Monitoring of the sciatic nerve during hamstring lengthening by evoked EMG

K. Katz, J. Attias, D. Weigl, A. Cizger, E. Bar-on
From Tel Aviv and Haifa Universities, Tel Aviv and Haifa, Israel

Traction injury to the sciatic nerve can occur during hamstring lengthening. The aim of this study was to monitor the influence of hamstring lengthening on conduction in the sciatic nerve using evoked electromyography (EMG).

Ten children with spastic cerebral palsy underwent bilateral distal hamstring lengthening. Before lengthening, the evoked potential was recorded with the patient prone. During lengthening, it was recorded with the knee flexed to 90˚, 60˚ and 30˚, and at the end of lengthening with the hip and knee extended.

In all patients, the amplitude of the evoked EMG gradually decreased with increasing lengthening. The mean decrease with the knee flexed to 60˚ was 34% (10 to 77), and to 30˚, 86% (52 to 98) compared with the pre-lengthening amplitude. On hip extension at the end of the lengthening procedure, the EMG returned to the pre-lengthening level.

Monitoring of the evoked EMG potential of the sciatic nerve during and after hamstring lengthening, may be helpful in preventing traction injury.

Shortening of the hamstring muscles can cause flexion contracture of the knee, crouch gait, high energy expenditure, and poor sitting posture in children with spastic cerebral palsy. Treatment by surgical elongation of the hamstrings may be indicated.1 In patients with shortening of the sciatic nerve secondary to a flexion contracture of the knee, hamstring lengthening may overstretch and damage the nerve.1,2 The amount of lengthening is usually based on clinical judgment. Some authors have suggested a pre-operative calculation to avoid damage to the sciatic nerve.2 Others have proposed elongating the hamstring up to an angle of flexion of the knee of 20˚.3 A decrease in nerve conduction can be measured as an indicator of possible nerve damage.4

The aim of our study was to determine the efficacy and feasibility of evoked electromyography (EMG) for monitoring the influence of hamstring lengthening on conduction in the sciatic nerve.

Patients and Methods

Five girls and five boys with spastic diplegia or total-body cerebral palsy underwent bilateral distal hamstring lengthening. The mean age at surgery was ten years (5 to 17). The indications for surgery were crouch gait or poor sitting posture due to short hamstrings.

The amount of shortening of the hamstrings was determined by measuring the angle of flexion of the knee.5 The mean angle before surgery was 70˚ (60 to 80).

At surgery the children were placed prone. The tendons of the hamstrings were exposed through a transverse incision just above the popliteal crease. The gracilis tendon was divided and the semitendinosus tendon was Z-lengthened. The semimembranosus and biceps muscles were elongated by an incision in the overlying aponeurosis. The patient was then placed supine, the hip and knee were flexed to 90˚, and then the knee was slowly extended up to an angle of flexion of 30˚, as recommended in previous studies (Fig. 1).1 The hip and knee were then extended. The knee was immobilised in plaster for three weeks in almost full extension, without applying any force. In this position the EMG amplitude returns to baseline. If any residual flexion contracture remained, no attempt was made to correct it by force.

The Explorer system (Biologic Corp, Mundelein, Illinois) was used for muscle stimulation and recording. To stimulate the nerve, two needle electrodes were placed near the peroneal nerve, and square impulses of amplitude from 0.18 to 1.2 mA were delivered for a duration of 100 µsec. Muscle responses were recorded using two needle electrodes inserted into the tibialis anterior muscle. The level of stimulation was fixed until a small twitch of the ankle was noted. The evoked EMG ampli-
tude was recorded before lengthening at the normal position of the hip and knee fully extended (baseline), during lengthening at angles of flexion of the knees of 90˚, 60˚ and 30˚ and again at the normal position of hip and knee fully extended (Fig. 2). The changes in EMG responses were measured and calculated relative to baseline.

Results
The changes in evoked EMG amplitude during hamstring lengthening relative to the pre-lengthening amplitude are shown in Table I. At an angle of flexion of the knee of 90˚, there was a mean increase of 10% in EMG amplitude from baseline. At an angle of 60˚, there was a mean decrease in the amplitude of 35% (10 to 77) on the left side and 32% (16 to 75) on the right. In most patients, there was a marked difference in the amount of decrease between the right and left sides (Table I). At an angle of 30˚, the mean decrease was 89% (70 to 98) on the left side and 83% (52 to 98) on the right.

In all patients, the evoked EMG amplitude returned to baseline on extension of the hip and knee to the normal position. None of the patients had signs of sciatic nerve injury at follow-up.

Discussion
Our study confirms previous reports that monitoring evoked EMG potentials enables serial real-time assessment of nerve conduction. A decrease in motor potential...
amplitude provides quantitative information about possible nerve injury. The cut-off decrease in motor potential amplitude, beyond which traction injury occurs in the sciatic nerve during slow hamstring elongation remains unknown. Our study shows that a gradual decrease of up to 98% for a few seconds does not cause nerve injury. In patients with short hamstrings and an angle of flexion of the knee of less than 80°, elongation of the hamstring up to an angle of 30° is apparently safe. In patients with short hamstrings and an angle of 90° or more, elongation of the hamstrings to within the normal range may be associated with sciatic nerve injury. In these patients we recommend that the hamstrings be elongated under EMG monitoring, up to a decrease of 98% in the motor potential amplitude.

Immobilation of the leg for a long time can overstretch the sciatic nerve and result in nerve damage. One experimental study found that even minimal traction for more than two minutes can cause sciatic nerve damage. To avoid this complication, we recommend immobilising the leg in the position at which the evoked EMG amplitude returned to the pre-lengthening amplitude. If any residual flexion contracture remains after hamstring lengthening, no attempt should be made to correct it by force.

In conclusion, monitoring the evoked EMG potential amplitude during and after hamstring lengthening may avoid sciatic nerve traction injury.

The authors thank Gloria Ginzach and Charlotte Sachs of the Editorial Board, Rabin Medical Center, Beilinson Campus, for their assistance.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References