even cause the take-up area to be larger in infants than in adults[11] (Blok and Stegeman, personal communication), increasing the number of visible MUAPs even further. The amplitude of the muscle fiber potentials will be smaller, due to their smaller diameters, but we doubt that this seriously affects the judgment of the recruitment pattern. If this hypothesis is correct, electromyographers have to recalibrate their sense for the relation between strength, recruitment pattern, and degree of nerve fiber loss in infants, requiring the study of normal muscles.

Second, central nervous system (CNS) plasticity is involved in regaining function after nerve repair in adults,[8] which may also be true in infants. Infant movements differ from adult ones.[4] Severe nerve damage might disrupt the normal maturation of CNS motor programs, leading to MUAPs that are not embedded in functional movement programs, rendering their action unnoticeable clinically. Motor unit estimation provided evidence for this view, by revealing a normal number of motor units in the biceps of a 3-year-old with OBPL despite abnormal use of the arm.[9] The abnormal use was thus in part due to a CNS cause and not to nerve damage alone.

Further studies must determine to what degree the clinical/EMG discrepancy in OBPL indeed derives from a methodological overestimation of the number of functional motor units by the electromyographer, and its functional underestimation by the clinician.

J. Gert van Dijk, MD, PhD
Department of Neurology and Clinical Neurophysiology
Leiden University Medical Centre
P.O. Box 9600
2300 RC Leiden, The Netherlands

Martijn J.A. Malessy, MD
Department of Neurosurgery
Leiden University Medical Centre
Leiden, The Netherlands

Dick F. Stegeman, PhD
Department of Clinical Neurophysiology
Nijmegen University Hospital
Nijmegen, The Netherlands

1. Bodensteiner JB, Rich KM, Landau WM: Early infantile surgery for birth-related brachial plexus injuries: justification requires a prospective controlled study. *J Child Neurol* 1994;9: 109–110.
2. Brooke MH, Engel WK: The histographic analysis of human muscle biopsies with regard to fiber types. 1. Adult male and female. *Neurology* 1969;19:221–233.
3. Brooke MH, Engel WK: The histographic analysis of human muscle biopsies with regard to fiber types. 4. Children's biopsies. *Neurology* 1969;19:591–605.
4. Hadders-Algra M, Nakae Y, Van Eykern LA, Klip-Van Den Nieuwendijk AWJ, Prechtl HFR: The effect of behavioural state on general movements in healthy full-term newborns. A polymyographic study. *Early Hum Dev* 1993;35:63–79.
5. Jahnke AH, Bovill DF, McCarroll HR, James P, Ashley RK: Persistent brachial plexus birth injuries. *J Pediatr Orthop* 1991; 11:533–537.
6. Laurent JP, Lee R, Shenaq S, Parke JT, Solis IS, Kowalik L: Neurosurgical correction of upper brachial plexus birth injuries. *J Neurosurg* 1993;79:197–203.
7. Laurent JP, Lee RT: Birth-related upper brachial plexus injuries in infants: operative and nonoperative approaches. *J Child Neurol* 1994;9:111–117.
8. Malessy MJA, Van Dijk JG, Thomeer RTWM: Respiration-related activity in the biceps brachii muscle after intercostal-musculocutaneous nerve transfer. *Clin Neurol Neurosurg* 1993;95(suppl):S95–S102.
9. McComas AJ, Pape K, Kirsch S: Apraxia in congenital brachial palsy. *Can J Neurol Sci* 1993;20:362.
10. Piatt JH: Neurosurgical management of birth injuries of the brachial plexus. *Neurosurg Clin North Am* 1991;2:175–185.
11. Roeleveld K, Stegeman DF, Vingerhoets HM, van Oosterom A: Single motor-unit contribution to the surface electromyogram. *Acta Physiol Scand* (in press).

NEUROMYOPATHY SECONDARY TO OMEPRAZOLE TREATMENT

We report a case of subacute neuromyopathy secondary to Omeprazole treatment. A 48-year-old man was admitted in July, 1995 because of walking disability. His medical history revealed repeated courses of Omeprazole treatment for duodenal ulcer from 1990 to 1995. The patient was neither alcoholic nor diabetic, taking no other medication. In 1992, while treated with Omeprazole, he had episodic paresthesia and mild weakness in the lower limbs. These symptoms rapidly resolved after discontinuation of the drug. He had recurrence of the same symptoms associated with myalgia in 1993, and again in 1994, when treated with Omeprazole. In February 1995, he was started on Omeprazole 20 mg/day. In May, the patient began to suffer from lower limb paresthesia at night. The dosage of Omeprazole was increased to 40 mg in June. The patient developed weakness in the lower limbs and walking disability a few days later.

Clinical examination on admission showed a waddling gait, weakness of the pelvic and peroneal muscles, and absent stretch reflexes. Sensation to vibration and pinprick were both altered distally in a stocking distribution. There was no upper limb involvement.

Normal laboratory studies included cell count and protein level of cerebrospinal fluid, erythrocyte sedimentation rate, glycemia, serum creatinin, blood cell count, and lederfoline and cyanocobalamine levels. Antinuclear antibodies, anti-DNA antibodies, rheumatoid factor, anti-Sjögren's syndrome antigen A and B antibodies, antineutrophil cytoplasmic antibodies, and immune circulating complexes were negative. A search for porphyria was negative. Creatine kinase (CK) was increased to 776 IU (upper normal limit: 180 IU).

Electrophysiological studies demonstrated an axonal sensory–motor neuropathy (Table 1). A biopsy of the superficial peroneal nerve confirmed acute axonal degeneration, which involved 40% of myelinated fibers by testing. A biopsy of the peroneus brevis demonstrated type II fiber atrophy.

Clinical and electrophysiological improvement was evident a few months after cessation of Omeprazole. In November 1995, the patient no longer had paresthesia. He had normal strength and stretch reflexes. Electrophysiological studies demonstrated marked improvement from previous abnormalities (Table 1). CK returned to normal.

Table 1. Electrophysiological data.

Nerve	July 1995 Amplitude*	July 1995 CV (m/s)	November 1995 Amplitude*	November 1995 CV (m/s)
R superfical peroneal	1.7	56	3.6	48
R sural	3.5	50	4.5	46
L superfical peroneal	3.7	44	nr	nr
L sural	1.8	48	2.8	50
R peroneal	0.3	35	2.3	39
R tibial	0.7	46	3.9	46
L peroneal	1.6	39	1.8	40
L tibial	0.8	41	1.8	45

nr, not recorded; cv, conduction velocity.
**Microvolts for sensory and millivolts for motor potentials.*

Only two previous reports have related neuromuscular disorders on Omeprazole treatment. Sellapah[2] described a 73-year-old woman who had lower limb paresthesia, diminished pinprick sensation, and absent Achilles reflexes after a 3-month course of Omeprazole. She recovered within 10 days after the discontinuation of treatment. No electrophysiological data were reported.

Garrot et al.[1] also reported a 78-year-old woman who developed proximal weakness with reduced reflexes 14 days after initiation of Omeprazole treatment. CK was increased. A biopsy of quadriceps demonstrated type II fiber atrophy. Her weakness resolved after Omerpazole was discontinued only to recur after its reinstitution.

In our case, a causal link is suggested by the close temporal relationship between clinical symptoms and repeated Omeprazole courses. The frequency of neuromyopathy in patients treated with Omeprazole is probably very low because the drug is widely prescribed. Nonetheless, clinicians should be attentive to symptoms of neuromyopathy in this setting, as illustrated by our case.

J.M. Faucheux
P. Tournebize
A. Viguier
M.C. Arne-Bes
V. Larrue
G. Géraud
Service de Neurologie
Hôpital de Rangueil 31403
Toulouse cedex 4, France

1. Garrot FJ, Lacambrac D, Del Sert T, Garcia B, Obeso G, Solis J: Subacute myopathy during Omeprazole therapy. *Lancet* 1994;340:672.
2. Sellapah S: An unusual side effect of Omeprazole: a case report. *BJ Gen Pract* 1990;389.

CONDUCTION VELOCITY IS INVERSELY RELATED TO AXONAL LENGTH IN THE MEDIAN SENSORY NERVE

Investigators have demonstrated that motor conduction velocities (CVs) to the proximal muscles are faster than to the distal muscles.[3,6,12] Zwarts and Guechev found that the axonal length was inversely related to motor CV.[13] Sensory CV also has an inverse relation to body height,[1,5,8–11] and is slower in the leg than in the arm.[4,7] We conduct this study to test the hypothesis that sensory CV is inversely related to axonal length.

Thirty healthy volunteers (15 male, 15 female; mean age 35, range 22–54 years) were examined. The median nerve length was measured from 3 cm proximal to the distal wrist crease to the middle of the wrist, then to the tip of the thumb and the middle finger. Nerve conduction studies were performed on 60 healthy subjects (24 male, 36 female; mean age 35, range 24–54 years). The median sensory nerve action potentials (SNAPs) were obtained in the right arm of all subjects by stimulating at 3 cm above the proximal wrist crease and at the antecubital fossa. Ring electrodes were placed on the thumb and the middle finger for recording. The SNAPs were obtained with wrist and elbow stimulation and the CV across the forearm was calculated using the difference of onset latencies.

The results showed that the distance from the wrist to the tip of the middle finger (21.6 ± 1.3 cm) was always greater than the distance from the wrist to the tip of the thumb (17.0 ± 1.0 cm), and the difference was significant ($P < 0.001$). The mean forearm CV for the median sensory axons innervating the middle finger (60.0 ± 3.9 m/s) was slower than the CV for the median sensory axons innervating the thumb (61.4 ± 4.1 m/s, $P = 0.0012$).

The median sensory axons innervating the thumb and the middle finger arise primarily from the C6 and C7 dorsal root ganglion, respectively.[2] The distances from the C6 and C7 dorsal root ganglion to the point where the median sensory fibers unite in the lateral cord are approximately equal.[13] The median sensory fascicles to the thumb and middle finger run the same course until emerging from the carpal tunnel. The distance from the wrist to the tip of the middle finger was always greater than the distance to the tip of the thumb as demonstrated. As a result, the length of the C7 fascicles to the middle finger is longer than the C6 fascicles to the thumb. This study has shown that the median sensory CV is faster in the shorter fibers innervating the thumb than in the longer fibers to the middle finger, and it confirms that the inverse relation of CV and axonal length reported in motor axons also applies to the sensory nerves. Potential factors influencing CV such as age, height, temperature, and the nerve or segment of nerve tested are not relevant variables in this study, since the CVs were compared in different fascicles of the median nerve across the same forearm segment in the same subject.

Peter B.J. Wu, MD, MPH
John Neff, MD
Wade S. Kingery, MD
Elaine S. Date, MD
Division of Physical Medicine and Rehabilitation
Department of Functional Restoration
Stanford University Medical Center
Stanford, CA 94305, USA

Physical Medicine & Rehabilitation Service
VA Palo Alto Health Care System
Palo Alto, CA, USA