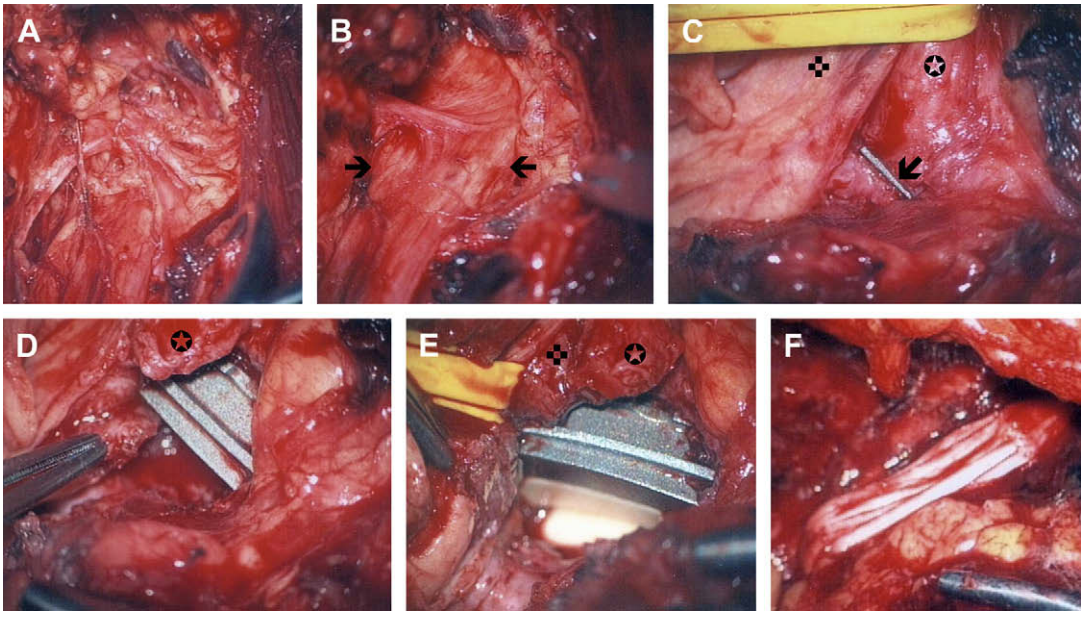

Fig. 9. Progressive blunt transection of right sciatic nerve with delayed onset. A 76-year-old woman suffered from progressive weakness of right foot dorsiflexion and L4/5 hyp- and dysesthesia, starting 5 months after implantation of a total hip endoprosthesis. Until then, she was able to drive her car. Within 2 months she developed a drop foot. Electric shock–like dysesthesias radiating from the lower buttock to the ankle severely aggravated her symptoms. At the time of presentation, she was unable to sit because of excruciating pain. Preoperative motor examination indicated the following: gluteus maximus and medius 5/5 (British Medical Research Council), biceps femoris 5/5, peronei 0/5, extensor hallucis longus/toe extensors 0/5, tibialis posterior 2/5, toe flexors 4–5/5, gastrocnemius 5/5. MRI excluded tumorous involvement; some scar could be appreciated in the area of the implant. Pelvic radiograph revealed no peculiarities. Findings at surgery: (*A*) A posterior approach to expose the sciatic nerve from the subgluteal fold to the notch area was chosen. Before the nerve was encountered, scar tissue was appreciable. (*B*) Eight centimeters caudal to the notch area, a severe adhesion of the sciatic became evident; the lateral circumference was found transected and embedded in scar tissue. Sciatic nerve and scar within arrows. (*C*) Further dissection revealed a metal strip abutting the nerve (*arrow*). (*D*) The metal edge was soon identified to be part of the acetabular joint thread, which was not completely embedded in bone. (*E*) The thread had completely transected the peroneal division of the sciatic (*star*), and a minor tibial portion (*cross*). (*F*) The neuromatous segments were resected for a split type of repair. In an attempt to prevent painful neuroma formation, the sciatic was reconstructed using eight strands of autologous sural nerve graft. A pedicled fat flap was created to cover the interspace between nerve and hip implant (hip revision was not an option). Postoperatively, the typical pain had vanished and despite her advanced age and the extensive approach, she was only on nonsteroidal antiinflammatory drugs for a week. After days, she ambulated on crutches. She remained pain free but, as predicted, did not recover from drop foot. Adequate management of high sciatic lesions with a severe pain component in the elderly is subject to controversy.

Other Nerves

A complete list of other nerves would be extensive. Some of the more notable examples are included here. During thyroid and anterior cervical spine surgery, the right recurrent laryngeal nerve is at risk for compression from retractor blades, but most will recover spontaneously.[62,63] Transections have been reported only rarely. Wisdom tooth extraction can damage lingual and inferior alveolar nerves;[64–66] the lesion rate is five times higher under general than local anesthesia. Cranial and perimastoid incisions can damage the facial and occipital nerve branches.

FAULTY RESECTION OF BENIGN NERVE SHEATH TUMORS WITH NERVE

Every nerve surgeon comes across cases where a whole nerve segment or parts of the brachial plexus have been "eradicated" en bloc on purpose, together with a "malignant-looking tumor." Neurofibromas and schwannomas are often preoperatively not identified as such; percutaneous needle biopsies are contraindicated because they can damage the nerve and are not diagnostic enough to differentiate between benign and malignant tumors. Kehoe and colleagues[67] found that in 88 pathologically proven neurofibromas or

schwannomas resected between 1959 and 1990, the correct diagnosis was only made in 7 cases, or 8%. It must be mentioned, however that most of the cases referred to in this study had been treated before more sophisticated MRI sequences were in widespread use. Benign schwannomas can be resected microsurgically without functional deficit, which also applies to the technically more demanding neurofibroma resection. Because nerve tumors are rare, the number of surgeons who have experience with this tumor entity is small. That is why, once the diagnosis is suspected, this type of surgery should be reserved for experienced nerve surgeons. An important aspect is the low predictive value of MRI in discerning malignant from benign tumors. A peripheral nerve sheath tumor with features suggesting malignancy on imaging (eg, cystic or ''ancient variants'') might intraoperatively turn out to be benign, if approached and dissected in the proper way. En bloc resections of benign nerve tumors are obviously contraindicated; nevertheless, they happen. Such lesions should be explored and grafted as soon as possible.[68] Lipoma resection in proximity to nerves predisposes to nerve damage when undertaken by way of minimized skin incisions without careful circumferential dissection (comparable to lymph node biopsy; see above).

Indirect Damage and Nonoperative Causes

Injection injury

The most common neural injection sites are the sciatic nerve at buttock level and the radial nerve at the lateral upper arm.[69,70] Marantic adults and children are at higher risk because of thinner soft tissue layers. Higher case rates of gluteal injection injuries have been reported from countries where serial intramuscular injections were performed (eg, chloroquine malaria prophylaxis). These injuries more frequently affect children under age 5.[71–73] In principle, intraneural injection needs to be differentiated from perineural.[2] In their experimental study, Gentili and colleagues[74,75] stressed the particular danger of intrafascicular injection. Signs of intraneural injection are instant electric shock–like pain radiating down the limb, with paresthesias, radicular burning pain, and numbing and complete or incomplete paralysis. A delayed onset of symptoms, usually in conjunction with a severe burning component, points more toward perineural deposition of an agent. Apparently, in 10% of cases, symptoms present with delay.[76] It is important to clarify if the injected agent was neurotoxic because the instant damage and the developing reactive intraneural fibrosis will be worse. Among the more commonly used intramuscular agents, diazepam, chlorpromazine, and dexamethasone are neurotoxic. For neurotoxic agents, immediate exploration is a consideration. Two slightly different approaches to injection injuries have been described. One is an initially more observant one,[77] the other is urgent exploration in cases of instant paralysis following injection of a neurotoxic agent within the course of a main nerve.[1,2] Instant exploration and epineurotomy aim to prevent delayed internal compression by the fibrosis that inadvertently will occur because of neurotoxicity. Irrigation with saline is used in an attempt to dilute the agent. If in severe pain, Bonney[2] advised leaving a perineural catheter in place for direct analgesia with a lignocaine or bupivicaine solution. However, chances for instant referral of such a patient will be close to zero. If, theoretically, 2 to 3 days after the event, a patient were referred with injection injury, Dr. Bonney would still recommend the outlined procedure if the patient had persistent severe pain, complete paralysis within hours of injection, or progression of paralysis. Fortunately, most injection injuries will show spontaneous recovery and have no element of neurotoxicity, which is why the New Orleans group and others are more inclined most of the time to observe recovery over a 2- to 4-month period. Recovery, though, takes a long time and likely will be incomplete. In the absence of progress, grafting or split repair of the damaged segment might be necessary. Ongoing pain or severe deficits are then seen as indications for exploration. If no nerve action potential can be recorded across the injured segment intraoperatively, the lesion is resected and grafted. The best preventive measures include knowledge of pertinent anatomy and landmarks, appropriate needle length in conjunction with proper angle of injection, and accounting for the body habitus of the patient rather than injecting in a stereotyped manner.

Compressive Arterial Bleeding

At particular risk are the supra-and infraclavicular brachial plexus after attempted central line insertion, the brachial artery in the arm (attempted catheterization may cause bleeding into the fascial sheath affecting the median nerve), the ulnar artery at the elbow (blood collection in the deep forearm compartment affecting the ulnar nerve), the radial artery at the wrist (median nerve compression), the femoral artery at the groin (catheter angiography affecting the femoral nerve), and the aorta in the abdomen (bleeding down the psoas sheath affecting the lumbar plexus). If findings on physical examination are not obvious (large groin

hematoma after catheter angiography and progressive pain or nerve deficits), MRI is helpful. MRI certainly is indicated to rule out potential intrapelvic or retroperitoneal hematomas, when new postoperative progressive deficits cannot be explained after orthopedic, abdominal, or vascular procedures in the vicinity of these spaces. Elderly patients under anticoagulation who undergo joint replacement are at particular risk for extraneural bleeding into a compartment causing nerve compression. A common procedure is hip arthroplasty. Intraneural hemorrhage after administration of anticoagulants like warfarin and heparin has also been described. The concurrent administration of heparin and warfarin with nonsteroidal anti-inflammatory drugs can potentiate their anticoagulative effects.[78,79] Sizeable hemorrhages resulting in symptomatic nerve compression need to be evacuated. Unfortunately, this group of patients is also at higher risk for rehemorrhage after hematoma evacuation, so placement of a drain after meticulous hemostasis has been achieved should be considered.

Tourniquet Palsy

The pathognomonic features of tourniquet palsy are that all nerves of an extremity are affected to various extents and a Hoffmann-Tinel's sign is absent. Incidences of 1 in 7000 surgeries have been quoted.[80] Several scenarios are conceivable. The chosen pressure above the patient's systolic blood pressure is too high because the surgeon does not know better (unlikely), or, more likely, the pressure is actually higher than what the manometer indicates. Safe time parameters are exceeded or the patient's body habitus has not been accounted for (eg, thin extremity). The pressure cuff can be too narrow for the extremity circumference, and multiple combinations of the above mentioned mechanisms. Recommended are cuffs of at least 14 cm width for the adult arm, and of 18 cm for the adult thigh. Application should be at the proximal part of the extremity with padding between cuff and skin. Recommended inflation pressures are 50 to 75 mm Hg above systolic for the upper limb, and 100 to 150 mm Hg above systolic for the lower limb. But recommendations vary, and 100 mm Hg above systolic is frequently used for the upper limb. A method to minimize the necessary pressure to the minimal effective pressure that maintains arterial closure has been suggested.[81] Inflation time should not substantially exceed 1.5 hours for the upper extremity, and 2 hours for the leg.[2,19] Tourniquets cannot be used over an arterial prosthesis. Some recommend releasing the cuff after 1 hour to allow for some bleeding before reinflation. It is only prudent to adjust these guiding measures and time frames to the actual findings of body habitus and to take neuropathy-predisposing factors into account (alcoholism, diabetes, renal insufficiency, inherited disorders, and so forth). An important point is to check the accuracy of the manometer regularly.

Bonney pointed out that, in principle, any inflation of a cuff produces an ischemic nerve lesion if pressure is maintained for more than 20 minutes. However, this lesion is transient and resolves quickly when the cuff is deflated. Several investigators studied and discussed the effects of a tourniquet on muscle and nerve to find out if pressure or ischemia is the primary pathogenetic mechanism.[19,82,83] A case report of tourniquet paralysis that was meticulously followed electrophysiologically demonstrated how a combination of axonal degeneration and conduction block resolves over time.[84]

Closed Pressure or Traction During General Anesthesia

Causes are intraoperative positioning with excess pressure against body parts, where nerves lie superficially and course around or in close proximity to bone (eg, peroneal nerve at the fibular neck and ulnar nerve at the medial epicondyle), extensive pull, and constriction by inappropriately narrow straps (nerves at wrist).[85–87] At times, no distinct mistake in positioning and padding is identifiable but rather, an unfavorable combination of predisposing factors has to be accounted for,[86] including extremes of body habitus, preexisting compressive neuropathy, neuropathy in diabetics, alcoholics, dialysis patients, or hereditary neuropathy with liability to pressure palsies, and extremes of body position for a sustained amount of time (eg, lithotomy and femoral nerve lesion, lateral decubitus position and peroneal nerve injury, taping down of the shoulders resulting in brachial plexus stretch injury). All these predisposing factors are augmented by general anesthesia and use of muscle relaxants. Excessive shoulder pull (eg, taping down the shoulders with the head turned to the contralateral side) and shoulder abduction under general anesthesia are to be avoided (preferably not more than 70°); combining shoulder abduction with lateral rotation at the glenohumeral joint poses an additional risk. Yet some of these positions might be unavoidable at times (eg, for approaches that necessitate access to the inner aspect of the arm and the axilla). It is good practice to test the range of motion preoperatively with the patient awake. Bone-to-bone contact needs to be prevented by pads (eg, knees). The additional

importance of hand positioning (palms up, hand supinated) when the patient is placed supine with the arms at the side to prevent compression of the ulnar nerve at elbow level has been pointed out.[77,88] The ulnar is one of the nerves most frequently affected from malpositioning. An unfavorable position is full extension of the elbow, when forearm and hand are pronated. The pressure points are in the olecranon notch or groove. Full elbow flexion is also unfavorable.[85,86]

Irradiation

The brachial plexus is the usual site of radiation neuropathy. It appears with a time lag of several years, usually after radiation for breast carcinoma or malignant neck tumors.[89] Features from history (timing or high radiation dose), physical examination (eg, skin sclerosis, telangiectasias), and electrophysiology (eg, myokymia, continuous quivering and undulation of skin surface, and spontaneous repetitive discharge of motor unit potentials) give decisive clues.[2,90] Pain and sensory symptoms are the first features, followed by motor deficit. In the beginning, symptoms might point toward the median or ulnar nerve and to a lower trunk level, and therefore they can be mistaken for carpal or cubital tunnel syndrome. It is important to include metastasis in the differential diagnosis and to obtain an MRI (or fluordeoxyglucose–positron emission tomography). The plexus elements and the surrounding soft tissue, including muscle and vascular tissue, undergo progressive sclerosis. Consequently, the nerve not only develops a diffuse intrinsic lesion but also is entrapped in scar. The role of surgery is limited to microsurgical decompression by division of the epineurium and overlying sclerotic tissue. Some investigators with considerable experience also advocate transfer of muscle or omentum to attempt revascularization. Surgery mainly aims at pain reduction and slowing of progression. Graft reconstruction is unsuccessful.

Differential Diagnosis Brachial Plexitis

Brachial plexitis, or neuralgic amyotrophy, is known under the eponym Parsonage-Turner syndrome[91–93] and thought to be inflammatory or immune-mediated in nature. It occurs after physical stress and exertion. The underlying pathomechanism still is unrevealed. It also has been diagnosed perioperatively.[94] In contrast to iatrogenic injury to the brachial plexus, its onset is delayed. The time course, intense initial shoulder girdle pain, and the subsequent and delayed onset of shoulder and limb weakness discern it from the more focal traumatic lesions to the brachial plexus. The syndrome affects several peripheral nerve distributions in a patchy pattern that could not possibly stem from a focal cord to nerve origin (suprascapular, radial, axillary, long thoracic). To complicate matters, cases of single nerve, and bilateral plexus affection, have been described.[91]

MANAGEMENT PRINCIPLES

For treatment of iatrogenic nerve lesions, the same principles apply as for any other traumatic nerve injury. Attempts at minimizing time delay are most important for the functional prognosis of reconstructed nerves. However, as discussed, injection injuries, positioning injuries, and radiogenic neuropathy are approached differently.

Delayed Referral

Rarely, patients will be referred from the primary surgeon. Only one third of our patients could be operated on within an appropriate time interval of 6 months after the injury. Other groups report similar experiences. Frequently, patients are referred through a neurologic colleague who has been consulted on the patient's own initiative because of sustained sensorimotor deficits or painful sequelae.

Prognostic Factors Nonconditional to Surgeon

These factors are given by the type, mechanism, and level of the lesion, nerves affected, concomitant injuries, and patient age, and as such, are not conditional to the treating surgeon. Isolated, focal nerve injuries without concomitant severe arterial or bony component in younger patients generally will fare better, as do radial and spinal accessory lesions if compared with peroneal nerve injuries.[95]

Prognostic Factors Conditional to Surgeon

Other factors, however, are conditional to the treating surgeon, namely, judgment, timing of repair, and technique used. A treatment algorithm relating to injury type, with timing and repair technique, is given in **Fig. 10**. Timing of reconstructive surgery is a crucial factor for functional, useful recovery.[96,97] In general, functional outcome is adversely affected if more than 6 months have passed between injury and repair.[98–102] Repair should be attempted as early as possible, when the lesion has been judged to have insufficient potential for useful spontaneous recovery. Progressive neuronal death, ischemia, and fibrous proliferation are important limiting factors for useful recovery.[103] Experimental evidence exists for progressive neuronal death to occur after a critical

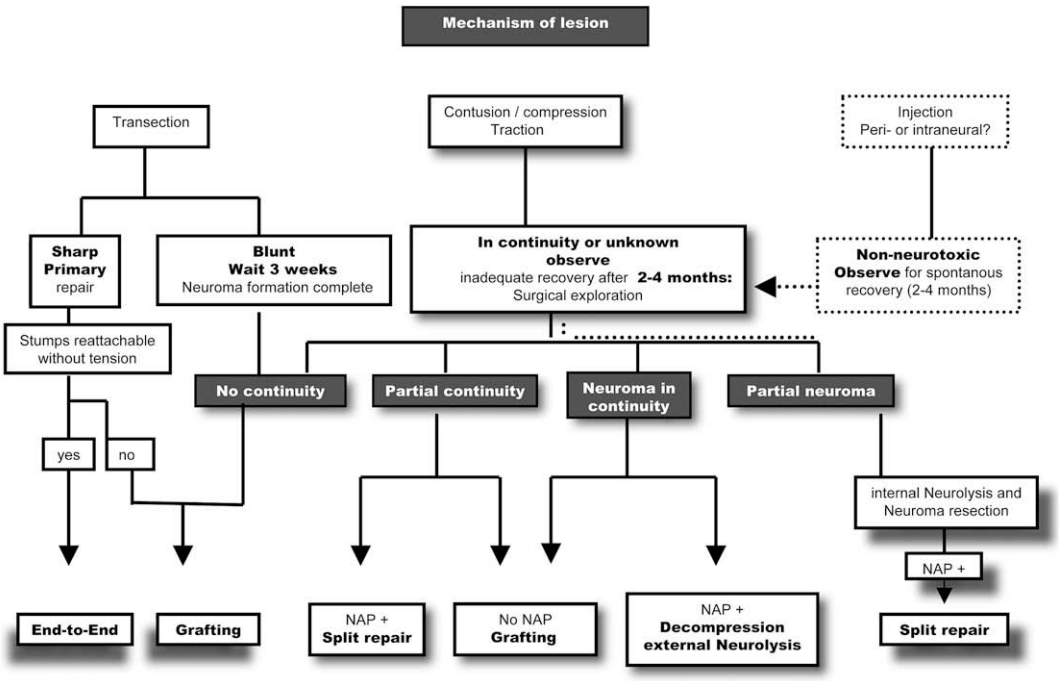

Fig. 10. Treatment algorithm for iatrogenic nerve injuries, specifying timing and different repairs depending on injury type. NAP, nerve action potential.

time window has passed. Sensory and motor neurons appear to differ in the onset of such cell loss. Evidence also indicates that early nerve repairs can stop this process of neuron loss.[104] It seems that early repair improves the reconstructed nerve's regenerative capacity. In summary, the efficacy of axonal regeneration is significantly affected by the amount of cell loss already present at the time of repair.[105–108] As far as technique is concerned, microsurgical principles apply and, as such, microinstruments, magnification devices, and proper illumination are mandatory for attempts at repair and neurolysis.

Appropriate Treatment

For those who inflicted a severe injury, we recommend contacting an experienced nerve surgeon. The first step is to evaluate the lesion mechanism. The most frequent intraoperative direct causes are transection, stretch, contusion, compression, and combinations thereof. Furthermore, the continuity of the lesion needs to be evaluated, and an attempt at grading the degree of damage helps to judge the potential for spontaneous recovery. Good electrophysiologic examinations can be a helpful adjunct. Despite the accentuated fragmentation of the lesion types outlined below, one should be aware that an affected nerve segment could show a spectrum of lesion degrees. For instance, a partial laceration with neuromatous change of the flanking fascicles can exhibit only neurapractic changes at the marginal fascicles. Injection and thermal injuries will most likely be observed first (see earlier discussion and **Fig. 10**).

Sharp Transection

After sharp transection, immediate primary end-to-end suture is indicated. Nevertheless, instant retraction of the nerve ends might sometimes necessitate an interpositional graft or a transpositional maneuver to gain nerve length. Only the inflicting surgeon can judge if the nerve is sharply transected. However, straightforward cases like these are rare in clinical practice.

Blunt Transection

A waiting interval of three weeks is recommended if the transection is due to pull and tear (eg, stretch with tear, or drilled and wound-up nerve), and as such has a blunt element with contused margins, and if such a mechanism cannot be ruled out. The interval allows for complete formation of the stump neuromas along the longitudinal axis.[96] Then, an ''early secondary repair'' will be performed. The idea is to prevent coaptation of two putatively healthy-looking stumps that have no potential for recovery. The trauma might have had a far more proximal impact than could possibly

be appreciated with exploration at the time of injury. In other words, it would not be clear how far back the stumps needed to be resected to meet healthy fascicular structure that does not transform into neuroma. Such a neuromatous formation would hinder successful outgrowth of axons because of its fibrous tissue barrier.

At repair, nerve stumps are cut back to healthy-looking fascicular structure, and the resulting gap is bridged with interpositional grafts. Indicators of ''healthy'' fascicular structure are a moist, glossy-looking surface with protruding fascicles, and lack of scar tissue; if in doubt, a frozen section can help.

Mechanism Unknown or Lesion in Continuity

Most nerve injuries will only be detected in the postoperative course because of the inflicted symptoms of pain and sensorimotor deficits. As a rule, the signs and symptoms will occur directly after surgery. For an extremely rare exception, we refer to **Fig. 9**. Unless the inflicting surgeon knows better, the extent and type of injury will be unclear (sharp or blunt transection). The spectrum ranges from a lesion in continuity with potential for spontaneous recovery (focal conduction block, neurapraxia) to a transection (neurotmesis). In such cases, an observational period is indicated to monitor for spontaneous recovery. Sweating in the autonomous zone of a nerve precludes a complete lesion. If regenerative signs are insufficient after a 2- to 4-month period (adjusted to level of injury), the nerve is explored; unclear lesions in continuity are intraoperatively evaluated with nerve action potentials.[77] In conjunction with palpation and microscopic inspection, potential for spontaneous recovery is assessed. Graft reconstruction implies at least a Sunderland lesion type III with a Millesi type C fibrosis.[109,110] By means of a three-prong proximal stimulation electrode and a distal two-prong recording electrode, a test is done to see if a self-conducting compound nerve action potential can be elicited across the lesion. If a potential can be recorded, the microsurgical intervention is limited to decompression from surrounding scar and external neurolysis. If no potential can be recorded, the lesioned segment is excised and the resulting gap bridged with an interpositional nerve graft. Sometimes, only a sector of the nerve is so severely damaged that it needs graft reconstruction. If the rest of the circumference still conducts and contains healthy fascicles, a split nerve repair is indicated, which implies at least partial internal neurolysis to split the neuromatous portion from the sector with intact fascicular pattern. Graft interposition in split repairs is pleasing because the healthy nerve portion splints the graft bundle.

OUTCOMES

Outcomes depend on many different factors: the specific nerve affected, timing of repair, age of patient, level and type of lesion, and associated injuries most likely better as concomitant injuries. Reconstructive measures of nerve injury beyond 9 months have been associated with poorer results. It appears that appropriate microsurgical measures initiated in a timely manner can result in useful outcomes in more than two-thirds of cases. Seventy percent of all the iatrogenic injuries operated on in our series improved.[4] In view of the fact that that two thirds of these patients were not referred in a timely manner (delay more than 6 months post trauma), and that repairs included nerves with poorer prognosis (peroneal injury), these results should encourage attempts at early microsurgical repair. Favorable results usually can be obtained with accessory and radial nerve reconstruction. Most patients who have positioning-related nerve injuries will not require surgery. Incomplete lesions recover about 90% of the time,[85] but recovery may be lengthy. Sometimes, a good functional outcome is precluded because a patient is involved in litigation. Perioperative Parsonage-Turner syndrome is treated conservatively and has a reasonably good prognosis regarding pain and recovery of functionality. Frequently however, recovery will be incomplete and take months to years; residua like scapular winging might remain. As an adjunct to clinical examination, progress can be monitored by objective changes in MRI and electrophysiology.

MEDICOLEGAL ASPECTS

Injuries to peripheral nerves represent a sizeable proportion of medical liability cases. We assume that few of theses cases are the result of true negligence. A survey from the 1950s calculated a perioperative nerve lesion rate of 1 in 1000 operations during a 6-year period.[111] Bonney[112] remarked 22 years ago, ''It is hard to know whether there has in fact been an increase in the incidence of negligence, or whether the standards by which doctors are judged have risen, or whether instances of negligence previously hidden by the enormous cost of legal action are now being revealed.''

To date, the only study that specifically screened for peripheral nerve injuries among such cases evaluated the records of 2500 medical liability proceedings for the year 1984 in a former

East German county. Muller-Vahl and colleagues' analysis revealed that 638 of the cases were based on neurologic symptoms. Of these, 13% were due to direct iatrogenic damage of the involved part of the nervous system: 90% of these affected peripheral and cranial nerves and 60% of these lesions were inflicted during surgery. Injection and needle stick accounted for 22%; the remainder were due to positioning/bandage/tourniquet palsy (12%), medication (2%), radiation (1%), and other (3%). Unfortunately, longitudinal updates are not available.

It was calculated that 98.609 adverse events in 2.671863 patients occurred. Twenty-eight percent, or 27,179 of these adverse events, involved negligence.[113] Estimates based on two United States studies and one United Kingdom study suggest that negligence in the National Health Science in England may cause around 90,000 adverse events per year, involving 13,500 deaths. These adverse events apparently result in around 7000 claims (7.7% of adverse events) and 2000 payments (2.2% of adverse events).[114] The rate of nerve injuries was not extracted.

Within 4 years, from 2000 through 2003, the North German ''Schlichtungsstelle'' concluded 10,513 panel proceedings on medical negligence. More than 75% related to surgical and postoperative therapy. Twenty-five percent of patients claimed deficient doctor–patient communication in their initial correspondence to the panel.[115] Clearly, good communication with the patient and documentation of such conversations are important once an adverse event has occurred.

It is also obvious that precise operating room notes can be a crucial factor to support that, in fact, an injury was not the result of true negligence. Operating room notes can serve to demonstrate awareness of procedure-inherent specific risks and adequacy of preventive measures. Evident postoperative nerve damage must be addressed, treated, and likewise documented. Having records that account for presumptive diagnosis and differential, and the measures that were taken, will not only be a major step to prevent litigation, but will also inevitably lead to early and adequate therapy. Consultation with an experienced nerve surgeon is highly advisable, especially if the primary surgeon does not have enough experience with evaluation and treatment of traumatic nerve injuries.

SUMMARY

Iatrogenic nerve lesions are not infrequent; their treatment, however, is frequently inappropriate. Delayed diagnosis and referral prevail. In contrast, timely intervention has a good chance of improving the outcome. Patients benefit from early diagnosis and treatment management by a specialist in peripheral nerve surgery. Fear of litigation should not prevent proper treatment. Apart from initiation of appropriate treatment, proper documentation and communication with the patient are paramount to circumvent and confront allegations of negligence.

REFERENCES

1. Birch R, Bonney G, Parry CW. Iatropathic injury. Surgical disorders of the peripheral nerves. Edinburgh (UK): Churchill Livingstone; 1998. p. 293–333.
2. Bonney GLW. Iatropathic lesions of peripheral nerves. Curr Orthop 1997;11:255–66.
3. Khan R, Birch R. Iatropathic injuries of peripheral nerves. J Bone Joint Surg Br 2001;83(8):1145–8.
4. Kretschmer T, Antoniadis G, Braun V, et al. Evaluation of iatrogenic lesions in 722 surgically treated cases of peripheral nerve trauma. J Neurosurg 2001;94(6):905–12.
5. Kim DH, Murovic JA, Tiel R, et al. Management and outcomes in 353 surgically treated sciatic nerve lesions. J Neurosurg 2004;101(1):8–17.
6. Kim DH, Kline DG. Surgical outcome for intra- and extrapelvic femoral nerve lesions. J Neurosurg 1995;83(5):783–90.
7. Kim DH, Cho YJ, Tiel RL, et al. Surgical outcomes of 111 spinal accessory nerve injuries. Neurosurgery 2003;53(5):1106–12 [discussion: 1102–3].
8. Wilbourn AJ. Iatrogenic nerve injuries. Neurol Clin 1998;16(1):55–82.
9. Stöhr M. Iatrogene nervenläsionen. Injektion, operation, lagerung, strahlentherapie. 2nd edition. Stuttgart (Germany): Thieme; 1996.
10. Komurcu F, Zwolak P, Benditte-Klepetko H, et al. Management strategies for peripheral iatrogenic nerve lesions. Ann Plast Surg 2005;54(2):135–9 [discussion: 140–2].
11. Dellon AL. Invited discussion: management strategies for iatrogenic peripheral nerve lesions. Ann Plast Surg 2005;54(2):140–2.
12. Kretschmer T, Antoniadis G, Borm W, et al. [Iatrogenic nerve injuries. Part 1: frequency distribution, new aspects, and timing of microsurgical treatment]. Chirurg 2004;75(11):1104–12.
13. McGeorge D, Sturzenegger M, Buchler U. Tibial nerve mistakenly used as a tendon graft. Reports of three cases. J Bone Joint Surg Br 1992;74(3):365–6.
14. Geldmacher J. Median nerve as free tendon graft. Hand 1972;4(1):56.
15. Weber RV, Mackinnon SE. Median nerve mistaken for palmaris longus tendon: restoration of function with sensory nerve transfers. Hand 2007;2:1–4.

16. Oppikofer C, Tschopp H. Tibial nerve used as a tendon graft. Helv Chir Acta 1990;57:923–9.
17. Swan H, Virtue RW, Blount SG Jr, et al. Hypothermia in surgery; analysis of 100 clinical cases. Ann Surg 1955;142(3):382–400.
18. Birch R, Bonney G, Dowell J, et al. Iatrogenic injuries of peripheral nerves. J Bone Joint Surg Br 1991;73(2):280–2.
19. Tackmann W, Richter H-P, Stöhr M. Kompressionssyndrome peripherer nerven. Berlin: Springer; 1989.
20. Kretschmer T, Antoniadis G, Borm W, et al. [Pitfalls of endoscopic carpal tunnel release]. Chirurg 2004;75(12):1207–9 [in German].
21. Ferrari GP, Gilbert A. The superficial anastomosis on the palm of the hand between the ulnar and median nerves. J Hand Surg [Br] 1991;16(5):511–4.
22. Lanz U. Anatomical variations of the median nerve in the carpal tunnel. J Hand Surg [Am] 1977;2(1): 44–53.
23. Stancic MF, Micovic V, Potocnjak M. The anatomy of the Berrettini branch: implications for carpal tunnel release. J Neurosurg 1999;91(6):1027–30.
24. Cannieu J. Note sur une anstomose entre la branch profonde du cubital et lemedian. Bull Soc d'Anat Physiol Bordeaux 1897;18:339–49.
25. Riche P. Le nerf cubital et les muscles de l"eminence thenar. Bulletins et Mémoires de la Société d'Anatomie de Paris 1897;5:251–2.
26. Gruber W. Ueber die Verbindung des Nervus intermedius mit dem Nervus unaris am Unterarme des Menschen und der Saeugetiere. Archiv für Anatomie und physiologische wissenschaftliche Medizin leipzig 1870;37:501–22.
27. Lee KS, Oh CS, Chung IH, et al. An anatomic study of the Martin-Gruber anastomosis: electrodiagnostic implications. Muscle Nerve 2005;31(1):95–7.
28. Taams KO. Martin-Gruber connections in South Africa. An anatomical study. J Hand Surg [Br] 1997;22(3):328–30.
29. Martin R. Tal om Nervers allmanna Egenskaper i Manniskans Kropp. Stockholm (Sweden): L Salvius; 1763.
30. Meenakshi-Sundaram S, Sundar B, Arunkumar MJ. Marinacci communication: an electrophysiological study. Clin Neurophysiol 2003;114(12):2334–7.
31. Stancic MF, Burgic N, Micovic V. Marinacci communication. Case report. J Neurosurg 2000;92(5):860–2.
32. Marinacci A. Diagnosis of "all median hand." Bull Los Angel Neuro Soc 1964;29:191–7.
33. Kimura I, Ayyar DR, Lippmann SM. Electrophysiological verification of the ulnar to median nerve communications in the hand and forearm. Tohoku J Exp Med 1983;141(3):269–74.
34. Gutmann L. AAEM minimonograph #2: important anomalous innervations of the extremities. Muscle Nerve 1993;16(4):339–47.
35. Bazner UM, Braun V, Richter HP, et al. [Management of iatrogenic lesions of the spinal accessory nerve]. Nervenarzt 2005;76(4):462–6 [in German].
36. Bostrom D, Dahlin LB. Iatrogenic injury to the accessory nerve. Scand J Plast Reconstr Surg Hand Surg 2007;41(2):82–7.
37. Kierner AC, Zelenka I, Heller S, et al. Surgical anatomy of the spinal accessory nerve and the trapezius branches of the cervical plexus. Arch Surg 2000;135(12):1428–31.
38. Aramrattana A, Sittitrai P, Harnsiriwattanagit K. Surgical anatomy of the spinal accessory nerve in the posterior triangle of the neck. Asian J Surg 2005; 28(3):171–3.
39. Matz PG, Barbaro NM. Diagnosis and treatment of iatrogenic spinal accessory nerve injury. Am Surg 1996;62(8):682–5.
40. Coessens BC, Wood MB. Levator scapulae transfer and fascia lata fasciodesis for chronic spinal accessory nerve palsy. J Reconstr Microsurg 1995; 11(4):277–80.
41. Kim DH, Kam AC, Chandika P, et al. Surgical management and outcome in patients with radial nerve lesions. J Neurosurg 2001;95(4):573–83.
42. Hochwald NL, Levine R, Tornetta P 3rd. The risks of Kirschner wire placement in the distal radius: a comparison of techniques. J Hand Surg [Am] 1997;22(4):580–4.
43. Vernadakis AJ, Koch H, Mackinnon SE. Management of neuromas. Clin Plast Surg 2003;30(2): 247–68, vii.
44. Stahl S, Rosenberg N. Surgical treatment of painful neuroma in medial antebrachial cutaneous nerve. Ann Plast Surg 2002;48(2):154–8 [discussion: 158–60].
45. Carrozzella J, Stern PJ, Von Kuster LC. Transection of radial digital nerve of the thumb during trigger release. J Hand Surg [Am] 1989;14(2 Pt 1): 198–200.
46. Belkas JS, Shoichet MS, Midha R. Peripheral nerve regeneration through guidance tubes. Neurol Res 2004;26(2):151–60.
47. Archibald SJ, Krarup C, Shefner J, et al. A collagen-based nerve guide conduit for peripheral nerve repair: an electrophysiological study of nerve regeneration in rodents and nonhuman primates. J Comp Neurol 1991;306(4):685–96.
48. Mackinnon SE, Dellon AL. Clinical nerve reconstruction with a bioabsorbable polyglycolic acid tube. Plast Reconstr Surg 1990;85(3):419–24.
49. Lundborg G. Alternatives to autologous nerve grafts. Handchir Mikrochir Plast Chir 2004;36(1): 1–7.
50. Dahlin LB, Lundborg G. Use of tubes in peripheral nerve repair. Neurosurg Clin N Am 2001;12(2): 341–52.

51. Nahabedian MY, Dellon AL. Outcome of the operative management of nerve injuries in the ilioinguinal region. J Am Coll Surg 1997;184(3):265–8.
52. Muto CM, Pedana N, Scarpelli S, et al. Inguinal neurectomy for nerve entrapment after open/laparoscopic hernia repair using retroperitoneal endoscopic approach. Surg Endosc 2005;19(7): 974–6.
53. Krahenbuhl L, Striffeler H, Baer HU, et al. Retroperitoneal endoscopic neurectomy for nerve entrapment after hernia repair. Br J Surg 1997;84(2): 216–9.
54. Kim DH, Murovic JA, Tiel RL, et al. Surgical management of 33 ilioinguinal and iliohypogastric neuralgias at Louisiana State University Health Sciences Center. Neurosurgery 2005;56(5): 1013–20 [discussion: 1013–20].
55. Liszka TG, Dellon AL, Manson PN. Iliohypogastric nerve entrapment following abdominoplasty. Plast Reconstr Surg 1994;93(1):181–4.
56. Ducic I, Dellon AL. Testicular pain after inguinal hernia repair: an approach to resection of the genital branch of genitofemoral nerve. J Am Coll Surg 2004;198(2):181–4.
57. Choi PD, Nath R, Mackinnon SE. Iatrogenic injury to the ilioinguinal and iliohypogastric nerves in the groin: a case report, diagnosis, and management. Ann Plast Surg 1996;37(1):60–5.
58. Schmalzried TP, Amstutz HC, Dorey FJ. Nerve palsy associated with total hip replacement. Risk factors and prognosis. J Bone Joint Surg Am 1991;73(7):1074–80.
59. Henrich M. [Sciatic nerve: special topography and possibility of injuries in surgery of the hip joint (author's transl)]. Langenbecks Arch Chir 1978;346(4): 273–81 [in German].
60. Hudson AR, Hunter GA, Waddell JP. Iatrogenic femoral nerve injuries. Can J Surg 1979;22(1):62–6.
61. Schumm F, Stohr M, Bauer HL, et al. [Peripheral nerve injury due to total replacement of the hip-joint (author's transl)]. Z Orthop Ihre Grenzgeb 1975; 113(6):1065–9 [in German].
62. Kahraman S, Sirin S, Erdogan E, et al. Is dysphonia permanent or temporary after anterior cervical approach? Eur Spine J 2007;16(12):2092–5.
63. Jung A, Schramm J, Lehnerdt K, et al. Recurrent laryngeal nerve palsy during anterior cervical spine surgery: a prospective study. J Neurosurg Spine 2005;2(2):123–7.
64. Brann CR, Brickley MR, Shepherd JP. Factors influencing nerve damage during lower third molar surgery. Br Dent J 1999;186(10):514–6.
65. Handschel J, Figgener L, Joos U. [Forensic evaluation of injuries to nerves and jaw bone after wisdom tooth extraction from the viewpoint of current jurisprudence]. Mund Kiefer Gesichtschir 2001; 5(1):44–8 [in German].
66. Cornelius CP, Roser M, Ehrenfeld M. [Microneural reconstruction after iatrogenic lesions of the lingual nerve and the inferior alveolar nerve. Critical evaluation]. Mund Kiefer Gesichtschir 1997;1(4):213–23 [in German].
67. Kehoe NJ, Reid RP, Semple JC. Solitary benign peripheral-nerve tumours. Review of 32 years' experience. J Bone Joint Surg Br 1995;77(3): 497–500.
68. Kretschmer T, Antoniadis G, Heinen C, et al. Nerve sheath tumor surgery: case-guided discussion of ambiguous findings, appropriateness of removal, repeated surgery, and nerve repairs. Neurosurg Focus 2007;22(6):E19.
69. Pandian JD, Bose S, Daniel V, et al. Nerve injuries following intramuscular injections: a clinical and neurophysiological study from Northwest India. J Peripher Nerv Syst 2006;11(2):165–71.
70. Kline DG, Kim D, Midha R, et al. Management and results of sciatic nerve injuries: a 24-year experience. J Neurosurg 1998;89(1):13–23.
71. Villarejo FJ, Pascual AM. Injection injury of the sciatic nerve (370 cases). Childs Nerv Syst 1993;9(4): 229–32.
72. Fatunde OJ, Familusi JB. Injection-induced sciatic nerve injury in Nigerian children. Cent Afr J Med 2001;47(2):35–8.
73. Ohaegbulam SC. Peripheral nerve injuries from intramuscular injection of drugs. West Afr J Pharmacol Drug Res 1976;3(2):161–7.
74. Gentili F, Hudson A, Kline DG, et al. Peripheral nerve injection injury: an experimental study. Neurosurgery 1979;4(3):244–53.
75. Gentili F, Hudson AR, Kline D, et al. Early changes following injection injury of peripheral nerves. Can J Surg 1980;23(2):177–82.
76. Midha R, Guha A, Gentili F. Peripheral nerve injection injury. In: Omer G, Spinner M, Beek AV, editors. Management of peripheral nerve problems. 2nd edition. Philadelphia: WB Saunders; 1999. p. 406–13.
77. Kline D, Hudson A, Spinner R, et al. Kline & Hudson's nerve injuries. Operative results for major nerve injuries, entrapments, and tumors. 2nd edition. Philadelphia: Saunders-Elsevier; 2007.
78. Chan TY. Prolongation of prothrombin time with the use of indomethacin and warfarin. Br J Clin Pract 1997;51(3):177–8.
79. Cosma Rochat M, Waeber G, Lamy O, et al. [Oral anticoagulation and the risk of major bleeding]. Rev Med Suisse 2007;3(131):2461–2.
80. Landi A, Saracino A, Pinelli M, et al. Tourniquet paralysis in microsurgery. Ann Acad Med Singapore 1995;24(Suppl 4):89–93.
81. Levy O, David Y, Heim M, et al. Minimal tourniquet pressure to maintain arterial closure in upper limb surgery. J Hand Surg [Br] 1993;18(2):204–6.

82. Klenerman L, Biswas M, Hulands GH, et al. Systemic and local effects of the application of a tourniquet. J Bone Joint Surg Br 1980;62(3):385–8.
83. Klenerman L. Tourniquet paralysis. J Bone Joint Surg Br 1983;65(4):374–5.
84. Trojaborg W. Prolonged conduction block with axonal degeneration. An electrophysiological study. J Neurol Neurosurg Psychiatry 1977;40(1):50–7.
85. Winfree CJ, Kline DG. Intraoperative positioning nerve injuries. Surg Neurol 2005;63(1):5–18 [discussion: 18].
86. Alvine FG, Schurrer ME. Postoperative ulnar-nerve palsy. Are there predisposing factors? J Bone Joint Surg Am 1987;69(2):255–9.
87. Sawyer RJ, Richmond MN, Hickey JD, et al. Peripheral nerve injuries associated with anaesthesia. Anaesthesia 2000;55(10):980–91.
88. ASA. Practice advisory for the prevention of perioperative peripheral neuropathies: a report by the American Society of Anesthesiologists Task Force on Prevention of Perioperative Peripheral Neuropathies. Anesthesiology 2000;92(4):1168–82.
89. Rietman JS, Dijkstra PU, Hoekstra HJ, et al. Late morbidity after treatment of breast cancer in relation to daily activities and quality of life: a systematic review. Eur J Surg Oncol 2003;29(3):229–38.
90. Roth G, Magistris MR, Le Fort D, et al. [Post-radiation brachial plexopathy. Persistent conduction block. Myokymic discharges and cramps]. Rev Neurol (Paris) 1988;144(3):173–80 [in French].
91. Spillane J. Localized neuritis of the shoulder girdle: a report of 46 cases in the MEF. Lancet 1943;ii: 532–5.
92. Turner J. Neuralgic amyotrophy (paralytic brachial neuritis): with special reference to prognosis. Lancet 1957;ii:209–12.
93. Parsonage M, Turner J. Neuralgic amyotrophy: the shoulder girdle syndrome. Lancet 1948;i:973–8.
94. Malamut RI, Marques W, England JD, et al. Postsurgical idiopathic brachial neuritis. Muscle Nerve 1994;17(3):320–4.
95. Roganovic Z, Pavlicevic G. Difference in recovery potential of peripheral nerves after graft repairs. Neurosurgery 2006;59(3):621–33 [discussion: 621–33].
96. Kline DG. Surgical repair of peripheral nerve injury. Muscle Nerve 1990;13(9):843–52.
97. McAllister RM, Gilbert SE, Calder JS, et al. The epidemiology and management of upper limb peripheral nerve injuries in modern practice. J Hand Surg [Br] 1996;21(1):4–13.
98. Kline DG, Hackett ER. Reappraisal of timing for exploration of civilian peripheral nerve injuries. Surgery 1975;78(1):54–65.
99. Kandenwein JA, Kretschmer T, Engelhardt M, et al. Surgical interventions for traumatic lesions of the brachial plexus: a retrospective study of 134 cases. J Neurosurg 2005;103(4):614–21.
100. Millesi H. Reappraisal of nerve repair. Surg Clin North Am 1981;61(2):321–40.
101. Rosen B, Lundborg G. Sensory re-education after nerve repair: aspects of timing. Handchir Mikrochir Plast Chir 2004;36(1):8–12.
102. Susarla SM, Kaban LB, Donoff RB, et al. Does early repair of lingual nerve injuries improve functional sensory recovery? J Oral Maxillofac Surg 2007; 65(6):1070–6.
103. Xu QG, Zochodne DW. Ischemia and failed regeneration in chronic experimental neuromas. Brain Res 2002;946(1):24–30.
104. Ma J, Novikov LN, Kellerth JO, et al. Early nerve repair after injury to the postganglionic plexus: an experimental study of sensory and motor neuronal survival in adult rats. Scand J Plast Reconstr Surg Hand Surg 2003;37(1):1–9.
105. McKay Hart A, Brannstrom T, Wiberg M, et al. Primary sensory neurons and satellite cells after peripheral axotomy in the adult rat: timecourse of cell death and elimination. Exp Brain Res 2002; 142(3):308–18.
106. West CA, Davies KA, Hart AM, et al. Volumetric magnetic resonance imaging of dorsal root ganglia for the objective quantitative assessment of neuron death after peripheral nerve injury. Exp Neurol 2007;203(1):22–33.
107. Terenghi G, Calder JS, Birch R, et al. A morphological study of Schwann cells and axonal regeneration in chronically transected human peripheral nerves. J Hand Surg [Br] 1998;23(5):583–7.
108. Ma J, Novikov LN, Wiberg M, et al. Delayed loss of spinal motoneurons after peripheral nerve injury in adult rats: a quantitative morphological study. Exp Brain Res 2001;139(2):216–23.
109. Sunderland S. Nerves and nerve injuries. 2nd edition. Edinburgh (UK): Churchill Livingstone; 1978.
110. Millesi H, Rath T, Reihsner R, et al. Microsurgical neurolysis: it's anatomical and physiological basis and classification. Microsurgery 1993;14:430–9.
111. Dhuner K. Nerve injuries following operations: surveys of cases occurring during a 6 year period. Anesthesiology 1950;11:289–93.
112. Bonney G. Iatrogenic injuries of nerves. J Bone Joint Surg Br 1986;68(1):9–13.
113. Brennan TA, Leape LL, Laird NM, et al. Incidence of adverse events and negligence in hospitalized patients. Results of the Harvard Medical Practice Study I. N Engl J Med 1991;324(6):370–6.
114. Towse A, Danzon P. Medical negligence and the NHS: an economic analysis. Health Econ 1999; 8(2):93–101.
115. Neu J, Scheppokat KD, Vinz H. [Medical risk and iatrogenic injury–"accident reports" from the North German Schlichtungsstelle (expert panel for extrajudicial claims resolution)]. Z Arztl Fortbild Qualitatssich 2004;98(7):567–74.