Lower Extremity Injection Nerve Injury: Black, White, and Shades of Gray

Viraat Harsh[1] James M. Barry[2] Ali Jalali[3] Anil Kumar[1] Shashikant Patil[4]

[1]Department of Neurosurgery, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India
[2]Department of Neurosurgery, LSU Health Sciences Center, Shreveport, Louisiana, United States
[3]Department of Neurosurgery, Baylor College of Medicine, Houston, Texas, United States
[4]Athens Brain and Spine Hospital, Athens, GA, United States

Address for correspondence Anil Kumar, MBBS, MS, MCh, Department of Neurosurgery, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand 834009, India (e-mail: dr.anilkumarranchi@gmail.com).

J Peripher Nerve Surg

Abstract

Injection nerve injury of the lower extremity is a fairly common but incompletely quantified occurrence in medical practice. Numerous medical procedures including intramuscular injections, joint aspirations, arterial lines, and nerve blocks place these nerves at risk of injury. Some of the nerves at risk include the sciatic, femoral, saphenous, superior gluteal, posterior femoral cutaneous, and the lateral plantar, with the sciatic being the most commonly involved. The possible mechanisms of injury include direct needle trauma, pressure injury, chemical neurotoxicity, compression neuropathy, ischemia, scar formation, and allergic response. Presentation varies widely depending on the nerve injured but can be generalized to consist of paresthesia, pain, numbness, and weakness consistent with the enervated distribution of the nerve. The diagnosis of such an injury is mostly clinical but several confirmatory tests exist to quantify the damage, the most common of which are electromyography and nerve conduction study. In the case of nonsevere injuries with minimal weakness and pain, conservative therapy with pain medication, physical therapy, and regular follow-ups is recommended for 3 to 6 months. In cases with severe functional disability and/or pain or no improvement after initial conservative measures, surgical options are pursued. These include neurolysis or, in rare cases, nerve grafting. The goal of this article is to elaborate in detail the mechanisms, pathophysiology, clinical presentation, and treatment of lower extremity injection injuries in the aforementioned nerves.

Keywords
- nerve injection injury
- sciatic nerve palsy
- intramuscular injections

Introduction

Injection- and needle-induced nerve injuries are fairly common, although their exact incidence is unknown. Nerves may be injured directly by the needle upon attempted intravenous line placement, joint aspiration, nerve blocks, phlebotomy, or intramuscular (IM) injections,[1] or, indirectly from the injected substance itself. Common nerves reported to undergo such injuries include sciatic, radial, axillary, ulnar, median, tibial, and peroneal nerves. Since the buttock is the most common site of IM injections, it is unsurprising that the sciatic nerve is the most commonly affected nerve.

The injury may be broadly attributed to misguiding of the needle and the injected material or to anatomic variations in the course of the nerve with an otherwise correctly guided needle. The patients mostly present with sharp pain and paresthesias in the sensory distribution of the nerve at the time of injection itself, but there may also be a delayed presentation. The importance of medical history pertaining to injections in the recent past cannot be stressed more in patients presenting with such symptoms. Infants may present with clubfoot and older children with leg length deformities. Unless the symptoms are debilitating, there is no agreement on the exact time of surgical intervention in these cases. Yet, most neurosurgeons advocate waiting 3 to 6 months

DOI https://doi.org/10.1055/s-0040-1701355.

for spontaneous recovery with supportive conservative management including physiotherapy and adjunctive pain management. Surgical intervention in these cases is usually neurolysis if a positive nerve action potential (NAP) is present in nerve conduction studies (NCS). Otherwise, suture or graft repair should be performed. Timely conservative management and surgical intervention, if required, can lead to remarkable recovery.

In this article, we review the literature on injection injuries of nerves of the lower extremities, the mechanism(s) of injury, pathology involved, options in management of these cases, and precautions which can prevent them.

Pathophysiology and Classification

Several mechanisms of injury have been proposed. Other than direct needle injury,[2-9] suggested mechanisms are chemical neurotoxicity[6,7,10-13] including free radical damage,[14] compression neuropathy from the substance injected,[2,6] external scar formation,[15,16] ischemia,[17-22] and allergic neuritis.[23,24] In an animal model with the help of light and electron microscopy and a fluorescent tracer, Gentili et al[20] found that pathological changes could be seen in the nerves as early as 30 minutes after injection. They proposed the damage of the blood–nerve barrier as an important cause of nerve injection injuries. Neurotoxic chemicals placed within the epineurium in the extrafascicular or intrafascicular region can disrupt blood–nerve barrier leading to degeneration of the nerve[4,22,25-27] as has been observed in animal models as well.[28] Intrafascicular injections are more likely to cause the damage for obvious reasons.[29] Injury may also occur from progressive inflammation and fibrosis leading to neuritis.[4,25-27,30,31] In such cases, the presentation is often delayed.

Sunderland[32] classified nerve injection injury into three degrees depending on the severity of the injury. First-degree injuries suffer reversible conduction blocks, and conservative management alone is sufficient. Second-degree injuries include Wallerian degeneration with reactive fibrosis. In these cases, recovery is slow and often incomplete, and neurolysis is indicated. Third-degree injuries have necrosis and fibrosis due to neurotoxicity of the agent. There is no spontaneous recovery in these cases and flushing may help early on to limit the neurotoxicity. However, typically, the drug is completely absorbed by the time of surgery.

Mechanism of Injury

Injection site and technique play an important role in nerve injection injuries.[33-36] Many cases of nerve injection injury particularly in developing countries are, thus, suspected to have accrued after injection by untrained or under-trained medical practitioners.[37-41] In many developing countries, parents of ill children have a misconception that compared with oral medications; injections will make their child heal faster. As a result, these children are more prone to receive injections by untrained individuals, which increase the chances of nerve damage.[40]

Greensmith and Murray[42] suggested that low pressure injections are less damaging to the nerves than high pressure injections (> 11 *psi*), which cause severe fascicular injury. Damage caused may be partial[43] or complete depending on the needle tip placement[44,45] and the neurotoxic agent used.[44] A large or fixed/confined nerve is injured more commonly than a smaller one or one that is relatively free and thus has the capacity to move and hence avoid the needle.[42,44] Wrong methods like restraining an agitated child or angulating the needle while injecting also constitute other risk factors.

In many instances, the peripheral neuritis and nerve damage occur even with the use of correct technique. Chemical neurotoxicity of the agent injected has been blamed in many such cases. A variety of substances including analgesics, antibiotics, antiemetics, steroids, vaccines, and vitamin injections have been reported to cause chemical neurotoxicity and have been discussed individually within the context of each nerve injury. Yaffe et al[46] performed an experiment on rats with 11 different agents and found that penicillin G, diazepam, and chlorpromazine were the most neurotoxic. Their results also showed that neurolysis performed after 24 hours alleviated further damage to the nerves, and that there was no additional benefit to flushing the nerves with saline or Celestone (steroid betamethasone). Among steroids, dexamethasone has been observed to be less toxic than hydrocortisone and triamcinolone that can cause significant nerve damage. Steroids cause direct toxicity on peripheral nerve fibers and cause ischemic changes to the nerve.[34] Penicillin has been shown to cause granuloma formation when administered into sciatic nerves in dogs.[47] In a study by Senes et al,[18] the authors noticed that hydroxybenzoate, alcohol, and diacetin are most neurotoxic, sodium cefuroxime and phenobarbital are moderately neurotoxic, while other drugs in the group such as naloxone (with hydroxybenzoate), vitamin K (with glycocholic acid and lecithin), and glycocholic acid and lecithin when used individually are comparatively least neurotoxic, but nevertheless can still cause nerve injury. Gentili et al[45] noticed a moderate degree of rat sciatic nerve damage by injections of chloramphenicol, gentamycin, imferon, and cephalothin with more damage in the large fibers and relative sparing of the smaller ones. Also, extrafascicular nerves were damaged when benzyl penicillin, diazepam, and chlorpromazine were used as agents. The authors proposed that extrafascicular damage was related to intraneural pathology and was not due to external scar compression.

Anatomic variations may be another reason for injection injury. Examples include absence of or abnormal subdivision of piriformis muscle, peroneal nerve passing through the piriformis muscle and proximal margin of superior gemellus, and tibial nerve rounding the inferior margin of superior gemellus and over the internal obturator muscle. These variations may lead to injection injury even if the technique used is correct.[18]

Presentation and Diagnosis

Most patients present with sharp stabbing pain at the time of injection,[7,48] and with neurosensory loss along the peripheral nerve distribution. Some patients report a delayed presentation hours or days after injection.[38,49,50] Any presentation of pain along a nerve distribution and/or neurosensory loss with a history of injection use in the recent past should

raise suspicion of nerve injection injury. Motor function of the muscles supplied by the respective nerve may also be compromised. Electromyography (EMG), compound muscle action potential (CMAP), nerve conduction velocity, sensory nerve action potential (SNAP), amplitude measurement, magnetic resonance imaging (MRI), and magnetic resonance (MR) neurography can be used to investigate or confirm the diagnosis.[50] On EMG, the initial change would be a reduction in motor unit recruitment. Assuming Wallerian degeneration occurs, high insertional activity, positive spontaneous fibrillation, and sharp waves will develop in 1 to 4 weeks in the muscles being studied in acute denervation, while chronic denervation with reinnervation presents with polyphasia, high amplitude, lowered interference pattern, and recruitment in the muscles being studied.[50-52] MR neurography shows increased signal intensity on T2-weighted images of injured nerves and corresponding muscles 24 hours after injury[53-56] and can also help in diagnosis of postinjury neuroma formation typically several months later.[57] Injection injury should be considered as an important differential diagnosis in all patients presenting with neurogenic pain syndromes and other sensory or motor deficits.

Management

Once the diagnosis is confirmed, if the injury is not severely incapacitating and if the pain is only minimal, the attending physician should wait for at least 3 to 6 months before surgical exploration along with NAP recording.[7,18,58,59] During this period, conservative management for pain with physiotherapy to maintain muscle tone has shown to have beneficial effects. If, however, no significant evidence of recovery is seen electrophysiologically or clinically after 3 to 6 months of conservative management, surgical intervention is considered. Treatment is individualized depending on the patient history, presentation, clinical examination, and electrophysiological reports. If NAP is present beyond the lesion, external neurolysis alone or in combination with internal neurolysis is performed. Negative NAP beyond the lesion warrants suture or graft repair. The use of real-time ultrasonography in the preoperative phase and during surgery helps in detection of the type of injury, position of stumps, neuroma formation, and excessive scar formation.[60]

Synthetic graft repairs with collagen bioabsorbable nerve conduit,[61,62] proliferated Schwann cells and homologous dermal acellular matrix,[63] keratin-based hydrogel filler,[64] chitosan gel sponges containing bone marrow stromal cells-derived Schwann cells,[65] and poly-DL-lactide-ε-caprolactone conduits[66] have shown promising outcomes in animal models. Rosson et al[67] repaired short-gap motor nerve injuries with bioabsorbable polyglycolic acid nerve conduits and reported good results in all six patients. Such conduits can be used for small diameter nerves with gaps of less than 30 mm. Moore et al[68] reported four patients with injury of large-diameter nerves who underwent nerve repair with absorbable nerve conduits; these patients failed to achieve a good clinical outcome. Vascular endothelial growth factor (VEGF) has been described by some, to have not only angiogenic but also neurogenic properties and Fu et al[69] described the beneficial effects of using local phVEGF$_{165}$ to promote regeneration of nerves and acceleration of recovery in injured sciatic nerve of rats.

Sciatic Nerve

Sciatic nerve is the most commonly involved nerve in injection injuries.[13,15,18,38,70-77] As early as in 1882, the first account of injection injury of the sciatic nerve was described as "sciatic neuritis due to injection."[78] Also, injection injury is the second most common cause of iatrogenic sciatic neuropathy only after hip arthroplasty.[72] Seemingly, injection injuries of the sciatic nerve have been recognized almost since the time IM injections were developed and a significant number go unnoticed and unreported owing to its frequent incidence.[79] Kline et al[33] in their study of 230 patients suffering from injection and needle injuries to the nerve reported sciatic nerve injury in ~ 60% of the patients. Villarejo et al[15] reported 370 cases of sciatic nerve injection injury alone. About 50% of them were infants. The authors suggest that higher incidence of injection injury to the sciatic nerve in children can be attributed to less gluteal covering and lower gluteal mass volume to sciatic nerve size ratio.[36]

Direct Injury

Sciatic nerve in children is in the middle of the gluteal region and needle inserted perpendicularly in that region has a relatively high likelihood of injuring the nerve.[80] Also, in infants, gluteal area has a high fat content and any nonlipophilic chemical will tend to be absorbed much less slowly and pressure effects from the chemical might damage the nerve.[81] Thin gluteal covering also makes elderly, underweight patients and those with wasted gluteal muscles prone to sciatic nerve injection injury.[33,82,83] Although thin and underweight patients are more at risk for sciatic nerve injury from injections, Ross Kerr and Wood[84] suggest that if the patient is obese or unable to turn well to expose the entire buttock, locating landmarks can be difficult and the precision of any single avoidance technique may suffer. Other inappropriate practices such as incorrect technique, wrong site of injection, incorrect length of needle or angle of the needle with respect to the gluteal plane, and improper positioning of the patient during injection are prominent reasons for these injuries. Injections outside the upper outer quadrant of the dorsogluteal region, especially medial and inferior quadrant of the buttock, can cause sciatic nerve injury.[33,85] Abnormal postures, like flexed hips and extended legs, stretch the nerve and take away its ability to yield when the needle meets it.[15] Hirasawa et al[86] studied the effect of different types of needles on the degree of injury to sciatic nerve during nerve block injections in rabbits. Their study showed that tapered injection needles did not cause any damage or tearing of the nerve fibers and caused the least damage to the perineurium, while short- and long-beveled needles cause reduced damage when face of the bevel is inserted parallel to the fibers.

Ndiaye et al[12] performed sciatic nerve gluteal dissection on both sides in 10 adult African cadavers. All 20 nerves but 1 was in the subpiriformis canal. For all 20, pathway was

identical with oblique and vertical part running down the ischiotrochanteric channel. They thus proposed that the projection of sciatic nerve is far from the skin if the injection is given in the upper outer quadrant of the buttock and due to the paucity of anatomic anomalies they concluded that it must be the nature of the agent (quinine given for malaria treatment in this case) causing neurotoxicity rather than physical trauma, if injections are given at the correct site.

Chemical Injury

Sciatic nerve injury after quinine injection was reported as early as 1920 by Turner[87] and since then has been reported several times by others.[87-89] Other antimalarials,[37,41] ketorolac,[27] diclofenac,[38] and other analgesics,[37,50,76] local anesthetics, antiemetics (demerol with vistaril or phenergan),[33] penicillin, ampicillin, tetracycline, paraldehyde, optalgin,[71] cephalosporin[18] and other antibiotics,[37,76] phenobarbital, steroid, vaccines, and vitamin K injections[18,33] have also been reported to cause neurotoxicity. Hanson[90] suggested that the degree of injury depends on the vehicle of suspension. Antiemetics with promethazine and dimenhydrinate caused more neuronal damage than prochlorperazine. Whitlock et al[29] showed that ropivacaine when given intraneurally in sciatic nerve in rats caused severe demyelination and Wallerian degeneration. Local anesthetics used for total hip replacement can also cause sciatic nerve injury. Uppal et al[91] report that while operating for sciatic nerve injury after a total hip replacement, local anesthetic was seen leaking from the nerve sheath. Lidocaine, procaine, and tetracaine split the myelin lamellae and cause more severe damage than bupivacaine.[29]

Pathophysiology

In a series of seven patients, Kolb and Gray proposed that necrosis was the main pathologic mechanism involved in injury from penicillin injections.[23] A diverse picture of pathological changes was reported by Gilles and Matson[59] in 1970. On resecting the peroneal and tibial branches of the sciatic nerve, they noticed cicatrix formation, multinucleate giant cells, and abnormal circumferential collateral sprouting of axons. They also noted that there was fibrosis and collagen deposition in some patients. The number of distal axons was low and macrophages were noted throughout. Central axons were damaged more than the peripheral ones. Grossly, an in-continuity neuroma with a yellowish discoloration and hourglass deformity or with bulky appearance may be seen as injury causes nerve fiber thickening.[18]

Presentation and Clinical Diagnosis

Patients usually present with causalgia and paresthesia, symptoms that are immediate in 90% of the injury cases,[1,15,33,38,48-50,92] but paralysis of the muscles supplied by the sciatic nerve may also be seen. Obach et al[21] described four different types of paralyses: (quoting verbatim) "1- Immediate neurogenic involvement with instant pain, 2-Most frequently obvious paralysis, without pain, 3- Subacute or late paralysis, without immediate pain, 4-Immediate pain and late paralysis." If the patient complains of numbness/paresthesia/pain and other symptoms of nerve injection injury at the time the injection is given, immediate flooding of subgluteal space with 50 to 100 mL of physiologic solution may dilute the agent and prevent the injury.[59] Generally there is more severe impairment of the motor functions than neurosensory loss.[11] This view is supported by Clark et al,[10] who also noted that larger fibers were damaged earlier and more severely than smaller ones after injection injury in cats. The artery supplying the sciatic nerve between piriform foramen and half-way down the thigh is the inferior gluteal artery, involvement of which in the injury might explain the perineural lesions of the sciatic nerve presenting down to the thigh.[21] Complete sciatic nerve lesion proximal to hamstring leads to impaired knee flexion, loss of dorsi- and plantar-flexion of foot, loss of functions of posterior tibialis, foot invertors and peroneus longus and brevis foot evertors, and sensory loss to posterior thigh, lower lateral leg, and complete foot, which may lead to pressure sores, infections, claw toes and may later lead to a limb amputation.[82] However, this type of complete sciatic nerve injury is extremely rare with injections.

A majority of weakness presentations is related to peroneal division lesions,[15,18,33,38,50,59] more so because it is more laterally located than the tibial division[15,82] and has poor support of the surrounding connective tissue.[76] The other reason for more frequent involvement of the peroneal nerve is that the tibial division is relatively fixed only at the sciatic notch, whereas the peroneal nerve is relatively fixed both at the sciatic notch and around the neck of the fibula. Patients present with paralytic foot drop[13,40,41,50] and paralysis of hamstrings and all muscles below the knee causing difficulty in walking.[76]

For obvious reasons pain and neurosensory loss may be difficult to evaluate in neonates and infants; hence, the diagnosis in them is mainly limited to the motor loss.[18] Since common peroneal nerve is more commonly involved, foot drop is a more common presentation.[43] A foot drop in an infant may be wrongly diagnosed as a congenital clubfoot; hence, it is critical to evaluate all infants presenting with clubfoot for nerve injury.[18] Napiontek and Ruszkowski[93] observed that children who present with equinus or equinovarus deformity after nerve injection injury were diagnosed on an average much earlier (3.8 months) than those presenting with foot drop (5.1 years). Also, children presenting with foot drop later had a higher predisposition to develop gluteal fibrosis at a later stage. Cavovarus and calcaneovarus foot deformities have also been reported.[94] If neglected for long, palsy of the sciatic nerve in children may lead to decreased growth of the foot on the involved side, over time presenting with leg-length deformities.[11,16,18,95,96]

Electrophysiologic Investigations

Electrophysiologic studies with needle EMG study in tibialis anterior, medial head of gastrocnemius, and short head of biceps femoris[50] may help establish the diagnosis. Loss of SNAP of sural and superficial peroneal nerves[38] may be observed. No response on EMG suggests axonal loss. If peroneal division is more damaged than the tibial, correlative EMG results will be seen, such as no or minimal response

on EMG from the peroneal division and minimal or moderate from the tibial division.[91] Diffusion tensor tractography (DTT), a type of MRI, has also been used to monitor the state of regenerating neurons. Fractional anisotropy value, a parameter in the DTT decreases after nerve injury and has been used to correlate with the total number of regenerating axons and may have prognostic importance in predicting functional improvement.[97,98]

Maqbool et al[38] used motor NCS on common peroneal and posterior tibial nerves with electrodes placed on extensor digitorum brevis (EDB) and abductor hallucis longus muscles. Sensory NCS of sural and superficial peroneal nerves, EMG studies on EDB, tibialis ant, medial head of gastrocnemius, and short head of biceps femoris were performed on 106 injury cases. They used the Medical Research Council grading score from 0 to 5 to grade the motor function. They assessed the relative involvement of common peroneal and posterior tibial nerves by comparing the muscle strength in dorsiflexors versus the plantar flexors and CMAP in EDB, tibialis anterior, and gastrocnemius in the first electrophysiological study. Based on the CMAP amplitude, 50 out of 106 patients had prominent common peroneal nerve involvement, 19 had posterior tibial nerve involvement, and 28 had equal involvement of both. Eighty-eight patients had abnormal F-wave. Active denervation was revealed by needle EMG in 25 patients, while chronic reinnervation in 41 patients. Electrophysiological recovery was noticed in 27% cases, which also achieved clinical recovery.

Management

Many neurosurgeons believe that neurolysis should be performed as soon as possible after injury.[99] If performed within 24 hours of the injection injury, neurolysis may prevent paralysis[13] while delay may cause fibrosis.[18] Topuz et al[100] propose that if nerve transection is suspected, surgical exploration should be performed without waiting for electrophysiological substantiation. Delay in diagnosis confirmation leads to poor prognosis postoperatively.

Conservative

If there is no functional motor incapacitation or sensory loss and pain is the only prominent complaint, conservative analgesic therapy with physiotherapy is recommended to prevent atonia.[15] Keskinbora and Aydinli[43] proposed the use of gabapentin for the treatment of neuropathic pain caused by sciatic nerve injection injury in an 8-year-old boy. They used gabapentin starting at 300 mg thrice a day introduced over 3 days to avoid overdose or adverse effects. After 3 months, as pain was controlled, patient was asked to continue on a maintenance dose of 100 mg thrice a day for allodynia. At 8 months follow-up, patient did not have allodynia, and gabapentin was stopped.

Chen et al[101] and Unezaki et al[102] have reported the importance of nerve growth factor (NGF) in the regeneration of nerves in rats. NGF increases the density of unmyelinated axons and along with glial cell line-derived neurotrophic factor promotes nerve recovery in rats significantly.[102] Sulaiman and Gordon[103] studied the effect of tacrolimus (FK506) and transforming growth factor-β (TGF-β) in a rat model. FK506 has been found to increase number of regenerated neurons and axons. TGF-β reactivated Schwann cells and increased their capacity to support axonal regeneration. Wilson et al[104] also reported acetyl-L-carnitine to considerably improve nerve regeneration.

When being managed conservatively, follow-up for monitoring the nerve regeneration is important. T2-weighted images that show hyperintense signals for an injured nerve return to normal. This has been reported to correlate with nerve regeneration and its clinical manifestations.[53-55] Behr et al[105] suggest that in the first 3 weeks postinjury, persistent T2 signals indicate a more serious lesion, which can be compared with neurotmesis, whereas decrease in signals before 4 weeks postinjury may indicate regeneration. Clark et al[10] suggested that surgery should be contemplated 6 months after the injury, but Villarejo and Pascual[15] obtained excellent results when the patients were operated on between 3 and 6 months postinjury.

Surgery

Indications for surgery include but are not limited to incapacitating or complete deficit of tibial or peroneal distributions with no sign of recovery clinically or on EMG over 3 to 6 months,[15,33] or formation of a palpable granuloma.[59] If the intraoperative NAP is positive, good prognosis has been reported with neurolysis alone. Furthermore, the peroneal division has typically a worse prognosis than tibial, except in children.[7,33] Surgery (both neurolysis and nerve graft repair) has better prognosis when performed on the tibial division of the nerve than on the peroneal division.[82,92] The tibial nerve has more extended blood supply than the peroneal, which may be a reason for better regeneration of the tibial division. Internal neurolysis should be performed carefully to save the small fibers that might recover spontaneously if no intervention is done.[106]

Negative intraoperative NAP leaves the option of suture or graft repair. On the operation table, it may be tough to define the extent of injury in the nerve. It is suggested that the damaged segment of the nerve is opaque to transmitted light, and hence transillumination test may be used to determine the exact limit of resection.[59] According to Sunderland,[32] nerve grafting results of proximal portion of sciatic nerve are poor and hence neurolysis alone may suffice. In children with incomplete recovery results, dynamic elastic splint may be initially applied and later at the age of 3 years, musculotendinous transposition of posterior tibial tendon on the insertion of anterior tibial tendon may be done.[18]

Senes et al[18] reported in their series that the surgical outcome did not depend on the nature of injected agent; those children who were injected with antibiotics (cephalosporin) had worse outcome as compared with others, although a definite relationship of the outcome with the agent has not been established. Prognosis depends on the division involved, level of injury, age of the lesion, timing of repair, and patient's overall medical status.[92] Presence of CMAP in the EDB within 6 months and early functional recovery are positive prognostic factors.[10] Bay[85] suggests that sciatic nerve

injection injury is almost always fully reversible, although the course of recovery may be biased by psychological factors as compensation claims, negligence suit and in such cases only pain is the main symptom.

Injecting the Gluteus

In the superolateral quadrant injections method, some authors consider that the injection is being given in the gluteus medius[107] and some think that gluteus maximus is the aim.[108] Several authors[12,35,37,38] suggest total avoidance of IM injections if other routes can be used; if not, avoiding the gluteal region especially in children < 5 years of age is best, so as to avoid sciatic nerve injury by injections. Some physicians advise that if the injection site is above and lateral to the midpoint of the imaginary superolateral line drawn joining posterior superior iliac spine and the greater trochanter, the nerve can be escaped.[81,109,110] Ventrogluteal/anterolateral site for injection has been suggested by many to possibly spare the sciatic nerve.[17,76,77,81] This is especially important for hydrophilic chemicals as subcutaneous fat is thinner in the anterolateral region and muscle thickness is greater and these chemicals will therefore be absorbed well.

Mishra and Stringer[76] suggest that even in the ventrogluteal region, the injection should be given in the gluteal triangle, which can be formed by the palm of opposing hand placed on the greater trochanter and pointing finger on the anterior superior iliac spine, and the middle finger pointing toward the iliac crest. The position of the middle finger should be as separated from the index finger as possible.[111-113] The needle should then be inserted at an angle of 90 degrees to the skin in this triangle. Injections can be given in the ventrogluteal site in supine, prone, and side lying position without the fear of damaging the sciatic nerve.[114] A limited number of studies report the successful use of this technique.[112,113,115] Müller-Vahl[116] in 1983 reported a case of ventrogluteal site injection complication in which the patient suffered from paralysis of the tensor fascia latae muscles post injection. There is no consensus regarding the safe volume of substance that can be injected in the gluteal mass, although it has been suggested that the volume of drug injected might play a role in these injuries.[45] Some authors suggest that not more than 3 mL should be injected into the gluteal mass,[84] although some authors suggest that up to 4 mL[110] and yet other group say 5 mL volume injection is safe.[117]

Femoral Nerve Injection Injury

The femoral nerve arises from the posterior divisions of the ventral rami of L2–L4. It runs through the psoas emerging at its lateral border and giving off several small branches to the iliacus within the abdomen. The nerve then enters the leg lateral to the femoral sheath and is divided by the lateral circumflex artery, forming the anterior and posterior divisions typically 1 to 4 cm distal to the inguinal ligament.[118] The anterior division supplies motor branches to the sartorius and pectineus and two sensory branches that are the intermediate and medial cutaneous nerves of the thigh. The posterior division gives off motor branches to each division of the quadriceps femoris. The branch to the rectus femoris gives rise to the articular branch of the hip joint. The knee joint receives small branches from the vastus lateralis, vastus medialis, and vastus intermedius. Additionally, branches from vastus intermedius supply the articularis.[119,120] Through these muscular branches, the femoral nerve primarily helps in flexion of the hip, extension of the leg at the knee, and in transmitting sensation from the anterior and medial thigh.[121]

The femoral nerve is particularly prone to injury as IM injections in the anterolateral thigh into the vastus lateralis are recommended in infants up to the age of 7 months.[22,122] They are given at one hand's width below the greater trochanter and at one hand's width above the knee, that is, along the middle third of the lateral thigh.[115] Pain at the time of presentation may have an aching, cramping, tingling, or burning character with distal radiation to the knee. Motor dysfunction may accompany. Over the long term, there might be anterior thigh muscular atrophy, difficulty in hip flexion and leg extension, and dysesthesia or anesthesia in the anteromedial thigh and medial leg.

Haber et al[22] reported two such cases after meperidine injections. In both cases, patients received multiple lateral thigh injections for pain control following surgery. Each of the patients recalled an episode of severe pain following one specific injection. Both complained of continued pain at the injection site followed by atrophy of the lateral thigh despite physical rehabilitation. One patient noticed a bulge just proximal to the flare of the left femoral metaphysis 2 months after the injection. At 6 months after the injection, the fluctuant bulge was aspirated and a sanguineous vicious fluid was obtained, which did not show signs of inflammation or infection. One patient lacked sensory deficit, while the other experienced hyperesthesia in a nonfemoral nerve distribution. In each case, MRIs showed increased T2 signal of the vastus lateralis consistent with inflammation secondary to muscle degeneration. Both patients had EMGs consistent with isolated denervation of the vastus lateralis at the time of presentation that returned to normal after several months of follow-up. In each case, conservative management was pursued and at 5 to 6 months follow-up muscle atrophy remained but pain resolved and sensation returned to normal.

Saphenous Nerve Injection Injury

The saphenous nerve is a large sensory cutaneous branch arising from the femoral nerve. It runs with the femoral artery in the adductor canal beneath the sartorius and descends along the medial side of the knee where it pierces the fascia lata between the sartorius and gracilis. The nerve continues down the leg just behind the medial border of the tibia. At the bottom third of the tibia, the nerve divides into two branches, the first of which ends at the medial side of the ankle and the second wraps around the anterior ankle.[119] It provides sensation to the medial aspect of the leg starting at the knee and continuing to the medial maleolus.[121]

The saphenous nerve is most vulnerable during knee injections for osteoarthritis. Unfortunately, there is little

consensus on which approach works best, leaving it up to the practitioner to select from four variations in the lateral or medial approach.[123,124] Notably, anatomy of the saphenous nerve has been described as fairly uniform across cadaveric specimens with a course generally between the sartorius and gracilis.[125] Thus, to avoid saphenous nerve injury, a lateral approach when injecting for knee disorders is advocated especially in obese patients with unclear surface anatomy.[124,126]

Iizuka et al[126] reported a case of saphenous nerve injury after hyaluronic acid injection into the knee via the medial approach for osteoarthritis. The patient was described to have developed saphenous nerve distribution numbness 1 hour after the injection. Examination 3 weeks later showed that the patient had decreased sensation of the infrapatellar area and in the midcalf, with normal strength and reflexes. Conservative therapy was pursued and at 8 months follow-up the patient began experiencing allodynia in the same distribution that could be elicited by percussion of the nerve.

Superior Gluteal Nerve Injection Injury

The superior gluteal nerve (SGN) arises from the posterior divisions of L4-S1 leaving the pelvis through the greater sciatic foramen above the piriformis. It travels with the superior gluteal artery and vein, ending in the gluteus minimus and tensor fasciae latae.[119] This nerve innervates the gluteus medius, gluteus minimus, and the tensor fascia lata, and hence its injury causes difficulty in abduction and medial rotation of the hip, classically resulting in Trendelenburg's gait.[81] In addition, damage to this nerve can cause contracture of the gluteus medius.[127] In some cases, the SGN has been damaged despite the use of correct injection technique.[128]

Obach et al[21] reported two cases of isolated SGN injury. In one, a woman who received intragluteal injection of triamcinolone was referred by her primary care physician for a swaying gait. By her appointment time, 3 months later, her gait had improved but exam showed mild weakness of the gluteus medius and SGN damage on EMG. In the second case, a child who had received gluteal injection 1 year prior presented with claudication of the lower left limb with severe atrophy of the gluteus medius and minimus. EMG showed neurogenic changes of the SGN. No follow-up was mentioned for either of the above patients.

Posterior Femoral Cutaneous Nerve Injection Injury

The posterior femoral cutaneous nerve arises from the dorsal divisions of S1–S2 and the ventral divisions of S3–S4 and exits the pelvis through the greater sciatic foramen below the piriformis. It travels with the inferior gluteal artery running beneath the gluteus maximus and traveling down under the fascia lata. It passes over the biceps femoris piercing the deep fascia and running with the small saphenous vein to the lower portion of the popliteal fossa.[119] Due to its proximity to the sciatic nerve, isolated posterior cutaneous femoral nerve (PFCN) intragluteal injection injuries are very rare making up only 1% of reported cases.[19] This nerve primarily functions providing sensation to the posterior thigh and popliteal fossa.[121] Pain, anesthesia on back of thigh, gluteal area, and perineum have previously been described with injury of this nerve.[50,79]

Tong and Haig[129] reported an incidence of a 25-year-old woman who received two right buttock injections after hospitalization for chronic headaches. While she was unsure of the medication given, she endorsed numbness in the right posterolateral thigh and buttock starting at the time of discharge and continuing until presentation 5 months later. Aside from numbness, her physical examination was unremarkable at that time. NCS were consistent with isolated right PFCN neuropathy. In addition, Kim et al[130] described a remarkably similar case of a 22-year-old woman who presented with numbness and paresthesia in the left lower gluteus and posterior thigh 3 weeks after receiving IM antibiotics for a respiratory infection. She described "stinging" pain down her posterior thigh at the time of injection. NCS of the PFCN was consistent with isolated neuropathy. Her symptoms failed to resolve after 6 weeks of conservative management. Interestingly both of these patients showed numbness in the inferior gluteal region. This symptom points to additional damage to the inferior medial cluneal nerve, a branch of the PFCN that arises just after it leaves the sciatic foramen.[129]

In both of the cases above, authors discuss the use of NCS technique described by Dumitru and Nelson.[131] This technique allows the use of the midline of the thigh and the mid-popliteal region as consistent landmarks to place stimulating electrodes for PFCN evaluation. In their study of 40 healthy subjects (20 men and 20 women), all patients had strong responses in the PFCN bilaterally making this the gold standard technique for PFCN testing.

Lateral Plantar Nerve Injection Injury

The lateral plantar nerve runs along the lateral side of the foot, medial to the lateral plantar artery. It lies between the flexor digitorum brevis and the quadrates plantae and divides into superficial and deep branches at the abductor digiti minimi[119]. This nerve supplies sensation to the skin of the lateral portion of the fourth toe and the entire fifth toe.[121]

Snow et al[132] described the case of a 41-year-old man who developed numbness in the left third, fourth, and fifth toes following a 40 mg Depo-medrone/lignocaine intraneural injection given by the medial approach for plantar fasciitis. This injection is most commonly given through the thin skin over the medial calcaneal tuberosity heading anterolaterally toward the plantar fascia. At 4 years follow-up the patient had both numbness in the lateral foot and weakness of toe flexion with a confirmatory absence of nerve conduction in the lateral plantar nerve. The authors did not report management prior to their encounter with the patient or further follow-up. Notably, they concluded that the risk of lateral plantar nerve injury could be minimized by proper understanding of the anatomy of this nerve.

Conclusion

Injection injury of peripheral nerves, especially the sciatic nerve, is a persistent global problem. With addition of newer injectable drugs in the last few decades, a higher number of cases are being seen and an even higher number are going unreported by medical practitioners. This problem is more severe in developing countries where administration of injections is left to poorly trained individuals.[37,40] More worrisome and difficult to overcome are uninformed patient's strong beliefs that IM injections are more efficacious than other routes of administration. World Health Organization estimated that annually ~ 12 billion injections were administered globally, out of which 50% were unsafe and 75% unnecessary.[133] In several instances, the courts have ruled that IM injections can cause sciatic nerve injection injury.[134] Mishra and Stringer[76] compiled and published medicolegal reports of sciatic nerve injury (1989–4/2009) and the total compensation is surprisingly more than $4,097,449.

As can be seen, sciatic nerve injection injury is a complication which can be easily minimized by taking appropriate precautions. Physicians should talk to patients about the risks of injections and make every attempt to recommend and/or use other mechanisms of drug delivery especially when they are of equal efficacy. Where possible, injections should be avoided, especially in the case of children whose anatomy makes them more prone to injury. When injections are given, a detailed knowledge of anatomic relationships is critical as the vast majority of nerve injuries occur in the setting of normal anatomy.

Finally and most critically, the signs of nerve injury should not go unheeded. If a patient complains of pain/paresthesia during injection, immediate flushing of the area with 50 mL saline should be done and patient should be followed up till recovery is assured. If the condition does not improve within a reasonable timeframe, consultation with pain management and neurological team should be made. When required, neurosurgical consultation should be scheduled at the earliest opportunity, especially if a profound paralysis is noted clinically. Appropriate timely surgical intervention may help in decreasing the morbidity of these patients and some people might recover fully.

References

1 Preston D, Logigian E. Iatrogenic needle-induced peroneal neuropathy in the foot. Ann Intern Med 1988;109(11):921–922
2 Kasten SJ, Louis DS. Carpal tunnel syndrome: a case of median nerve injection injury and a safe and effective method for injecting the carpal tunnel. J Fam Pract 1996;43(1):79–82
3 Allensworth M, Scheinberg L. Sciatic neuropathy in infants related to antibiotic injections. Pediatrics 1957;19(2):261–265
4 Bhatia M, Jindal AK. Injection induced nerve injury: an iatrogenic tragedy. J Assoc Physicians India 1996;44(8):532–533
5 Sawaizumi T, Sakamoto A, Ito H. Injury of superficial radial nerve on the wrist joint induced by intravenous injection. J Nippon Med Sch 2003;70(4):355–359
6 Stahl S, Kaufman T. Ulnar nerve injury at the elbow after steroid injection for medial epicondylitis. J Hand Surg Br 1997;22(1):69–70
7 Spinner RJ, Kline DG. Surgery for peripheral nerve and brachial plexus injuries or other nerve lesions. Muscle Nerve 2000;23(5):680–695
8 Barry JM, Harsh V, Patil S. Are our intramuscular injections nerve-friendly? What are we missing? Simple techniques to prevent, recognize and manage nerve injection injuries. Int J Students' Res 2014;4(2):25–28
9 Barry JM, Harsh V, Kumar A, Patil S. Injection nerve palsy: what's to blame? J Neurosci Rural Pract 2013;4(4):481
10 Clark K, Williams PE Jr, Willis W, McGavran WL III. Injection injury of the sciatic nerve. Clin Neurosurg 1970;17:111–125
11 Gilles FH, French JH. Postinjection sciatic nerve palsies in infants and children. J Pediatr 1961;58:195–204
12 Ndiaye A, Sakho Y, Fall F, Dia A, Sow ML. [Sciatic nerve in gluteal portion: application of sciatic nerve post injection lesion] [in French]. Morphologie 2004;88(282):135–138
13 Mayer M, Romain O. [Sciatic paralysis after a buttock intramuscular injection in children: an ongoing risk factor] [in French]. Arch Pediatr 2001;8(3):321–323
14 Saray A, Apan A, Kisa U. Free radical-induced damage in experimental peripheral nerve injection injury. J Reconstr Microsurg 2003;19(6):401–406
15 Villarejo FJ, Pascual AM. Injection injury of the sciatic nerve (370 cases) Childs Nerv Syst 1993;9(4):229–232
16 Combes MA, Clark WK, Gregory CF, James JA. Sciatic nerve injury in infants. Recognition and prevention of impairment resulting from intragluteal injections. JAMA 1960;173:1336–1339
17 Von Hochstetter A. [Problems and technics of intragluteal injections. I. Effects of the drug and the individuality of the patient on the etiology of injection injuries] [in German]. Schweiz Med Wochenschr 1955;85(47):1138–1144
18 Senes FM, Campus R, Becchetti F, Catena N. Sciatic nerve injection palsy in the child: early microsurgical treatment and long-term results. Microsurgery 2009;29(6):443–448
19 Darby CP, Bradham G, Waller CE. Ischemia following an intragluteal injection of benzathine-procaine penicillin G mixture in a one-year-old boy. Clin Pediatr (Phila) 1973;12(8):485–487
20 Gentili F, Hudson AR, Kline D, Hunter D. Early changes following injection injury of peripheral nerves. Can J Surg 1980;23(2):177–182
21 Obach J, Aragones JM, Ruano D. The infrapiriformis foramen syndrome resulting from intragluteal injection. J Neurol Sci 1983;58(1):135–142
22 Haber M, Kovan E, Andary M, Honet J. Postinjection vastus lateralis atrophy: 2 case reports. Arch Phys Med Rehabil 2000;81(9):1229–1233
23 Kolb LC, Gray SJ. Peripheral neuritis as a complication of penicillin therapy. J Am Med Assoc 1946;132:323–326
24 Young F. Peripheral nerve paralysis following use of various serums. J Am Med Assoc 1932;98:1139–1143
25 Ling CM, Loong SC. Injection injury of the radial nerve. Injury 1976;8(1):60–62
26 Sobel E, Huang EY, Wieting CB. Drop foot as a complication of acupuncture injury and intragluteal injection. J Am Podiatr Med Assoc 1997;87(2):52–59
27 Streib EW, Sun SF. Injection injury of the sciatic nerve: unusual anatomic distribution of nerve damage. Eur Neurol 1981;20(6):481–484
28 Hadzic A, Dilberovic F, Shah S, et al.Combination of intraneural injection and high injection pressure leads to fascicular injury and neurologic deficits in dogs. Reg Anesth Pain Med 2004;29(5):417–423
29 Whitlock EL, Brenner MJ, Fox IK, Moradzadeh A, Hunter DA, Mackinnon SE. Ropivacaine-induced peripheral nerve injection injury in the rodent model. Anesth Analg 2010;111(1):214–220

30 Choi HR, Kondo S, Mishima S, et al. Axillary nerve injury caused by intradeltoid muscular injection: a case report. J Shoulder Elbow Surg 2001;10(5):493–495
31 Desai KR, Nemcek AA Jr. Iatrogenic brachial plexopathy due to improper positioning during radiofrequency ablation. Semin Intervent Radiol 2011;28(2):167–170
32 Sunderland S. Miscellaneous Causes of Nerve Injury. London Churchill Livingstone; 1991
33 Kline DG, Kim D, Midha R, Harsh C, Tiel R. Management and results of sciatic nerve injuries: a 24-year experience. J Neurosurg 1998;89(1):13–23
34 Mackinnon SE, Hudson AR, Gentili F, Kline DG, Hunter D. Peripheral nerve injection injury with steroid agents. Plast Reconstr Surg 1982;69(3):482–490
35 Ahuja B, Dhamija K. Sciatic neuropathy. Indian Pediatr 1984;21(10):797–801
36 Sharma S, Kale R. Post injection palsy in Chhatisgarh region. Indian Pediatr 2003;40(6):580–581
37 Fatunde OJ, Familusi JB. Injection-induced sciatic nerve injury in Nigerian children. Cent Afr J Med 2001;47(2):35–38
38 Maqbool W, Sheikh S, Ahmed A. Clinical, electrophysiological, and prognostic study of postinjection sciatic nerve injury: an avoidable cause of loss of limb in the peripheral medical service. Ann Indian Acad Neurol 2009;12(2):116–119
39 Ahuja B. Post injection sciatic nerve injury. Indian Pediatr 2003;40(4):368–369
40 Tak SR, Dar GN, Halwai MA, Mir MR. Post-injection nerve injuries in Kashmir: a menace overlooked. J Res Med Sci 2008;5:244–247
41 Adetunji OO, Olusola EP, Joseph AO, Dare OJ, Ademola OG, SegunO. Injection-induced sciatic nerve injuries among children seen at a Nigerian physiotherapy unit. Internet J Third World Med 2006;3(2)
42 Greensmith JE, Murray WB. Complications of regional anesthesia. Curr Opin Anaesthesiol 2006;19(5):531–537
43 Keskinbora K, Aydinli I. Treatment of neuropathic pain due to sciatic nerve injury following intramuscular injection with gabapentin in a child. Paediatr Anaesth 2008;18(11):1134–1135
44 Strasberg JE, Atchabahian A, Strasberg SR, Watanabe O, Hunter DA, Mackinnon SE. Peripheral nerve injection injury with antiemetic agents. J Neurotrauma 1999;16(1):99–107
45 Gentili F, Hudson AR, Hunter D. Clinical and experimental aspects of injection injuries of peripheral nerves. Can J Neurol Sci 1980;7(2):143–151
46 Yaffe B, Pri-Chen S, Lin E, Engel J, Modan M. Peripheral nerve injection injury: an experimental pilot study of treatment modalities. J Reconstr Microsurg 1986;3(1):33–37
47 Holbrook TJ, Pilcher C. The effects of injection of penicillin; peanut oil and beeswax, separately and in combination, upon nerve and muscle; an experimental study. Surg Gynecol Obstet 1950;90(1):39–44,illust
48 Lee JB, Choi SS, Ahn EH, et al. Effect of perioperative perineural injection of dexamethasone and bupivacaine on a rat spared nerve injury model. Korean J Pain 2010;23(3):166–171
49 Midha R, Guha A, Gentili F, Kline DG, Hudson AR. Peripheral nerve injection injury. In: Omer GE, Spinner M, Vanbeek Al, eds. Management of peripheral nerve problems. Philadelphia: PA: WB Saunders; 1999:406–413
50 Pandian JD, Bose S, Daniel V, Singh Y, Abraham AP. Nerve injuries following intramuscular injections: a clinical and neurophysiological study from Northwest India. J Peripher Nerv Syst 2006;11(2):165–171
51 Preston DC, Shapiro BE. Electromyography and Neuromuscular Disorders: Clinical-electrophysiologic Correlations. Butterworth-Heinemann; 2005
52 Aminoff MJ. Clinical Aspects of needle electromyography. In: Aminoff MJ, ed. Electromyography in Clinical Practice. New York, NY: Churchill Livingstone 1998:87–112
53 Bendszus M, Stoll G. Technology insight: visualizing peripheral nerve injury using MRI. Nat Clin Pract Neurol 2005;1(1):45–53
54 Shen J, Zhou CP, Zhong XM, et al. MR neurography: T1 and T2 measurements in acute peripheral nerve traction injury in rabbits. Radiology 2010;254(3):729–738
55 Cudlip SA, Howe FA, Griffiths JR, Bell BA. Magnetic resonance neurography of peripheral nerve following experimental crush injury, and correlation with functional deficit. J Neurosurg 2002;96(4):755–759
56 Pham M, Wessig C, Brinkhoff J, Reiners K, Stoll G, Bendszus M. MR neurography of sciatic nerve injection injury. J Neurol 2011;258(6):1120–1125
57 Chhabra A, Williams EH, Wang KC, Dellon AL, Carrino JA. MR neurography of neuromas related to nerve injury and entrapment with surgical correlation. AJNR Am J Neuroradiol 2010;31(8):1363–1368
58 Wood MB. Peroneal nerve repair. Surgical results. Clin Orthop Relat Res 1991; (267):206–210
59 Gilles FH, Matson DD. Sciatic nerve injury following misplaced gluteal injection. J Pediatr 1970;76(2):247–254
60 Cokluk C, Aydin K, Senel A. Presurgical ultrasound-assisted neuro-examination in the surgical repair of peripheral nerve injury. Minim Invasive Neurosurg 2004;47(3):169–172
61 Waitayawinyu T, Parisi DM, Miller B, et al. A comparison of polyglycolic acid versus type 1 collagen bioabsorbable nerve conduits in a rat model: an alternative to autografting. J Hand Surg Am 2007;32(10):1521–1529
62 Alluin O, Wittmann C, Marqueste T, et al. Functional recovery after peripheral nerve injury and implantation of a collagen guide. Biomaterials 2009;30(3):363–373
63 Wei AL, Liu SQ, Tao HY, Peng H. Repairing peripheral nerve defects with tissue engineered artificial nerves in rats. Chin J Traumatol 2008;11(1):28–33
64 Apel PJ, Garrett JP, Sierpinski P, et al. Peripheral nerve regeneration using a keratin-based scaffold: long-term functional and histological outcomes in a mouse model. J Hand Surg Am 2008;33(9):1541–1547
65 Ishikawa N, Suzuki Y, Dezawa M, et al. Peripheral nerve regeneration by transplantation of BMSC-derived Schwann cells as chitosan gel sponge scaffolds. J Biomed Mater Res A 2009;89(4):1118–1124
66 Bertleff MJ, Meek MF, Nicolai JP. A prospective clinical evaluation of biodegradable neurolac nerve guides for sensory nerve repair in the hand. J Hand Surg Am 2005;30(3):513–518
67 Rosson GD, Williams EH, Dellon AL. Motor nerve regeneration across a conduit. Microsurgery 2009;29(2):107–114
68 Moore AM, Kasukurthi R, Magill CK, Farhadi HF, Borschel GH, Mackinnon SE. Limitations of conduits in peripheral nerve repairs. Hand (N Y) 2009;4(2):180–186
69 Fu C, Hong G, Wang F. Favorable effect of local VEGF gene injection on axonal regeneration in the rat sciatic nerve. J Huazhong Univ Sci Technolog Med Sci 2007;27(2):186–189
70 Sevim S, Kaleagasi H. Sciatic injection injuries in adults: is dipyrone a foe to nerve.? Acta Neurol Belg 2009;109(3):210–213
71 Ohaegbulam SC. Peripheral nerve injuries from intramuscular injection of drugs. West Afr J Pharmacol Drug Res 1976;3(2):161–167
72 Plewnia C, Wallace C, Zochodne D. Traumatic sciatic neuropathy: a novel cause, local experience, and a review of the literature. J Trauma 1999;47(5):986–991
73 Bramhall RJ, Deveraj VS. Traumatic sciatic nerve palsy after gluteal injections. Eur J Plast Surg 2010;34(2):137–138

74 Di Marco A. [On paralysis of the sciatic nerve caused by intragluteal injections of drugs] [in Italian]. Clin Pediatr (Bologna) 1961;43:230–241
75 Ramos-Fernández JM, Oliete-García FM, Roldán-Aparicio S, Kirchschläger E, Barrio-Nicolás A. [Neonatal sciatic palsy: etiology and outcome of 21 cases] [in Spanish]. Rev Neurol 1998;26(153):752–755
76 Mishra P, Stringer MD. Sciatic nerve injury from intramuscular injection: a persistent and global problem. Int J Clin Pract 2010;64(11):1573–1579
77 Marcuse EK, MacDonald NE. Neurologic injury after vaccination in buttocks. CMAJ 1996;155(4):374
78 Wexberg E. Neuritis und polyneuritis. In: Bumke O, Foerster O, eds. Handbuch der Neurologie. Berlin: Springer-Verlag; 1935;87
79 Iyer VG, Shields CB. Isolated injection injury to the posterior femoral cutaneous nerve. Neurosurgery 1989;25(5):835–838
80 Esteban B, Díaz J. Lesiones del nervio ciático postinyección glutea. 1. EstudioClínico. Rev Orthop Traum 1981;25:67–80
81 Bergeson PS, Singer SA, Kaplan AM. Intramuscular injections in children. Pediatrics 1982;70(6):944–948
82 Kim DH, Murovic JA, Tiel R, Kline DG. Management and outcomes in 353 surgically treated sciatic nerve lesions. J Neurosurg 2004;101(1):8–17
83 Harsh V, B Chengazhacherril R, Sharma K, et al. Sciatic nerve injection palsy in children. Iran J Child Neurol 2016;10(4):86–87
84 Ross Kerr JC, Wood MJ. Canadian Fundamentals of Nursing. 2nd ed. Toronto: Mosby; 2001
85 Bay E. Injektionsschäden des Nervus ischiadicus. Dtsch Med Wschr 1961;86:505–508
86 Hirasawa Y, Katsumi Y, Küsswetter W, Sprotte G. [Experimental studies on peripheral nerve injuries caused by injection needles] [in German] Reg Anaesth 1990;13(1):11–15
87 Turner GG. The site for intramuscular injections. Lancet 1920;2:819–825
88 Bourrel P, Souvestre R. [A particular nervous traumatology: lesions of sciatic nerve by quinine intragluteal injections (author's transl)] [in French]. Med Trop (Mars) 1982;42(2):209–213
89 Kpadonou GT, Biaou O, Fiossi-Kpadonou E, Hans-Moevi AA, Alagnide E, Odoulami H. [Hip paraosteoarthropathy after sciatica nerve injury by quinine intramuscular injection]. Ann Readapt Med Phys 2007;50(1):42–47
90 Hanson DJ. Intramuscular injection injuries and complications. GP 1963;27:109–115
91 Uppal HS, Gwilym SE, Crawfurd EJ, Birch R. Sciatic nerve injury caused by pre-operative intraneural injection of local anaesthetic during total hip replacement. J Bone Joint Surg Br 2007;89(2):242–243
92 Yeremeyeva E, Kline DG, Kim DH. Iatrogenic sciatic nerve injuries at buttock and thigh levels: the Louisiana State University experience review. Neurosurgery 2009;65(4, Suppl) :A63–A66
93 Napiontek M, Ruszkowski K. Paralytic drop foot and gluteal fibrosis after intramuscular injections. J Bone Joint Surg Br 1993;75(1):83–85
94 Bigos SJ, Coleman SS. Foot deformities secondary to gluteal injection in infancy. J Pediatr Orthop 1984;4(5):560–563
95 Ramtahal J, Ramlakhan S, Singh K. Sciatic nerve injury following intramuscular injection: a case report and review of the literature. J Neurosci Nurs 2006;38(4):238–240
96 Curtiss PH Jr, Tucker HJ. Sciatic palsy in premature infants. A report and follow-up study of ten cases. JAMA 1960;174:1586–1588
97 Takagi T, Nakamura M, Yamada M, et al. Visualization of peripheral nerve degeneration and regeneration: monitoring with diffusion tensor tractography. Neuroimage 2009;44(3):884–892
98 Lehmann HC, Zhang J, Mori S, Sheikh KA. Diffusion tensor imaging to assess axonal regeneration in peripheral nerves. Exp Neurol 2010;223(1):238–244
99 Huang Y, Yan Q, Lei W. [Gluteal sciatic nerve injury and its treatment] [in Chinese]. Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi 2000;14(2):83–86
100 Topuz K, Kutlay M, Simşek H, Atabey C, Demircan M, Senol Güney M. Early surgical treatment protocol for sciatic nerve injury due to injection–a retrospective study. Br J Neurosurg 2011;25(4):509–515
101 Chen Y, Wang D, Wang Z, Weng Z, Deng Z. [Effect of adenovirus expressing NGF on sciatic nerve injury in rats] [in Chinese]. Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi 2009;23(8):947–953
102 Unezaki S, Yoshii S, Mabuchi T, Saito A, Ito S. Effects of neurotrophic factors on nerve regeneration monitored by in vivo imaging in thy1-YFP transgenic mice. J Neurosci Methods 2009;178(2):308–315
103 Sulaiman OA, Gordon T. Role of chronic Schwann cell denervation in poor functional recovery after nerve injuries and experimental strategies to combat it. Neurosurgery 2009;65(4, Suppl) :A105–A114
104 Wilson AD, Hart A, Wiberg M, Terenghi G. Acetyl-l-carnitine increases nerve regeneration and target organ reinnervation - a morphological study. J Plast Reconstr Aesthet Surg 2010;63(7):1186–1195
105 Behr B, Schnabel R, Mirastschijski U, Ibrahim B, Angenstein F, Schneider W. Magnetic resonance imaging monitoring of peripheral nerve regeneration following neurotmesis at 4.7 Tesla. Plast Reconstr Surg 2009;123(6):1778–1788
106 Robert H. Interet de la neurolyse apres paralysie sciatique par injection intra-fessiere chez l'enfant. fre [The value of neurolysis following sciatic paralysis due to intrabuttock injections in children]. Chir Pediatr 1985;26(3):197–200
107 Gilsenan I. A practical guide to giving injections. Nurs Times 2000;96(33):43–44
108 Dugas B, Esson L, Ronaldson S. Nursing Foundations: A Canadian Perspective. Scarborough: Prentice; 1999
109 Johnson EW, Raptou AD. A study of intragluteal injection. Arch Phys Med Rehabil 1965;46:167–177
110 Craven RF, Hirnle CJ. Fundamentals of Nursing: Human Health and Function. Philadelphia: Lippincott, Williams Wilkins; 2003
111 Brandt PA, Smith ME, Ashburn SS, Graves J. IM injections in children. Am J Nurs 1972;72(8):1402–1406
112 Zelman S. Notes on techniques of intramuscular injection. The avoidance of needless pain and morbidity. Am J Med Sci 1961;241:563–574
113 Brandt PA, Smith ME, Ashburn SS, Graves J. IM injections in children. Am J Nurs 1972;72(8):1402–1406
114 Kozier B, Erb G, Berman AJ, Burke K. Fundamentals of Nursing: Concepts, Process, and Practice. 6th ed. Prentice Hall Health in Upper Saddle River, New Jersey: Prentice Hall Health; 2000
115 Hughes WT. Complication resulting from an intramuscular injection. J Pediatr 1967;70(6):1011–1013
116 Müller-Vahl H. Adverse reactions after intramuscular injections. Lancet 1983;1(8332):1050
117 Rodger MA, King L. Drawing up and administering intramuscular injections: a review of the literature. J Adv Nurs 2000;31(3):574–582
118 Orebaugh SL. The femoral nerve and its relationship to the lateral circumflex femoral artery. Anesth Analg 2006;102(6):1859–1862
119 Standring SM. The Anatomical Basis of Clinical Practice. 40th ed. Philadelphia Elsevier; 2008
120 McMinn RMH. Last's Anatomy. Regional and Applied. 9th ed. Philadelphia, PA: Elsevier; 1998:156

121 Moore K, Dalle AF, Agur AMR. Clinically Oriented Anatomy. 6th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2009
122 Beyea SC, Nicoll LH. Administration of medications via the intramuscular route: an integrative review of the literature and research-based protocol for the procedure. Appl Nurs Res 1995;8(1):23–33
123 Nicholas JJ, Lennard TA. Joint and Soft Tissue Injection Techniques. Braddom RL, editor. Philadelphia: WB Saunders; 2000
124 Zuckerman JD, Meislin RJ, Rothberg M. Injections for joint and soft tissue disorders: when and how to use them. Geriatrics 1990;45(4):45–52
125 Hunter LY, Louis DS, Ricciardi JR, O'Connor GA. The saphenous nerve: its course and importance in medial arthrotomy. Am J Sports Med 1979;7(4):227–230
126 Iizuka M, Yao R, Wainapel S. Saphenous nerve injury following medial knee joint injection: a case report. Arch Phys Med Rehabil 2005;86(10):2062–2065
127 Belart W. Butazolidin als Antirheumatikum. Dtsch Med Wschr 1953;78:129–131
128 Lachman E. Applied anatomy of intragluteal injections. Am Surg 1963;29:236–241
129 Tong HC, Haig A. Posterior femoral cutaneous nerve mononeuropathy: a case report. Arch Phys Med Rehabil 2000;81(8):1117–1118
130 Kim JE, Kang JH, Choi JC, Lee JS, Kang SY. Isolated posterior femoral cutaneous neuropathy following intragluteal injection. Muscle Nerve 2009;40(5):864–866
131 Dumitru D, Nelson MR. Posterior femoral cutaneous nerve conduction. Arch Phys Med Rehabil 1990;71(12):979–982
132 Snow DM, Reading J, Dalal R. Lateral plantar nerve injury following steroid injection for plantar fasciitis. Br J Sports Med 2005;39(12):e41
133 Miller MA, Pisani E. The cost of unsafe injections. Bull World Health Organ 1999;77(10):808–811
134 Small SP. Preventing sciatic nerve injury from intramuscular injections: literature review. J Adv Nurs 2004;47(3):287–296