Sensory Nerve Conduction in Chronic Uremic Patients During the First Six Months of Hemodialysis

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ABSTRACT: In an attempt to find the best electrophysiological indicator of improvement for the neuropathy present in patients with chronic renal failure undergoing hemodialysis, several types of nerve conduction were studied at the beginning of dialysis and six months later. Sural nerve conduction and late response latencies were recorded in addition to conventional motor and sensory nerve conductions. After six months of hemodialysis, sensory nerve conduction velocities in the median, ulnar and sural nerves were improved. These values appear to be the most sensitive indices of the beneficial effect of hemodialysis on the neuropathy.

RÉSUMÉ: Nous avons essayé de déterminer le meilleur critère électromyographique d'amélioration de la polynévrite observée chez les malades insuffisants rénaux chroniques hémodialysés. Plusieurs types de vitesses de conduction ont été évalués incluant les plus conventionnelles, motrices et sensitives, les réponses tardives et la vitesse de conduction du nerf sural. Après six mois d'hémodialyse, les vitesses de conduction sensitives, médiane, cubitale et surale s'améliorent et semblent être le paramêtre le plus sensible pour juger de l'effet positif de l'hémodialyse sur la polynévrite.

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The peripheral neuropathy associated with chronic renal failure has been well described both clinically and electromyographically (Nielsen, 1971, 1973). Treatment of chronic renal failure with hemodialysis may be accompanied by slight improvement, stabilization, or deterioration of the neuropathy (Nielsen, 1974; Bolton, 1975, 1977). Marked improvement of the neuropathy has been reported following renal transplantation (Bolton, 1976). For many patients, however, long term hemodialysis remains the only form of treatment and better methods to evaluate the effect of hemodialysis on the neuropathy are required. Conventional studies of nerve conduction velocity have been of limited value in assessing the effects of hemodialysis on the peripheral neuropathy (Nielsen, 1974; Bolton, 1975; Dyck, 1979).

In an attempt to find an electrophysiological index of response to treatment, conduction studies were done in a group of patients with chronic renal failure at the beginning and after six months of hemodialysis. In addition to conventional motor and sensory nerve conduction studies we recorded sural nerve conduction velocities and latencies of the F wave.

MATERIAL AND METHODS

Fifteen patients with chronic hemodialysis form the basis of this report. Their ages ranged from 22 to 68 (mean age = 44). Patients with neuropathy due to other causes such as diabetes, alcoholism or toxic substances were excluded from the study.

Nerve conduction studies were done at the beginning of hemodialysis and then repeated six months later. The patients were on a two or three times a week dialysis schedule for three to six hours at a time. Dialysis was performed using the Polyacrylonitrile Membrane (RP - 610 Hospal).

Six of the fifteen patients had signs or symptoms suggesting a mild peripheral neuropathy. None had weakness. The other patients had a normal examination.

Nerve conduction studies were done on a TECA TE-4 system using surface recording electrodes. Motor conduction velocities were determined for the median, ulnar, peroneal and tibial nerves. Orthodromic sensory potentials were obtained from median and ulnar nerves at the wrist after stimulation of the index and little finger respectively with ring electrodes. The

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sural nerve potential was elicited by stimulation of the nerve behind the lateral malleolus. The response was recorded 14 cm proximally in the distal third of the leg lateral to the achilles tendon. With supramaximal stimulation of median, ulnar, peroneal and tibial nerves distally, an "F" wave was elicited in the abductor pollicis brevis, abductor digiti quinti, extensor digitorum brevis and abductor hallucis muscle, respectively. Ten responses were recorded and the shortest latency was measured. Needle electromyography was done on selected muscles in the upper and lower extremity. Skin temperature was recorded by a surface thermistor and maintained between 32 and 34 °C if needed. The results obtained in our patients were compared to the values from twenty normal subjects of the same age group. Statistical analysis were performed using student t-test for non-paired data.

RESULTS

The mean values for motor conduction velocity and amplitude of motor evoked responses for each nerve tested (median, ulnar, peroneal and tibial) were significantly lower in the uremic patients compared to normal controls both at the initiation of hemodialysis and on repeat testing six months later (tables 1 and 2). Furthermore, no difference was seen between the values measured in the first and the second study in the uremic population.

Table 1: Motor conduction velocities in 20 normal subjects and 15 uremic patients

| Groups | Conduction velocity (metres/sec.) | | | |
|-----------------|-----------------------------------|----------------|----------------|----------------|
| | Median | Ulnar | Peroneal | Tibial |
| Normal | *61.8 ± 4.9 | 62.6 ± 4.4 | 52.2 ± 5.7 | 49.7 ± 5.4 |
| Uremic patients | 53.1 ± 7.7 | 54.7 ± 7.2 | 44.1 ± 5.1 | 40.5 ± 3.6 |
| (1st study) | p < .0005 | .0025 | .0005 | .0005 |
| Uremic patients | 55.2 ± 4.9 | 53.3 ± 6.8 | 41.7 ± 3.6 | 40.1 ± 3.1 |
| (2nd study) | p < .0005 | .0005 | .0005 | .0005 |

^{*}mean ± S.D.

Statistical analysis for non-paired (P) data (normal vs. uremic group).

Table 2: Amplitude of motor evoked responses in millivolts

| | | NERVE | | |
|--------------------------------|-----------------------|------------------|-----------------|-----------------------|
| Groups | Median | Ulnar | Peroneal | Tibial |
| Normal | 12.1 ± 3 | 12.9 ± 3.1 | 8.2 ± 2.7 | 18 ± 6.4 |
| Uremic patients (1st study) | 11.5 ± 5 p NS | 11.5 ± 4.7 NS | 6.5 ± 4.5 NS | 13 ± 8.2 < .05 |
| Uremic patients (2nd study) | 9.8 ± 4.4 p < .05 | 11.3 ± 4.1 NS | 8.2 ± 3.7 NS | 10.2 ± 4.4 < .0005 |

The mean values for sensory terminal latencies in the median and ulnar nerve were significantly prolonged in uremic patients for both studies (table 3). Sural nerve conduction velocities were also significantly slower than normal (table 3). However, there was a tendency for each value in repeat study six months later to improve. Amplitude of sensory potentials were significantly lower than normal but there was no improvement (table 4).

Table 3: Sensory conduction studies

| | NERVE Median Ulnar | | Sural (Conduction Velocity m/s) | |
|-----------------------------|-----------------------------|------------------------|---------------------------------------|--|
| Groups | (latency in | | | |
| Normal | $2.38 \pm .32$ | 1.96 ± .27 | 54.8 ± 5.9 | |
| Uremic patients (1st study) | $2.89 \pm .32$ p < .0025 | $2.59 \pm .43$ $.0005$ | 42.0 ± 12.9 .0025 | |
| Uremic patients (2nd study) | $2.72 \pm .53$ p < .025 | 2.34 ± .38 .0025 | 44.5 ± 12.8 .005 | |

Table 4: Amplitude of sensory potentials in microvolts

| Groups | NERVE | | | |
|-----------------------------|-----------------------------|------------------------|-----------------------|--|
| | Median | Ulnar | Sural | |
| Normal | $*16.7 \pm 6.3$ | 17.6 ± 6.9 | 12 ± 4.7 | |
| Uremic patients (1st study) | 9.1 ± 4.5 p < .0005 | 9.8 ± 6.5 $.0025$ | 7.9 ± 5.4 $.0125$ | |
| Uremic patients (2nd study) | 10.5 ± 4.6 p < .0025 | 9.1 ± 4.8 .0005 | 8.4 ± 3.7 $.0125$ | |

^{*} mean ± S.D.

Table 5: Latencies of F responses (in milliseconds)

| Groups | NERVE | | | |
|--------------------------------|----------------------------|-----------------------|------------------------|------------------------|
| | Median | Ulnar | Peroneal | Tibial |
| Normal | *25.9 ± 1.4 | 26.6 ± 1.8 | 48.0 ± 3.6 | 49.6 ± 4.3 |
| Uremic patients (1st study) | 29.9 ± 2.2 p < .001 | 29.5 ± 2.8 $.001$ | 51.9 ± 5.2 .005 | 54.6 ± 4.3 .005 |
| Uremic patients (2nd study) | 29.6 ± 2.4 p < .001 | 29.6 ± 2.5 .001 | 52.2 ± 5.8 .005 | 52.7 ± 5.8 $.005$ |

^{*} mean ± S.D.

F wave latencies were increased in the renal patients at the time of both studies. No significant changes occurred in the values between the first and second studies.

Needle electromyography in these patients showed slightly reduced interference pattern in foot muscles in three patients without evidence of active denervation.

DISCUSSION

Several previous studies on chronic renal failure patients on hemodialysis have revealed slowing of nerve conduction velocity, confirming the presence of either clinical or subclinical peripheral neuropathy (Nielsen, 1973, 1974; Bolton, 1975, 1977; Ackil, 1981; Lowitzsch, 1981). In most of these studies only a limited number of nerve were examined. However Ackil (1981) included sural nerve conduction and late response latencies in his assessment and demonstrated that these techniques provided a greater diagnostic yield than the conventional motor and sensory conduction studies alone. In our study, the same techniques were used and the tests were repeated 6 months after the onset of hemodialysis. Serial evaluations have been done previously on

hemodialyzed patients (Nielsen, 1974; Bolton, 1975, 1977). In these reports conduction studies were obtained for motor and sensory median and motor peroneal nerve. In the abstract by Bolton (1975), the sural nerve is mentioned, but no change in nerve conduction velocities occurred during hemodialysis. Since late responses and sural conduction have proven to be more sensitive parameters in uremic neuropathy (Ackil, 1981) as well as in other types of neuropathies (Lefebvre-D'Amour, 1979; Lachman, 1975), they were used in a longitudinal analysis in an attempt to find the best electrophysiological indicator of improvement of the uremic neuropathy.

The data from the present study show that sensory nerve conduction velocities are the best electrophysiological indicator of improvement for the mild or subclinical neuropathy found in uremic patients on hemodialysis in the first six months of treatment. Values for motor conduction which were significantly different from normal at the beginning did not change during dialysis. Late response latencies were abnormal and remained unchanged after six months.

The pathological changes in uremic neuropathy include axonal and demyelinating lesions affecting distal fibers initially, followed by comparable changes at more proximal levels (Dyck, 1971). In other types of metabolic neuropathies such as those due to alcohol and diabetes, there is also predominant involvement of sensory fibers (Lefebvre-D'Amour, 1979; Buchthal, 1970). Distal segments are frequently the most abnormal (Casey and LeQuesne, 1972). In hemodialyzed uremic patients it is therefore understandable that sensory nerves should be particularly influenced by the pathological process and its treatment.

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