In the literature on spinal disorders, the development of postoperative cauda equina syndrome in patients undergoing lumbar discectomy is a well-documented complication. Although the origin of the condition has remained unknown, several theories have been proposed to explain the underlying pathogenetic mechanism. In the vast majority of cases, no explanation based on immediate postoperative MR imaging is usually recognized. In this communication we report two cases of postoperative cauda equina syndrome that occurred after lumbar microdiscectomy. No abnormalities were seen on the postoperative imaging studies, but significant changes were observed in the intraoperative SSEPs.

CLINICAL MATERIAL AND METHODS

Treatment Protocol

At our institution, intraoperative electrophysiological monitoring, including spontaneous EMG and SSEP readings, is routinely used during elective spinal procedures, including lumbar microdiscectomies. Over a period of 3 years, among the 1072 patients undergoing this procedure, two experienced cauda equina syndrome after an uneventful operation. In these cases, both cortical (Cz, 10–20 system, reference to Fz) and subcortical (C-5 spine, reference to Fz) SSEPs were monitored on intermittent stimulation of the posterior tibial nerve at the ankle (~ 30 mA) through tab electrodes. For EMG studies, spontaneous activity was monitored by placing subdermal needle electrodes in the abductor, vastus, tibialis anterior, and gastrocnemius muscle groups bilaterally. All of these electrical connections were grounded at the patient’s right shoulder. Stimulation and data acquisition were achieved using Endeavor hardware and software (version 2.5, Nicolet Biomedical, Madison, WI). General, endotracheally administered anesthesia was maintained with sevoflurane and propofol infusion throughout the procedure, according to our institutional protocol. The medical records, neuroimaging studies, and the data from intraoperative monitoring of these two patients were thoroughly reviewed.

Abbreviations used in this paper: EMG = electromyographic; MR = magnetic resonance; SSEP = somatosensory evoked potential.
CASE REPORTS

Case 1

History and Examination. This 44-year-old man had a 2-year history of severe low-back pain radiating to his left lower extremity. The clinical examination findings were consistent with left L-3 radiculopathy. His lumbar spine MR images revealed a large herniated nucleus pulposus at the left L2-3 level.

Operation. The patient was admitted for a left L2–3 lumbar microdiscectomy and underwent an uneventful procedure. Intraoperatively, no significant EMG activity was observed. Nevertheless, the amplitude of cortical and subcortical SSEPs abruptly decreased after the start of the procedure, during the phase of discectomy (Fig. 1). During this episode, anesthesia depth and blood pressure were maintained at the baseline levels, and assessment of monitoring equipment and setting did not reveal a technical explanation for the SSEP changes. Although a partial improvement of SSEP amplitude was noticed 30 minutes later, it remained significantly lower compared with the baseline until the operation was completed.

Postoperative Course. Postoperatively the patient experienced full cauda equina syndrome below the L-4 level with loss of rectal tone. A lumbar spine MR image was obtained on an emergency basis and revealed only normal postoperative findings, with no evidence of neuronal tissue compression (Fig. 2). A spinal cord injury Solu-Medrol protocol was initiated. On the 3rd postoperative day the patient had partial improvement of motor strength, although his sensory function was still below the L-4 level bilaterally. He was transferred to a rehabilitation center and attained significant improvement of motor and sensory function within the next 3 postoperative months. At 12 months postoperatively the patient had regained full bladder and bowel control. No motor weakness was evident, but he reported some minor dysesthesia in the distribution of the L-5 and S-1 nerve roots on the left side. His dysesthesia remained essentially unchanged at his 15-month follow-up review, which was his last evaluation.

Case 2

History and Examination. This 47-year-old man had previously undergone a left L4–5 lumbar microdiscectomy. He was admitted for the same procedure on the right side (in

Fig. 1. Case 1. Intraoperative SSEP stacked traces obtained in a patient undergoing lumbar microdiscectomy. An abrupt, significant (> 50%) decrease in the amplitude of SSEPs was observed during discectomy (red arrow). Partial improvement before the completion of the operation was noted (green arrow).

Fig. 2. Case 1. Postoperative sagittal T2-weighted MR image demonstrating no compressive pathological entity in this patient.
L3–4) after presenting with back pain radiating to the right lower extremity and MR findings consistent with right L3–4 herniated nucleus pulposus.

**Operation.** The patient underwent an uneventful procedure, although intraoperative electrophysiological monitoring revealed transient EMG discharges in bilateral anterior tibialis without significant SSEP changes at that time. During foraminotomy, the amplitude of cortical and subcortical SSEPs decreased abruptly without improvement until completion of the operation (Fig. 3). Interestingly, the SSEP changes could not be attributed to blood pressure, anesthesia depth, or technical errors, and no intraoperative event could explain these changes.

**Postoperative Course.** Postoperatively the patient reported progressive numbness and loss of motor function of the lower extremities. He experienced full cauda equina syndrome within 1.5 hours after the procedure. A lumbar spine MR image was obtained, and it revealed only normal postoperative findings with no evidence of compression of the thecal sac (Fig. 4). A spinal cord injury Solu–Medrol protocol was administered. The patient also underwent a lumbar myelogram, with no compression findings that could explain his clinical condition. He attained minimal improvement of motor function of the lower extremities, and on the 7th postoperative day was transferred to a rehabilitation center, with significant improvement of his clinical condition in the next months. In the 3rd postoperative month the patient had regained dorsiflexion and plantar flexion of his right foot, with a score of 3/5, whereas on the left side his motor strength was 1–2/5. He still had decreased sensation in his lower extremity over the L-5 and S-1 dermatome distributions. He had regained full control of his urinary and bowel functions. At his 6-month follow-up evaluation, the patient’s motor weakness had further improved, with dorsiflexion and plantar flexion of both the right and left foot at 3/5. His sensory deficits had remained unchanged. The patient was lost to follow up after that point.
DISCUSSION

Cauda equina syndrome in patients undergoing lumbar discectomy has been described in the literature, with an incidence ranging from 0.08 to 1.2%. Nevertheless, the possibility of an early postoperative cauda equina syndrome, such as underlying spinal canal stenosis along with low-lying conus medullaris, malpositioning of free epidural fat graft, incarcerated incarcera-

tion of the cauda equina through small, initially unrecognized dural defects, the effect of anesthetic agents, epidural abscess, retained surgical sponge, placement of excessive amounts of hemostatic agents (Surgicel and Gelfoam), epidural hematoma, enterotoxin C, and sudden alteration of the vascular supply of the cauda equina.

The venous congestion theory has gained popularity among spinal surgeons because it is the only one that has some experimental support. In their porcine model, Olmarker and Rydevik demonstrated that venous stasis was responsible for the pronounced effect on nerve impulse propagation; this observed effect was more profound in a two-level compared with a single-level compression. According to this model, if only one level is compressed the venous blood has the potential to drain away either proximally or distally; this mechanism can explain the epidemiological observation that most of the postoperative cauda equina syndrome cases have been observed in levels other than the lumbosacral one, as in our two cases. Furthermore, in their cadaveric study Hoyland, et al., established a direct correlation between venous congestion and neural fibrosis in the lumbar intervertebral foramen. Additionally, in their articles Parke and colleagues discussed the vulnerability of the venous return phase to pathological conditions of the spine and how this could lead to ischemic neuropathy. The rapid development of cauda equina syndrome in our two cases could be explained by a venous stasis–induced ischemia of the conus medullaris.

In regard to the role of postoperative MR imaging or postmyelography CT scanning, their value remains controversial. In the majority of the reported cases, as in our two, the imaging studies obtained revealed no evidence of compression. Nevertheless, the possibility of an early postoperative compression of the thecal sac caused by an acute hematoma or other mechanism needs to be excluded. Especially in cases of late-onset postoperative cauda equina syndrome, the imaging studies are essential in revealing a compressive origin.

We believe that all surgeons would welcome an intraoperative monitoring tool with real-time feedback that would allow them possibly to predict such a dreadful complication as the cauda equina syndrome. This could ideally allow for further intraoperative maneuvers, such as extension of the decompressive laminectomy during the initial procedure. The electrophysiological monitoring, via EMG readings and SSEPs, could potentially offer this possibility to the operating surgeon.

In our cases SSEP amplitude showed significant alterations intraoperatively, with no obvious surgical or technical explanation. Although the possibility of an untoward outcome alarmed the operating surgeon, there was no intraoperative event that could explain these findings, and therefore there was no possible reversing maneuver that could be performed. Furthermore, the susceptibility of SSEPs to changes in the parameters such as alteration of blood pressure or depth of anesthesia, along with frequent false-positive findings and even more frequent artifacts, made the decision to extend the decompressive laminectomy difficult in our cases. Nevertheless, the role of SSEP and spontaneous EMG intraoperative monitoring needs to be defined in the future, and the predictive value of these modalities could potentially be assessed in a large prospective clinical study.

The appropriate management of patients who suffer postoperative cauda equina syndrome has remained very controversial. In cases in which an underlying compressive pathology can be demonstrated, surgical treatment of this entity and adequate decompression of the dural sac are mandatory. In the majority of the cases, though, no compression can be identified. In these cases, Henriques, et al., advocated early surgical intervention with wide decompression and fusion if necessary. Similarly, Kardaun, et al., favored a wide decompression for treating postoperative cauda equina syndrome. It needs to be emphasized, though, that in their study the presence of this entity was ambiguous because it could represent both a consequence of lumbar discectomy and an indication for it; their data analysis did not allow the resolution of this ambiguity.

In contrast, McLaren and Bailey stated that if compressive pathological findings cannot be demonstrated, surgical decompression is not indicated. Not surprisingly, they reported one case with no compressive pathological entity in which surgical decompression further degraded the patient’s neurological status. It appears that the scenario of further vascular compromise of a cauda equina that is already tenuous from a vascular standpoint could be quite possible in cases with no apparent compressive pathology.

Regardless of the treatment used, the functional outcome in patients experiencing postoperative cauda equina syndrome has remained poor. McLaren and Bailey reported full recovery in only two of their six patients, whereas two of the six had bladder and/or bowel incontinence. Likewise, Henriques, et al., reported full neurological recovery in two of five patients, whereas three of five with postoperative cauda equina syndrome were bladder and/or bowel incontinent. Finally, Spangfort reported in his series that only 40% of the patients in whom postoperative cauda equina syndrome developed had any significant recovery.

CONCLUSIONS

Cauda equina syndrome is a very rare but troublesome complication of uneventful lumbar microdiscectomy. The importance of maintaining a high level of suspicion when the postoperative surgical course deviates from the routine cannot be overemphasized in cases in which a compressive pathological entity is the underlying cause. The origin of this dreadful complication has remained unknown, however, in a significant number of cases in which the imaging studies obtained have revealed no abnormalities. In our study, SSEP changes predicted the development of cauda equina syndrome in both reported cases. Intraoperative SSEP monitoring could lend itself to the early detection of these impending complications. The positive predictive
value, specificity, accuracy, and cost/benefit ratio of this modality may merit further investigation with prospective clinical studies.

Acknowledgments

We thank Ms. Bridgett Furhmann, R.N., B.S.N., and Ms. Sadie B. Fennell for their valuable assistance in the preparation of this manuscript.

References


Manuscript received April 24, 2005. Accepted in final form June 6, 2005. Address reprint requests to: Kostas N. Fountas, M.D., Ph.D., 840 Fine Street, Suite 880, Macon, Georgia 31201. email: knfountasmd@excite.com.