rable to that obtained using the microneurographic technique.[4,6,9] Being less affected by the conduction time of the afferent somatosensory fiber and by central processing, SuCV may be a useful parameter with which to evaluate efferent sudomotor function. Denišlič et al., however, noted that it is not based on the activity of the same unmyelinated axons and sweat glands.[1] Elie et al., who tried to determine SuCV in the lower extremities of normal subjects, failed to obtain SSRs at the proximal site (inner side of the thigh) in 13 of 30 subjects.[4] Further study is needed to determine the meaning of the decrease in SuCV.

The SSR amplitude varies greatly even in normal subjects; therefore, some authors have suggested that amplitude is an unreliable parameter for SSR,[3,9] but others have reported the SSR amplitude is important clinically.[8,10] We agree with the opinion expressed by Denišlič et al. that the presence or absence of SSR is only one aspect of the overall considerations. The problem of variability may be countered, to some extent, by using an amplitude parameter with reproducibility. Although all SSR amplitude parameters in our study showed considerable inter- and intrasubject variability, the maximum amplitude gave the best results in terms of reproducibility (Fig. 1). High intersubject variability, shown as a large standard deviation (SD) value or coefficient of variation across subjects (CVAS), makes it difficult to set a normative range of SSR amplitudes. For example, we found that the mean maximum amplitude (n = 35) minus 2 SD gives a value below 0 mV. We think a definition of normative range based on percentile would be practical. The log transformation also is used to achieve normality.[5]

Latency showed less variation than the SSR amplitude. In our study the mean value (n = 35) of the mean latencies of 20 SSRs in each subject was 7.1% of the CVAS, which corresponds to the value for minimum F latency (6.1%, median nerve and 7.0%, tibial nerve)[7] obtained for 45 normal subjects. In terms of the other reported SSR latency CVAS (4.7% to 12.9%),[4,6,8,9] our 7.1% is an intermediate value. If the normative range is defined as the mean ± 2.5 SD (range 1.16–1.66 s), only one measurement in the 82 total test sessions, including the follow-up examination (n = 35), would be beyond this range.

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VARIATION IN THE TREATMENT OF CARPAL TUNNEL SYNDROME

Carpal tunnel syndrome (CTS) is a common disorder, especially among female adults.[1] No consensus exists with respect to the treatment of choice of CTS. Some physicians prefer early surgery in electrophysiologically confirmed CTS cases without an underlying reversible disorder,[4] but others refer to the potential complications of surgery and support conservative therapy as the first treatment option of CTS.[2]

In order to assess the current practice of neurologists in The Netherlands regarding the treatment of CTS, a questionnaire was sent to each of 134 Dutch neurological practices. The questionnaire included queries regarding the preferred initial treatment, the annual frequency of the responders' application of various treatment modalities (never versus ever), and the responders' opinion of the acceptability of early surgery as the first treatment option of CTS.

The questionnaire was returned by 92 (69%) representatives of the 134 neurological practices. Early surgery was indicated as the preferred initial treatment of a CTS patient by 36 neurologists (39%), whereas 37 neurologists (40%) preferred conservative treatment. The remaining 19 responders (21%) reported either surgery or conservative therapy as the preferred initial treatment (Table 1). Regarding the annual frequency of the responders' application of various treatment modalities (data not shown), wrist splinting (76%) and referral for surgery (88%) were the most frequently mentioned CTS treatments. Local corticosteroid injections, systemic corticosteroid therapy, analgesics, physiotherapy, and pyridoxine were mentioned by 20 (22%), 2 (2%), 21 (23%), 6 (7%), and 1 (1%) responders, respectively. For only 4 neurologists (4%), early surgery was not acceptable.

We conclude that both in the medical literature[2,4] and

Table 1. Preferred initial treatment of patients with carpal tunnel syndrome according to 92 representatives of Dutch neurologic practices.

Treatment modality		No. of responders (%)
Surgery		36 (39.1%)
Conservative management		37 (40.2%)
Wrist splinting	24 (26.1%)	
Corticosteroid injection into the carpal canal	4 (4.3%)	
Splinting and/or corticosteroid injection	3 (3.3%)	
Splinting and/or analgesics	3 (3.3%)	
Splinting and/or systemic corticosteroids	1 (1.1%)	
Systemic corticosteroids	1 (1.1%)	
Pyridoxine (vitamin B6)	1 (1.1%)	
Either surgery or conservative management		19 (20.7%)
Surgery or wrist splinting	13 (14.1%)	
Surgery or another conservative treatment	6 (6.5%)	

Percentage of total between brackets.

in a relatively small country like The Netherlands there is no consensus regarding the optimal treatment of CTS. This might be due to the lack of scientific evidence of the efficacy of the various treatment modalities for CTS.[3] To date, the medical literature does not support the selection of either early surgery or conservative management as the treatment of choice for CTS. Conservative management seems to be attractive because it is easily applicable and not very demanding. However, recovery might occur more quickly after early surgery, resulting in a shorter duration of the disease.

Therefore, properly designed randomized clinical trials comparing the efficacy of early surgery with conservative management (e.g., immobilization of the wrist by splinting) with a sufficient number of patients and relevant outcome measures are urgently needed to enable the selection of the best treatment option for patients with carpal tunnel syndrome.

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SELECTIVE BILATERAL AMYOTROPHY OF THE ANTERIOR TIBIAL MUSCLE: A CASE REPORT

Numerous instances of unilateral muscular atrophy involving only a single muscle in either the upper or lower limb have been reported in Japan,[6–9] and less frequently in the western countries.[1,2,4,5] Most of these conditions involve muscle wasting of neurogenic origin. Here we describe a male patient, aged 22, with a selective amyotrophy affecting only the anterior tibial muscle in both legs.

The patient was in good health until age 19, when he started experiencing difficulties in flexing his feet. The patient was brought to our attention at age 21. Examination showed marked bilateral wasting of the tibialis anterior muscle. Dorsiflexion of the feet was not possible. Muscle strength was normal in all other muscles examined. Electromyogram (EMG) of the anterior tibial muscle showed bilateral changes with positive potentials and fibrillations. During maximal voluntary effort, only a single motor unit discharge per insertion could be recorded. F-wave latency recorded from extensor digitorum brevis by stimulating the deep peroneal nerve was normal. All other muscles were normal by EMG. A muscle computed tomography (CT) scan showed hypodensity of the anterior tibial muscles (Fig. 1B). By magnetic resonance imaging (MRI), the spinal cord was shown to be of normal diameter throughout. The muscle CT scan was normal for both parents. A skeletal muscle biopsy was obtained from the right tibialis anterior muscle. Light microscopy showed a markedly increased variation in fiber size. Interstitial connective tissue was increased (Fig. 1A). Some atrophic muscle fibers showed clusters of nuclei. Adenosine triphosphatase reaction was uniform under all conditions. We believe that this case can be classified with previously reported instances of selective muscle amyotrophy. To date no isolated cases in which only the anterior tibial muscle was affected in both limbs have been described. Similar cases previously described all significantly differ from the present case. Most of the similar cases reported in the literature are concerned with monomelic amyotrophy.[1,2,4–9] Some authors have described segmental atrophy of the spinal cord using myelography and postmyelographic CT.[10] We performed an MRI of the spinal cord in our patient, but could not demonstrate a segmental spinal cord atrophy. A clinical case similar to our patient's, but involving the bilateral quadriceps femoris, was reported in 2 patients by Furukawa et al.[3] and Serratrice et al.[12] A familial form of symmetrical anterior tibial muscular dystrophy has been described in Finland by Udd et al.[13] The lack of dystrophic change and the normal muscle CT scan in the parents of