Nerve Lesions Associated with Limb-Lengthening

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**Background:** Nerve injury is one of the most serious complications associated with limb-lengthening. We examined the risk, assessment, and treatment of nerve lesions associated with limb-lengthening.

**Methods:** We retrospectively studied the records on 814 limb-lengthening procedures. Nerve lesions were defined by clinical signs and symptoms of motor function impairment, sensory alterations, referred pain in the distribution of an affected nerve, and/or positive results of quantitative sensory testing with use of a pressure specified sensory device.

**Results:** Seventy-six (9.3%) of the limbs had a nerve lesion. Eighty-four percent of the nerve lesions occurred during gradual distraction, and 16% occurred immediately following surgery. The pressure specified sensory device showed 100% sensitivity and 86% specificity in the detection of nerve injuries. The patients in whom the lesion was diagnosed with this method, or with this method as well as with nerve conduction studies, had significantly faster recovery than did those diagnosed on the basis of clinical symptoms or nerve conduction studies alone (p = 0.02). Patients undergoing double-level tibial lengthening and those with skeletal dysplasia were at higher risk for nerve lesions (77% and 48%, respectively). Nerve decompression was performed in fifty-three cases (70%). The time between the diagnosis and the surgical decompression was strongly associated with the time to recovery (p = 0.0005). Complete clinical recovery was achieved in seventy-four of the seventy-six cases.

**Conclusions:** Early detection based on signs and symptoms or testing with a pressure specified sensory device improves the prognosis for nerve injury that occurs during limb-lengthening. Of the methods that we used to identify neurologic compromise, testing with the pressure specified sensory device was the most sensitive. Aggressive early treatment (slowing the rate of lengthening and/or performing decompression) allows continued lengthening without incurring permanent nerve injury. When indicated, decompression of the affected nerve should be performed as soon as possible, thereby improving the chances of and shortening the time to complete recovery.

**Level of Evidence:** Prognostic study, Level II-1 (retrospective study). See Instructions to Authors for a complete description of levels of evidence.

Nerve injury is one of the most serious complications associated with limb-lengthening. Acute methods of limb-lengthening are often complicated by nerve palsy and chronic neurogenic pain. Nerve injury is less common with gradual lengthening, but it has been reported in many series. Nerve injury has been reported to occur immediately postoperatively or during the distraction process. When the nerve injury occurs immediately postoperatively, the cause is usually apparent: it can be the result of direct surgical trauma from a wire, a drill bit, or instruments used to perform the osteotomy or it can be due to indirect stretch injury from acute correction of deformity. In contrast, the causes of nerve injuries occurring during the gradual distraction process are less obvious.

In an experimental study of limb-lengthening performed on dogs, Strong et al. concluded that nerve injury occurs at the level of the nerve roots. Paley reported peroneal nerve entrapment at the level of the neck of the fibula. Young et al. observed electromyographic changes that returned to normal after a period of time. Despite these findings, there has been no consensus regarding the etiology of nerve injury during lengthening.

Nerve injury has been considered to be a contraindication to further lengthening because of the risk of permanent injury. However, Paley reported that peroneal nerve decompression can lead to full recovery even if the lengthening process is continued. The purpose of the present study was to determine the prevalence, presentation, predisposing and causative factors, diagnostic and treatment modalities, and prognosis of nerve lesions associated with limb-lengthening.

**Materials and Methods**

Between January 1989 and December 1999, 814 limbs were lengthened in 650 patients by the two senior authors (D.P.
and J.E.H.). All of the procedures were accomplished by means of gradual distraction with an Orthofix (Orthofix SRL, Bussolegro, Verona, Italy), Heidelberg (Smith and Nephew, Memphis, Tennessee), or Ilizarov (Smith and Nephew) external fixator. Eighty-three upper and 731 lower extremities were lengthened. A retrospective review of the patients’ charts and results of nerve studies identified nerve lesions in seventy-six limbs in forty-two patients.

Double-level lengthening of one bone, double-level osteotomy with single-level lengthening of one bone, and ipsilateral femoral and tibial lengthening or radial and ulnar lengthening were each considered to be one limb-lengthening event. A simultaneous bilateral lengthening was considered to be two limb-lengthening procedures.

We classified all cases of nerve irritation, nerve compromise, and nerve injury that were either clinically evident or indicated by nerve studies as nerve lesions. Therefore, a nerve lesion was defined as the presence of sensory and/or motor signs and symptoms of nerve irritation or compromise, and/or positive results of nerve conduction studies, and/or (since 1995) positive results of quantitative sensory testing with use of a pressure specified sensory device (PSSD; NK Biotechnical Corporation, Minneapolis, Minnesota). The earliest symptom of nerve irritation was referred pain and/or paresthesia in the distribution of a sensory nerve that was unexplained by any other cause or source of pain (for example, pin-site pain, muscle cramp, or muscle contracture). Paresthesias and referred pain often could be confirmed by direct pressure at the point of entrapment of a nerve (for example, at the peroneal tunnel at the neck of the fibula) and, in some cases involving entrapment by a wire or half-pin, could be elicited by tapping on the offending wire or pin. Symptoms of nerve compromise included numbness or weakness in the foot or hand of the limb being lengthened. Most often, the earliest sign of nerve irritation was hyperesthesia in the distribution of a sensory nerve, with hypoesthesia occurring soon thereafter. Muscle weakness and paralysis were late signs of nerve lesions associated with gradual distraction.

Nerve lesions were classified as type 1, surgical nerve injury, or type 2, gradual stretch nerve injury. The surgical nerve injuries were further divided into three groups: type 1a, intraoperative direct injury by a wire, drill bit, half-pin, or other surgical instrument; type 1b, intraoperative acute stretch due to acute correction of a bone or joint deformity, a rotational osteosclerosis maneuver, or inadvertent acute distraction at the osteotomy site; and type 1c, an “acute-on gradual” stretch injury (an acute nerve stretch resulting from acute correction of a deformity or manipulation or surgery performed during the distraction phase). These lesions were diagnosed clinically during the immediate period after surgical intervention and before distraction was begun or resumed.

Gradual stretch nerve injury occurs during the distraction phase and is not present before the onset of distraction. This class of lesion was divided into two groups: type 2a, injury caused by gradual stretch only, and type 2b, gradual stretch injury associated with tethering or impingement of a nerve by an external fixation wire, half-pin, or displaced bone. For example, a type-2b injury might occur when a pin located near a nerve becomes a tether on the nerve as gradual traction is applied to the nerve during lengthening. These injuries are not considered surgical lesions because there is no evidence of a nerve injury before the onset of distraction. As distraction proceeds, the nerve becomes irritated and eventually compromised because of the tethering or impingement by the hardware or bone.

Patients were monitored for evidence of nerve compromise every two weeks during the distraction phase. The modalities with which nerve lesions were diagnosed and confirmed varied during the eleven-year study period. Between 1989 and 1995, the diagnosis of a nerve lesion was made clinically and confirmed with nerve conduction studies. Two types of electrical nerve studies were used: standard nerve conduction velocity studies and near-nerve conduction velocity studies. Standard nerve conduction velocity was measured by surface stimulation above and recording below the level of the lesion.

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In 1995, we began to perform quantitative sensory testing using the pressure specified sensory device (PSSD) every two weeks during the distraction phase. A baseline test was performed before the surgery, and another test was done after the surgery but before distraction was begun (during the first week after surgery). There was an overlap of cases in 1995, with patients undergoing both near-nerve conduction studies and PSSD testing.

The treatment of the nerve lesion was individualized for each patient. With few exceptions, the treatment was related to the classification of the nerve injury. Type-1a, 1b, and 1c surgical nerve injuries were treated early with surgical exploration and nerve decompression. Type-2a lesions were treated initially by slowing the distraction rate or temporarily discontinuing the distraction if there were only sensory symptoms and signs or positive PSSD results. If the sensory symptoms or signs did not abate, if motor signs were present, or if the results of the near-nerve conduction study became positive, the nerve was decompressed surgically. Type-2b lesions were treated by identifying and removing the offending wire or pin and performing nerve decompression.

The site of nerve decompression was the neck of the fibula for the peroneal nerve, the tarsal tunnel for the posterior tibial nerve, the radial neck and cubital fossa for the radial nerve, the carpal tunnel for the median nerve, and either Guyon’s canal or the cubital tunnel for the ulnar nerve. Standard methods of nerve decompression were used, except for treatment of the peroneal nerve, for which a modification of the conventional method was employed. This modification involves a long incision crossing the knee flexion crease and following the peroneal nerve from the posterior aspect of the
biceps muscle to its entrance into the peroneal muscle fascia. We modified it to a 3-cm incision at the level of the neck of the fibula, parallel with the oblique path of the nerve, without crossing the knee flexion crease. The dissection is continued down to and through the superficial fascia. The nerve is identified in a band of fat entering the peroneal muscle fascia, dissected free proximally, and then followed distally to the peroneal muscle fascia. The fascia is incised, the muscle is retracted, and the underlying fascial band over the nerve is identified and cut.

In addition to this well-known entrapment point for the common peroneal nerve is a second, unrecognized, entrapment point for the deep peroneal nerve, which courses through the lateral compartment of the leg to innervate the muscles of the anterior compartment. To enter the anterior compartment, this nerve must pass under the intermuscular septum that runs perpendicular to the nerve. Even in the normal situation, there is little clearance for the nerve under the septum. With lengthening, the septum becomes taut and can entrap the deep peroneal nerve. To decompress this second entrapment point, the intermuscular septum is incised from superficial to deep.

The extent of sensory and motor recovery was documented for all patients. The time to full clinical recovery was also documented. All patients were followed until complete recovery had been achieved or, if complete recovery did not occur, until maximum recovery had taken place, at five years after the nerve injury. The average duration of follow-up was five years (range, one to eleven years).

Results

Forty-two (6.5%) of the 650 patients and seventy-six (9.3%) of the 814 limbs were identified as having incurred a nerve lesion during the limb-lengthening procedure and/or process. The nerve lesion was diagnosed with clinical evaluation alone in seven cases (9%), nerve conduction studies in twenty-four (32%), PSSD testing in thirty-one (41%), and PSSD testing as well as nerve conduction studies in fourteen (18%). The signs and symptoms at the time of the diagnosis consisted of referred pain for eighteen (24%) of the nerve lesions, sensory changes for twenty (26%), and motor weakness for thirty-two (42%). Six (8%) lesions were asymptomatic.

PSSD testing was performed for 132 limbs. To study the sensitivity and specificity of this method, a nerve lesion was redefined as impairment of motor function, and/or sensory alterations (paresthesia, dysesthesia, or hypoesthesia), and/or referred pain in the distribution of the nerve not related to local pins or wires. Forty-five limbs had positive PSSD results. Six of these limbs exhibited no clinical signs or symptoms of nerve injury. None of the eighty-seven limbs with negative PSSD results ever had clinical or electrodiagnostic evidence of nerve injury (Table I). Therefore, the sensitivity of this method was 100% and the specificity was 87% relative to clinical signs.

| TABLE I Sensitivity and Specificity of Testing with the Pressure Specified Sensory Device (PSSD) in Relation to Clinical Signs and Symptoms of a Nerve Lesion* |
|---------------------------------|---|---|---|
| PSSD Results | Clinical Results | | |
| | Positive | Negative | Total |
| Positive | 39 | 6 | 45 |
| Negative | 0 | 87 | 87 |
| Total | 39 | 93 | 132 |

*The PSSD method showed 100% sensitivity and 87% specificity relative to clinical diagnosis.
and symptoms. Fourteen limbs with clinical evidence of nerve compromise were tested with both the near-nerve needle-electrode and the PSSD method. Two limbs had positive results for both tests, whereas twelve had positive PSSD results but initially negative results of the near-nerve testing, which later became positive.

A comparison of the different diagnostic tests with the times to nerve recovery was performed (Fig. 1). The patients in whom the lesion was diagnosed with PSSD testing or with PSSD testing and nerve conduction studies had significantly faster recovery than did those in whom the lesion was diagnosed on the basis of clinical symptoms or nerve conduction studies alone (p = 0.02).

Limb-length discrepancy was the indication for 683 of the lengthening procedures, and short stature was the indication for 131 (Table II). There were three types of limb-length discrepancy: congenital, developmental, and posttraumatic. There were two types of short stature: that due to skeletal dys-
plasia and constitutional short stature.

The average age of all of the patients who underwent lengthening was 22.2 years (range, six months to seventy years) at the time of the procedure. The average age of those who incurred a nerve injury was 15.4 years (range, five to forty-three years). Patients were divided into three age groups: younger than six years, six to 17.9 years, and eighteen years and older. The frequency of nerve injury was two (3%) of seventy-six in the first group, twenty-nine (11%) of 274 in the second group, and eleven (4%) of 300 in the third. There was no significant relationship between age group and prevalence of nerve lesions (p = 0.54) or time to recovery (r² = 0.1159).

Twelve (16%) of the nerve lesions were classified as type 1 (surgical nerve injuries), and sixty-four (84%) were classified as type 2 (gradual stretch nerve injuries). Five of the surgical nerve injuries were type 1a (intraoperative direct surgical injuries), five were type 1b (intraoperative acute stretch injuries), and two were type 1c (acute-on-gradual stretch injuries). There were fifty-seven type-2a lesions (injuries caused by gradual stretch only) and seven type-2b lesions (gradual stretch injuries associated with tethering or impingement).

Intraoperative direct surgical nerve injuries (type 1a) occurred most commonly during the earlier years of our experience (from 1991 to 1994). All type-1a injuries occurred in patients undergoing circular external fixation (Ilizarov); the injuries were associated with two procedures for lengthening the femur, one for lengthening both the femur and the tibia, and two for lengthening both the radius and the ulna. Acute stretch injuries occurred more commonly in the later years of the study, and it was our subjective impression that this correlated with our trend to perform more acute corrections of deformities. In contrast, type-2 gradual stretch injuries had no particular distribution according to the year of lengthening.

A nerve lesion occurred in thirty (77%) of the thirty-nine limbs undergoing double-level tibial lengthening, in twenty (16%) of the 127 undergoing simultaneous femoral and tibial lengthening (only one of which was undergoing double-level tibial lengthening), in fourteen (4%) of the 351 undergoing single-level tibial lengthening, in four (2%) of the 214 undergoing single-level femoral lengthening, in six (12%) of the forty-nine undergoing humeral lengthening, in two (33%) of the six undergoing combined radial and ulnar lengthening, and in none of the twenty-eight undergoing isolated radial or ulnar lengthening (Table III).

The average amount of lengthening was 10.3 cm (range, one to 16.5 cm) in the series as a whole. The average amount of lengthening at the time of the first finding of nerve compromise was 4.0 cm (range, 0 to 12.9 cm). Although there was no significant association between the amount of lengthening at the time of the first signs or symptoms and the prevalence of nerve lesions, there was a trend for type-2 lesions to occur in the range of 2 to 5.5 cm of lengthening (Fig. 2-A). A similar trend was observed for the limbs treated with double-level tibial lengthening (Fig. 2-B). However, the data clearly indicate that there was no specific amount or percentage of lengthening that correlated with the occurrence of nerve lesions, and that nerve compromise can occur at any point in the lengthening process from 0.5 to 12.9 cm.

Once a nerve lesion was diagnosed, the treatment was based first on the type of lesion (surgical) or gradual stretch and then on the clinical presentation and results of diagnostic testing. All surgical lesions were treated with surgical decompression. Of the gradual stretch lesions associated with motor weakness, twenty were treated with decompression and six, by slowing the distraction rate only; of those associated with sensory changes, thirteen were treated with decompression and two, by slowing the distraction rate (which was accomplished with pin removal in one case and by discontinuing the lengthening in the other); and of those associated with referred pain, nine were treated by slowing the distraction rate only, seven

### TABLE II Prevalence of Nerve Lesions According to the Indication for Limb-Lengthening

<table>
<thead>
<tr>
<th>Treatment of Short Stature</th>
<th>Dysplasia</th>
<th>Constitutional Short Stature</th>
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</thead>
<tbody>
<tr>
<td>No. of nerve lesions</td>
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<td>3</td>
</tr>
<tr>
<td>Total no. of lengthening procedures</td>
<td>115</td>
<td>3</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td>48</td>
<td>19</td>
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</tbody>
</table>

### TABLE III Prevalence of Nerve Lesions According to the Segment Lengthened

<table>
<thead>
<tr>
<th>Single-Level Femoral</th>
<th>Single-Level Humeral</th>
<th>Single-Level Tibial</th>
<th>Double-Level Tibial</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of lesions</td>
<td>4</td>
<td>6</td>
<td>14</td>
</tr>
<tr>
<td>Total no. of lengthening procedures</td>
<td>214</td>
<td>49</td>
<td>351</td>
</tr>
<tr>
<td>Prevalence (%)</td>
<td>2</td>
<td>12</td>
<td>4</td>
</tr>
</tbody>
</table>
were treated with decompression, and one was observed only. Of the forty-five cases with positive PSSD results, thirty-nine had clinical signs and symptoms of nerve injury and six were asymptomatic. Of the six asymptomatic cases, four were treated by slowing the distraction rate only, one was treated by discontinuing the lengthening, and one was treated with decompression. The patient who underwent decompression was already undergoing decompression on the contralateral side because of positive PSSD results and clinical symptoms on that side. All of these nerve lesions resolved within twenty-one days.

There were six cases of isolated motor weakness that were not treated with decompression. In two, the results of the near-nerve conduction recording were negative. In the other four cases, there were only subtle, mild alterations that were revealed by motor examination. All six patients were treated by slowing the distraction rate, and recovery was achieved in all within sixty days.

The time elapsed from the diagnosis to the decompression was strongly correlated with the time to recovery ($p = 0.0005$). The patients who were treated with decompression within fourteen days had recovery at an average of thirty-five days, whereas those treated after fourteen days had recovery at an average of 224 days (Fig. 3). Two patients had only partial resolution of the nerve lesion, and they were not included in this analysis. Both of those patients had complete peroneal nerve palsy, as a result of a direct injury by the drill bit in one and due to an acute valgus-to-varus angular correction of the tibia in the other. Neither was treated with early decompression. Decompression was finally performed 4.5 and nine months after the initial nerve injury. All other nerve lesions resolved completely, at one to 703 days after the diagnosis.

In total, fifty-three lesions were treated with decompression; twenty-one, by slowing the distraction rate only; one, by removing the offending pin; and one, with observation alone.

### Discussion

The prevalence of nerve injury, based on clinical diagnosis, has ranged from 0.7% to 30% in studies of limb-lengthening. The overall prevalence of nerve lesions in our study was 9.3%.

Double-level tibial lengthening was associated with the highest prevalence of nerve injury (77%); thirty of thirty-nine. Simultaneous femoral and tibial lengthening was associated with a much lower prevalence of nerve injury (16%; twenty of 127, which was not much higher than the 9.3% prevalence for the entire group. However, the risk associated with simultaneous femoral and tibial lengthening was substantially higher than that associated with isolated single-level femoral lengthening (2%); four of 214 or tibial lengthening (4%); fourteen of 351. Previous studies did not demonstrate a higher prevalence of nerve compromise with double-level lengthening of the femur or tibia compared with that associated with isolated femoral or tibial lengthening. Double-level lengthening greatly decreases the distraction and consolidation times since peroneal nerve decompression is a relatively minor procedure, it is worth performing double-level lengthening even if decompression will become necessary. All of the patients treated with double-level tibial lengthening in our series had complete resolution of the nerve lesion.

The diagnosis associated with the highest risk for nerve

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**TABLE III (continued)**

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lesions in association with lengthening was skeletal dysplasia, with a prevalence of 48% (fifty-five of 115). Patients with skeletal dysplasia are especially at risk if they undergo double-level tibial lengthening; therefore, double-level tibial lengthening in such patients must be performed in conjunction with additional monitoring of postoperative nerve function. We currently monitor high-risk patients for evidence of nerve compromise with biweekly PSSD testing. Patients must be warned about the early symptoms of nerve compromise and instructed to notify their surgeon immediately if they experience any of those symptoms.

It seems logical that the likelihood of nerve injury would be related to the amount or percentage of lengthening. However, previous studies have not demonstrated an association between the number of centimeters of lengthening and the occurrence of nerve injury. We also found no association between the prevalence of nerve lesions and the amount of lengthening at the time of the first clinical signs and symptoms. Interestingly, the majority of the nerve lesions, especially in the leg, presented after 2 to 5.5 cm of lengthening had occurred. This suggests that the problem may be an anatomic predisposition to peroneal entrapment at the neck of the fibula rather than stretch injury to the nerve, which would be expected to be more common with a greater amount of lengthening. Polo et al. also concluded, on the basis of electrodiagnostic studies, that the site of peroneal nerve injury must be close to the head of the fibula.

The five intraoperative direct surgical injuries (type 1a) all occurred between 1992 and 1993 and at the time of application of the Ilizarov external fixator; we believe that this represents the surgical learning curve. Injury from acute correction of a deformity (a type-1b nerve lesion) is well recognized and can be related to nerve stretch, nerve entrapment, and/or compartment syndrome. The prevalence of this type of nerve injury among our patients has increased in recent years as a result, we believe, of our increased performance of acute corrections of deformities. The risk associated with acute correction is amplified if the correction is performed during the distraction phase, when all of the soft tissues are already taut (a type-1c injury). Accordingly, acute angular correction during lengthening should either be avoided or be performed very selectively in conjunction with close monitoring of nerve function. In cases of direct surgical or acute stretch injury, the swelling that is expected to occur around the nerves leads to further compromise of the nerve within its tunnel. Therefore, decompression of the nerve tunnel allows the nerve to swell and recover without a second crush phenomenon. For this reason, decompression is indicated for these acute lesions.

Most (sixty-four [84%]) of the seventy-six nerve injuries associated with lengthening in our series were gradual stretch injuries (type 2). In contrast to acute stretch injuries and direct surgical injuries, which present with more profound motor and sensory signs, gradual stretch injuries present with subtle symptoms and are easily overlooked until more obvious sensory and motor signs are present.

Measurements of nerve conduction velocity are often misleading during limb-lengthening because usually the nerve is only partially injured. The conduction velocity may appear normal because conduction is measured along the fastest intact axons. The near-nerve conduction technique employs a very fine needle that is introduced directly into the nerve sheath. It measures a direct action potential from the nerve and can reveal evidence of partial nerve injury. Wexler et al. concluded that the near-nerve technique was more useful than standard mixed nerve or motor nerve conduction-velocity measurements for early detection of nerve injuries in patients undergoing limb-lengthening.

Tassler and Dellon compared the sensitivity of PSSD testing with that of electrodiagnostic testing for the monitoring of nerve function in four patients with peripheral nerve entrapment syndrome. They found the PSSD test to be more sensitive than the electrodiagnostic test for all four. Weber et al. compared nerve conduction velocity testing with PSSD testing with respect to sensitivity, specificity, and the pain associated with the test in a prospective, blinded evaluation of patients with carpal tunnel syndrome. They reported that PSSD testing was at least as sensitive and specific as electrodiagnostic methods and caused substantially less pain for the patients. Bhave et al. first reported the use of PSSD testing to monitor nerve function in patients treated with limb-lengthening. Our results in the present study demonstrated that quantitative sensory testing with the PSSD can detect evidence of nerve compromise even earlier than the near-nerve test can. The patients in whom the nerve lesion was diagnosed with PSSD testing had significantly faster recovery than did those diagnosed with nerve conduction velocity testing. PSSD evaluation is an objective, simple, noninvasive, painless, and relatively inexpensive technique that does not take a lot of time to perform and can easily be repeated every two to three weeks. A limitation of PSSD testing is that it requires the patient’s cooperation and thus cannot be used for patients with cognitive deficits or for children younger than eight years old.

We generally perform decompression in patients with motor signs; however, decompression was not performed in two such patients in whom the results of the near-nerve recording were negative and in four patients in whom the alterations were mild and it was possible to slow the rate of distraction and observe the clinical course. Slowing the distraction rate was our first step when a sign or symptom of nerve injury was detected or when nerve conduction studies or PSSD testing showed alterations in nerve function. This protocol was also used by Eldridge and Bell at the first indication of positive signs and symptoms.

It is not easy to slow or stop the distraction rate in patients being treated with an implanted distraction device. The risk of nerve injury associated with lengthening with an implanted device should be the same as that associated with conventional methods because most nerve injuries are related to the distraction process and not the device per se. Nerve decompression may be the only treatment alternative for patients with an implanted lengthener, and these patients should be advised accordingly.
We found a significant association between the time to nerve decompression and the time to nerve recovery (p = 0.0005). Earlier detection of nerve compromise leads to earlier treatment and earlier recovery. Although there are almost no reports on nerve compression associated with lengthening, there are numerous reports on peripheral nerve entrapment. In the study by Mont et al., peroneal nerve decompression was performed two months after nerve injury if the injury had not resolved. Recovery was achieved within thirty-six months after decompression in their patients. Decompression of the peroneal nerve is not a new procedure. It has been performed to treat entrapment neuropathies caused by external compression, ganglions, muscle herniation, and compartment syndrome. However, decompression is not a well-recognized treatment for nerve injury associated with limb-lengthening. The occurrence of nerve injury during lengthening has been considered a contraindication to continuing the distraction.

Our study demonstrated evidence of nerve entrapment and pathologic findings during all of the decompression procedures. The pathologic findings included hemorrhage, flattening of the nerve, indentation of the nerve at the entrance to the fascial tunnel, and lack of vascular injection in the region of entrapment. In some cases of motor paresis, intraoperative direct electrical nerve stimulation before decompression demonstrated no muscle contraction whereas repeat stimulation applied just after decompression demonstrated good muscle contraction. Such dramatic motor recovery, which in some cases occurred immediately after the surgery, confirms the validity of this approach as well as the site and diagnosis of nerve entrapment.

We did not have to abandon the treatment goal of lengthening in our patients with evidence of nerve injury. Instead, we were able to continue distraction without concern that nerve recovery would be adversely affected. Therefore, nerve decompression should be considered a staged secondary procedure necessary in the event of nerve compromise that does not respond to slowing the rate of distraction. In our opinion, it is no longer acceptable to allow a motor nerve lesion to progress or remain without recovery during a lengthening procedure. Aggressive early treatment (slowing the distraction rate and/or performing decompression) allows continued lengthening without incurring permanent nerve injury. When decompression is indicated, the affected nerve should be decompressed as soon as possible, thereby improving chances of recovery and shortening the time to complete recovery.

References

NERVE LESIONS ASSOCIATED WITH LIMB-LENGTHENING


