Seventeen children, 6 girls and 11 boys, aged 5–17 years with pediatric median mononeuropathies (PMM) were identified among 1809 who had EMGs primarily in the electromyographic laboratory at The Children's Hospital, Boston, between 1979 and 1993. Electromyography documented the PMM to be at the wrist in 7 children, including 3 children with idiopathic carpal tunnel syndrome (CTS)—1 whose symptoms were accentuated by skiing—2 with a systemic illness (mucolipidosis III and scleroderma), and in 1 child each the distal PMM was secondary to a cast or laceration. A proximal PMM was identified in 10 children, including 8 with trauma, 1 with an osteoid osteoma, and 1 with juvenile cutaneous mucinosis. Five children (3 with CTS and 1 each with mucolipidosis III and juvenile cutaneous mucinosis) had bilateral disease. The localization (59% proximal) and cause of these PMMs differed greatly from our experience with adult median neuropathies. 
Key words: pediatric median mononeuropathies • carpal tunnel syndrome • electromyography • nerve conduction study

**MUSCLE & NERVE 17:755–762 1994**

# PEDIATRIC MEDIAN MONONEUROPATHIES: A CLINICAL AND ELECTROMYOGRAPHIC STUDY

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Mononeuropathies occur infrequently in children. At The Children's Hospital (CH), Boston, mononeuropathies were seen in only 78 (6%) of the 1319 children referred to our pediatric electromyography (EMG) laboratory within an 11-year period (1979–1990).[20] In the upper extremity, pediatric median mononeuropathy (PMM) has been about equally encountered as ulnar[14] and radial[12] mononeuropathies. Although large series of adult median mononeuropathies have been reported at the wrist[42,49] and proximal near the elbow,[17] no similar major review of PMM is found on MEDEX search of the literature for the past 25 years. To define the clinical characteristics, pathophysiology, and EMG findings of PMM, we analyzed our experience with 17 children documented to have PMM during a 14-year period. In contrast to adults, in whom carpal tunnel syndrome (CTS) is 100 times more common than proximal median neuropathies,[17] 59% of our PMMs were proximally situated.

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Presented in part at the 39th annual scientific meeting of the American Association of Electrodiagnostic Medicine, Charleston, South Carolina, October 15–16, 1992.

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Accepted for publication January 16, 1994.

CCC 0148-639X/94/070755-08


## MATERIALS AND METHODS

**Patients.** The clinical records of children with PMM diagnosed by EMG criteria[23] who were evaluated at the EMG laboratories of CH, 13 children, and at the Lahey Clinic, 4 children, from March 1979 to October 1993 were reviewed. Studies of the 4 children performed at the Lahey Clinic were also included because both laboratories are staffed by the same neurologic electromyographers. Data were collected prospectively since 1979 when the pediatric EMG laboratory was organized by one of us (H.R.J.).

**Clinical Assessment.** All children included in this study had been examined by staff neurologists, neurosurgeons, or orthopaedic surgeons before our EMG evaluation. The time interval between the onset of PMM and the EMG was recorded. The temporal profile was classified as acute, chronic, or indeterminate. The pattern of weakness, sensory loss, and pain was delineated. The causes, predisposing factors, and follow-up data were defined by clinical examination, review of charts, or telephone conversations, and occasionally repeat EMG.

**EMG Evaluation.** Nerve conduction studies (NCS) and needle electromyographic examination (NE) were performed in all children using the Teca TE42 EMG machine (Teca Corporation, Pleasantville, NY) or the Dantec Counterpoint (Dantec Electronic, Tonsbakken, Skovlunde, Denmark) with standard filter settings using recognized techniques.[23] In each child, the results of NCS of the affected median nerve were compared with studies of the ipsilateral ulnar nerve and, in 12 of 17 instances, the contralateral median nerve. In the remaining 5 children with unilateral fracture or laceration, contralateral NCS did not warrant the added discomfort. Our results were compared with normal values established for children[37] up to age 5 years. Because NCS values for children over age 5 are similar to adults,[21] standard adult values were used for children aged 6–17.[23] Concentric needles were used for NE to assess the presence of abnormal insertional activity, i.e., fibrillation potentials and positive waves, and motor unit action potential (MUAP) morphology and recruitment patterns in median and ulnar innervated muscles, and appropriate muscles to exclude other peripheral nerve, plexus, or nerve root lesions. The grading system used for NE was that of Lambert as reported by Daube.[11]

The electrophysiologic criteria for inclusion in this study were the same over the entire 14 years and included NCS and EMG abnormalities confined to the median nerve and the median innervated muscles. The only change in technique during the period of the study was the introduction of orthodromic palmar stimulation during the mid-1980s. The localization was considered to be at the wrist when results of NE showed abnormalities of abductor pollicis brevis (APB)/opponens pollicis muscles only; involving the anterior interosseous nerve (AIN) when abnormalities were confined to the flexor pollicis longus (FPL), flexor digitorum profundus II and III, or pronator quadratus muscles; and involving a more proximal location when pronator teres, flexor carpi radialis (FCR), or flexor digitorum sublimis muscles showed abnormalities. When results of NE were normal, isolated distal latency (DL) prolongation determined a presumed localization at the wrist.

Specific NCS/EMG criteria established to define the type of injury included focal demyelination when median sensory or motor DL or both were prolonged (>115% of normal), or a conduction block (CB) with greater than 30% change in amplitude without significant dispersion, and/or conduction slowing (CS) greater than 10 m/s above and below the site of stimulation. Demyelination was presumed when median SNAP was absent and/or motor CV was slowed with normal NE; axonal injury when NE of the muscles revealed evidence of active denervation (i.e., fibrillation potentials and positive waves) without conduction changes or chronic denervation based on the presence of high-amplitude long-duration rapidly firing motor unit potentials (MUPs); and mixed injuries when both focal demyelination and axonal injury were present. When CMAPs and SNAPs could not be obtained to permit the calculation of DL and CV, the lesion was classified as axonal when denervation was present. Concomitant demyelination cannot be excluded in this group.

## RESULTS

Thirteen PMMs were diagnosed among 1809 EMGs performed on children aged 0–17 years between March 1979 and October 1993 at CH, and an additional 4 children with PMM were seen at the Lahey Clinic. These 17 PMM are subdivided into nontraumatic or traumatic groups summarized in Tables 1 and 2.

**Nontraumatic.** *Clinical.* Seven children, aged 8–17 years, were evaluated for intermittent numbness characteristic of CTS, pain and weakness in median nerve distribution, and painless weakness and atrophy of the thenar eminence. Contributing factors included storage of an abnormal metabolite, i.e., mucolipidosis III (ML-III); or infiltration secondary to a systemic illness, i.e., scleroderma, and juvenile cutaneous mucinosis (JCM). Three children had bilateral symptoms. One of the 3 children with CTS had a strong family history of CTS.

Although 1 child (Table 1, case 7) had initial complaints of elbow pain and thenar atrophy, EMG elsewhere led to a diagnosis of CTS based on a "prolonged DL." Wrist surgery did not alleviate the problem, and this teenager was referred to us. At CH, median motor CB/CS was present at the elbow with NE changes confirming a proximal PMM. Computed tomography documented a lesion at the distal humerus. At surgery, an osteoid osteoma at the elbow was associated with an inflammatory reaction encasing the median nerve.

*NCS/NE.* Median CMAP was absent in 1, of low amplitude in 2, and normal in 4 children. Motor CVs were abnormal in 3 of the 7 children with nontraumatic PMM, including 1 child with CB/CS at the elbow. Prolonged motor DLs were noted in 4 children. Median SNAPs were absent in the 3 with an infiltrative systemic illness and prolonged

**Table 1.** Nontraumatic PMM.

| Age, sex | Pathogenesis | Weakness/ atrophy | Numbness/ pain | Course | EMG interval | Median motor NCS | | Median sensory | Needle EMG | | | Site and type of injury | Outcome (follow-up period) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | CMAP | CV/DL | SNAP/DL | APB | FDP/FPL PQ | PT FCR | | |
| Case 1<br>14, male | Entrapment; CTS skiing | 0 | + | Chronic | 6 mo | R NI<br>L NI | NI/NI<br>NI/5.1 | 3.9 (p)<br>2.7 (p) | ND<br>−1 | ND<br>NI | ND<br>NI | Wrist, D | Improved after ski season (9 mo) |
| Case 2<br>14, female | Entrapment; CTS idiopathic | 0 | + | Chronic | 9 mo | R NI<br>L NI | NI/4.8<br>NI/4.2 | 2.5 (p)<br>2.5 (p) | −1<br>ND | NI<br>ND | NI<br>ND | Wrist, D | Unimproved; SD at wrist pending |
| Case 3<br>17, female | Entrapment; CTS familial | 0 | + | Chronic | 6 mo | R NI<br>L NI | NI/NI<br>NI/NI | 2.6 (p)<br>3.8 (p) | ND<br>−1, A | ND<br>NI | ND<br>NI | Wrist, A and D | Unimproved; SD at wrist pending |
| Case 4<br>8, male | Entrapment; mucolipidosis III | + | 0 | Chronic | years | R NR<br>L ↓ 1.5 | NR<br>NR/15.4 | NR (d)<br>10.3 (d) | −4<br>−3 | ND<br>ND | ND<br>ND | Wrist, D | Improved after CTS/SD (3 yr) |
| Case 5<br>15, female | Entrapment; scleroderma | + | 0 | Chronic | 3 wk | R ↓ 1.5<br>L NI | 46/NI<br>NI/NI | NR (d) | NI<br>ND | NI<br>ND | NI<br>ND | Wrist, D | Improved spontaneously; L radial palsy at 1.5 yr |
| Case 6<br>17, female | Entrapment; JCM | 0 | + | Acute | 1 mo | R NI<br>L NI | NR/4.8<br>NI/4.1 | NR (p)<br>NR (p) | −1, A<br>ND | −1, A<br>ND | NI<br>ND | Proximal, A and D | Spontaneous improvement (4 mo) |
| Case 7<br>14, male | Entrapment; osteoid osteoma | + | + | Chronic | 1 yr | R ↓ 0.3<br>L ND | CB/CS elbow<br>ND | 4.8 (d) | ND | −1, A | −1, A | Proximal, A and D | Lost to follow-up |

*Median SNAP normal values: palmar NI < 2.3 ms at 35°C; digital NI < 3.1 ms at 35°C.*
*A, axonal; APB, abductor pollicis brevis; CMAP, compound muscle action potential (mV); CB, conduction block; CTS, carpal tunnel syndrome; CS, conduction slowing; CV, conduction velocity; D, demyelinating; d, digital; DL, distal latency (ms); FCR, flexor carpi radialis; FPL, flexor pollicis longus; FPS, flexor pollicis sublimis; JCM, juvenile cutaneous mucinosis; L, left; NA, not available; NCS, nerve conduction study; ND, not done; NI, normal; NR, no response; P, proximal; p, palmar; PQ, pronator quadratus; PT, pronator teres; R, right; SD, surgical decompression; SNAP, sensory nerve action potential (μV); ↑, increased; ↓, decreased.*

**Table 2.** Traumatic PMM

| Age, sex | Pathogenesis | Weakness/ atrophy | Numbness/ pain | Course | EMG interval | Median motor NCS | | Median sensory | Needle EMG | | | Site and type of injury | Outcome (follow-up period) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | CMAP | CV/DL | SNAP/DL | APB | FDP/FPL PQ | PT FCR | | |
| Case 1<br>7, female | Compression, cast; forearm | + | + | Acute<br><br>Acute | 4 Mo<br>12 Mo | (1) ↓ 0.1 mV<br>(2) NI | NR/NI<br>↓/NI | NR (d)<br>2.9 (p) | −2<br>ND | OK<br>ND | OK<br>ND | Wrist, A and D | Complete recovery (2 yr) |
| Case 2<br>14, male | Compression; hockey | + | 0 | Acute | 2.5 Mo | NI APB (↓ FPL) 2 mV vs. 56 mV | NI/NI | NI (p)<br>NI | −1 | −4, A | OK | Elbow (AIN), A | Complete recovery (2 yr) |
| Case 3<br>13, male | Entrapment; elbow dislocated | + | + | Acute | 3 Mo | NI | 46/NI | NR (d, p) | −3, A | −4, A | −2, A | Elbow, A | Improving after SD (<6 mo) |
| Case 4<br>12, male | Entrapment; SCFx with osteomyelitis | + | + | ? | 9 Yr | ↓ 0.7 mV | 28, CB/NI | NR (d 1, 2)<br>3.8 (d 3) | −2 | −2, ± | −3, ± | Elbow, A and D | Not much improvement despite SD (11 mo) |
| Case 5<br>7, female | Entrapment; SCFx | + | + | Acute | 4 Mo | NI | NI/NI | NR | −2, A | −2, A | −1, A | Elbow, A | Improved after SD (5 mo) |
| Case 6<br>10, male | Entrapment; midradius Fx | + | + | Acute | 6 Mo | NR | NR | NR | −3, A | ND | −1 | Forearm, A (D?) | Improved after SD (6 mo) |
| Case 7<br>5, male | Indeterminate; SCFx | + | + | Acute | 7 Mo | ↓ 0.7 mV | NR/NI | NR (d) | −2, A | ?, A | −2, A | Elbow, A | Not much improvement (1.5 yr) |
| Case 8<br>5, male | SCFx; brachial artery laceration | + | 0 | Acute | 3 Mo<br>5 Mo | NR | NR | NR (d) | −3, A<br>−2, A | <br>OK | −3, A<br>−2, A | Elbow, A | Improved (3 yr) |
| Case 9<br>14, male | Laceration | + | + | Acute | 1 Yr | ↓ 0.5 mV | NI/NI | NR (d) | −2 | OK | OK | Wrist, ? A | Marginal improvement (1 yr) |
| Case 10<br>5, male | Laceration; indeterminate | + | + | Acute | 6 Mo | NI | NI/NI | 2.5 (d) | NI | −3, A | −3, A | Axilla, A proximal median | Surgical exploration (−); improved (11 mo) |

*Median SNAP normal values: palmar NI < 2.3 ms at 35°C; digital NI < 3.1 ms at 35°C.*
*A, axonal; AIN, anterior interosseous nerve; APB, abductor pollicis brevis; CMAP, compound muscle action potential (mV); CB, conduction block; CV, conduction velocity in meters/second; D, demyelinating; d, digital; DL, distal latency (ms); FCR, flexor carpi radialis; FDP, flexor digitorum profundus; FPL, flexor pollicis longus, Fx, fracture; NA, not available; NCS, nerve conduction study; ND, not done; NI, normal; NR, no response; P, proximal; p, palmar; PQ, pronator quadratus; PT, pronator teres, SC, supracondylar, SD, surgical decompression; SNAP, sensory nerve action potential (μV). ↑, increased; ↓, decreased.*

in the other 4 children. Evidence of median axonal injury was demonstrated in the 2 children with proximal lesions. Of 5 children with nontraumatic PMM at the wrist, only 2 had NE changes.

*Prognosis.* Symptoms improved in 4 children with atraumatic PMM in whom we have follow-up data. One of 3 teenagers with bilateral CTS improved concomitant with the end of the ski season; however, 2 others have persistent symptoms at 6–12 months and are currently being considered for surgery. The patient with ML-III improved after bilateral median nerve decompression at the wrist. The children with scleroderma and JCM spontaneously improved, but a radial palsy developed 16 months later in the child with scleroderma. The child with osteoid osteoma did not return.

**Traumatic.** *Clinical.* Eight of the 10 patients with traumatic PMM were boys. Five PMM occurred secondary to an elbow injury (fracture in 4 and dislocation in 1) and 2 to more distal fractures. (Entrapment at the site of injury was defined in 4, compression secondary to a cast in 1, and no precise mechanism in the other 2.) An adolescent hockey player hit his upper arm multiple times against the boards while playing. Transient pain developed around his elbow, and he experienced weakness of pinch the next day. This PMM cleared but recurred a few weeks later. A laceration was responsible in 2 other children.

*NCS/NE.* Median CMAP was absent in 2, of low amplitude in 4, and normal in 4 children. Motor CV could not be calculated in 4 and was decreased in 2 of the other 6 children. A CB was found in 1 child 9 years after an unsuccessful decompression of a fracture with subsequent osteomyelitis. The DL was normal in each child with a recordable CMAP. SNAPs were absent in 8 children but preserved in the 2 with predominant AIN damage, although 1 was low amplitude.

*NE.* Evidence of definite median axonal injury was demonstrated in 7 children. The other 3 children had at least a 50% decrease in the number of motor units.

*Prognosis.* There are too few traumatic PMMs to formulate specific prognostic conclusions. Complete recovery occurred in the 2 children with PMM secondary to nerve compression. Entrapment of the median nerve was documented at surgical exploration and decompression in 3 children with fractures and in 1 child with dislocation. Three of these 4 children had surgery within 6 months of injury; each improved initially, although 1 has severe cold-sensitive causalgia in the median innervated digits 6 months after operation. The child who underwent decompression at 11 months had no noticeable improvement; however, his supracondylar fracture (SCFx) was complicated by osteomyelitis. Another child with a SCFx was not operated on and had no improvement at 1.5 years. Another child with a SCFx had concomitant laceration of the brachial artery requiring immediate surgical intervention. Preoperatively, median nerve function was intact, but a postoperative PMM occurred possibly secondary to retractor injury, and recovery was incomplete at 3 years. Of the 2 PMM cases associated with laceration, the child with the wrist lesion had no improvement, whereas the child with an axillary laceration had an intact median nerve at exploration, and the PMM gradually improved.

An abnormal CMAP was associated with a variable prognosis. It should be emphasized that 7 of 10 EMG studies were performed 2.5–6 months after injury, 2 between 7 and 12 months, and 1 at 9 years after trauma. The 2 children with absent responses had incomplete improvement. Of the 4 children with CMAPs between 0.1 and 0.7 mV, 3 had marginal improvement at 1–9 years. The remaining child with an initial CMAP of 0.1 mV had complete improvement; however, her PMM was primarily a demyelinating lesion secondary to compression. All 3 children with PMM evaluated after surgical decompression and who had normal CMAPs were improving. The child with AIN compression and normal CMAP had complete return of function. Fibrillation potentials were a nonspecific finding vis-à-vis prognosis.

## DISCUSSION

Although CTS is common among adults, it is rare in children. Of our 7 PMM located at the wrist, 5 were of nontraumatic origin, and 3 of them had typical CTS symptoms. Some PMMs with CTS have been associated with sports or strenuous use of the hand,[47] including 3 adolescents whose symptoms were exacerbated by basketball, weight lifting, and golf.[16] Each was operated on, with resolution of symptoms. In contrast, our teenager with CTS related to skiing improved spontaneously after the ski season ended.

Most children with idiopathic or familial CTS have had bilateral pain and numbness. The presence of a concomitant foreshortening of the index finger suggested a congenital or chronic process in 1 child.[28] Transverse carpal ligament thickening was observed at surgical decompression in most patients with familial disease.[10,13,25,27,32–34,43,46]

CTS is associated with MPS-I H,[18,30,38] MPS-I S,[9,38] MPS-II,[44] MPS-IV,[9] ML-II,[18,30] and ML-III.[30,36,48] We have noted that some of these children, including our child with ML-III, have not had typical CTS symptoms but rather an insidiously progressive weakness or clumsiness of the hand with bilateral thenar atrophy.[9,38,48] This clinical picture may be the first indication of occult MPS/ML.[9,48]

Although CTS is a common cause of thenar atrophy in the adult, in children, a smaller than normal thenar eminence may be secondary to congenital thenar hypoplasia.[7] We have seen 1 such adolescent not reported in this study. NCS demonstrated a low-amplitude CMAP but normal CV and DL. No insertional activity was demonstrated in the APB, and no motor units were recruited. In contrast, the opponens pollicis had normal MUPs. Roentgenographic studies here demonstrate concomitant malformation and hypodevelopment of the adjacent carpal bones and phalanges of the thumb.[7]

Congenital constriction bands may cause PMM entrapment both at proximal and distal sites, sometimes with concomitant radial and ulnar entrapment.[31,53,54] Rarely, CTS may occur in children with trigger fingers.[33] Other nontraumatic causes for PMM have included unusual nerve tumors.[1–3,29,41,43] PMM was associated with scleroderma once in our experience but has not been described previously in children to our knowledge. Although a few adults[4,5,26,45,50] with scleroderma have had CTS, our 15-year-old girl had hand pain and trouble writing but did not have typical CTS symptoms. Asymptomatic PMM may be defined at the wrist in childhood Schwartz–Jampel syndrome.[8]

Proximal median neuropathies are unusual in adults[17]; however, in this study these were more common than distal PMM. Nontraumatic proximal PMM has rarely been reported to be associated with calcification of the flexor digitorum superficialis muscle,[19] with the ligament of Struthers,[6] a congenital supracondylar process,[39] and congenital constriction bands.[53] One of our 2 children with a proximal nontraumatic PMM had pain and diffuse median distribution weakness secondary to an osteoid osteoma of the distal humerus producing CB/CS at the elbow. We are unaware of previous reports of juvenile cutaneous mucinosis associated with proximal PMM. Our 17-year-old girl presented with evolving hand pain and numbness of the median fingers. She had firm nontender subcutaneous nodules over the elbows, forearms, fingers, and the bridge of the nose. The PMM spontaneously resolved in 2 months.

Traumatic median neuropathies in childhood have most frequently been associated with SCFx of the humerus[22,35,52] and less commonly with forearm fractures[55] or rarely with entrapment within the FPL tendon in a fracture of the radioulnar joint.[51] Seven of our 10 traumatic PMMs were associated with fractures; however, the mechanisms for the nerve injury were variable, including immediate or delayed entrapment; concomitant processes, such as osteomyelitis; and iatrogenic causes, such as nerve retraction while making an acute repair to a damaged artery or compression from a cast. Elbow dislocation may also cause PMM similar to a SCFx. Although children with PMM secondary to SCFx or dislocation may recover with conservative therapy,[35] surgical decompression needs to be considered early on to exclude entrapment within the fracture site.[15,52] Surgery was helpful in each of our 3 children with PMM who were operated on within less than 6 months of SCFx. This contrasts with a poor outcome in 2 PMM: 1 explored at 11 months, and in 1 child not operated on. One unusual mechanism for traumatic PMM in this series was recurrent proximal median neuropathy subsequent to blunt elbow trauma in a teenage hockey player. Brachial[40] and radial[24] arterial gas sampling has rarely been associated with newborn or infantile traumatic PMM.

EMG in children with PMM is helpful in localizing the site of the lesion, especially when surgical intervention is considered. The importance of complete distal and proximal NCS with careful NE in PMM is illustrated by our child with osteoid osteoma (Table 1, case 7), which is detailed in the Results section of this report. Although the pathophysiologic characteristics of our PMMs were well defined by EMG, prognosis was not as well defined. Children with normal CMAPs had a good prognosis; however, the converse did not apply. Children with PMM with absent or low-amplitude CMAPs had variable recoveries. These differences may relate to multiple factors, including the age of the child, the shorter distance a child's nerve needs to grow for muscle reinnervation, and neuronotrophic factors in children may be more effective. Such hypotheses await better definition. This study suggests that early surgical decompression may be indicated in traumatic PMM with potential for nerve entrapment. As electromyographers gain more experience with childhood mononeuropathies, careful prospective analysis may provide more definitive prognostic information.

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