SENSORY NERVE CONDUCTION STUDIES IN URAEMIC PATIENTS

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A neurological examination of a group of patients with chronic renal failure showed that
the clinical picture of peripheral neuropathy was dominated by sensory symptoms and signs.
Although these were often slight and mainly confined to the lower extremities, they might
represent a generalized affection of the peripheral afferent nerve system in uraemic patients.

To elucidate this problem the conduction of sensory action potentials was examined in the
median nerve in patients with and without clinical evidence of neuropathy.

CLINICAL INCIDENCE OF NEUROPATHY

The incidence of clinical symptoms and signs of neuropathy was evaluated in 51 patients with
chronic renal failure. Ten other patients were excluded, in whom the neuropathy might have
another aetiology. The material comprised 21 females and 30 males between the ages of 16
and 73 years. Ten patients were above the age of 50. Twenty-four hours endogenous creatinine
clearance averaged 9.2 ml per minute, ranging from 0 to 32. In 34 patients clearance was
below 10.

Signs were mainly confined to the lower extremity (Table 1), the distal part of which was
usually the site of the earliest affection and most prominent abnormalities. Impairment of
vibration sense and/or loss of deep tendon reflexes were the earliest, most frequent, and most
constant findings. The predominant complaint was paraesthesia.

Criteria of neuropathy are still a matter of dispute. In this study the criteria were impairment
of vibration sense or loss of deep tendon reflexes bilaterally in patients below the age of 50.
In those above this age both signs should be present bilaterally, or one of the signs should
be supplemented by another unequivocal sign of affection of the peripheral nerve system.
Symptoms without signs were regarded as inadequate for the diagnosis.

Nineteen patients presented neither symptoms nor signs, four had sensory complaints only,
and signs were considered of doubtful significance in another four patients. Twenty-four
patients (47%) presented evidence of neuropathy. No difference between sexes could be
detected with respect to incidence and severity of symptoms and signs. Due to a skewness
of distribution in the material nothing but a tendency towards increasing incidence with
decreasing renal function could be deduced at the present stage of the investigation.

SENSORY NERVE CONDUCTION STUDIES

Sensory nerve conduction velocity in the median nerve was measured in 22 patients. Renal
function was severely reduced in all cases as evidenced by 24-hours creatinine clearance
ranging from 0 to 11 ml per minute (average 4.6 ml per min.). Ten patients presented no
clinical manifestations of peripheral neuropathy, while 12 patients met the above mentioned
criteria. The only evidence of affection of the median nerve was slight impairment of vibration
sense of the thumb in one patient and intermittent paraesthesia in the fingers in three patients.
TABLE I
Clinical neurological findings in 51 patients with chronic renal failure

<table>
<thead>
<tr>
<th>Symptoms and signs of peripheral neuropathy</th>
<th>Lower limb (No. of cases)</th>
<th>Upper limb (No. of cases)</th>
<th>Total No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paraesthesia</td>
<td>15</td>
<td>11</td>
<td>18</td>
<td>35</td>
</tr>
<tr>
<td>Numbness</td>
<td>9</td>
<td>0</td>
<td>9</td>
<td>18</td>
</tr>
<tr>
<td>Pain</td>
<td>6</td>
<td>1</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Burning feet syndrome</td>
<td>9</td>
<td>—</td>
<td>9</td>
<td>18</td>
</tr>
<tr>
<td>Weakness</td>
<td>6</td>
<td>3</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Total no. of patients with symptoms</td>
<td>19</td>
<td>11</td>
<td>19</td>
<td>37</td>
</tr>
<tr>
<td>Signs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaesthesia/hypaesthesia</td>
<td>9</td>
<td>2</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Analgesia/hypalgesia</td>
<td>5</td>
<td>3</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>Vibration sense impaired or absent</td>
<td>20</td>
<td>1</td>
<td>20</td>
<td>40</td>
</tr>
<tr>
<td>Muscle tenderness</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Reflexes markedly diminished or absent</td>
<td>16</td>
<td>3</td>
<td>16</td>
<td>31</td>
</tr>
<tr>
<td>Loss of muscle power</td>
<td>6</td>
<td>4</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>Muscle atrophy</td>
<td>7</td>
<td>4</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>Total no. of patients with signs</td>
<td>27</td>
<td>7</td>
<td>28</td>
<td>55</td>
</tr>
</tbody>
</table>

Method

The technique was described in detail by Buchthal and Rosenfalck (1966).

The sensory fibers in the first and third finger were stimulated separately through ring electrodes. The stimulus was supramaximal (25 mA to 70 mA) corresponding to 7 to 27 times sensory threshold. Sensory action potentials were picked up at wrist, elbow, and axilla through needle electrodes placed close to the nerve (Fig. 1). The temperature was measured

![Diagram](image)

Fig. 1. Sensory action potentials (median nerve) recorded at wrist, elbow, and axilla after stimulation of finger I in a normal person and two uraemic patients without and with clinical evidence of neuropathy.

Figures on the potentials give conduction velocities (metres per sec.) in the distal, intermediate and proximal segment of the nerve.

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by a thermo-needle near the nerve and was kept at about 35° at all three sites of recording. Distances between electrodes were measured to an accuracy of 2 mm.

The latency of the recorded potential was measured in msec. from the stimulus artefact to the first positive peak in the potential. The amplitudes were measured peak to peak in microvolts. In potentials recorded at wrist with high sweep velocity (0.25 m sec. per mm) the duration of the potential was measured in m sec. from the first positive deflection to the intersection of the descending phase with the base line.

Normal values were obtained from the examination of 58 healthy persons between the ages of 19 and 65 years (Buchthal and Rosenfalek, 1966 and Nielsen, unpublished data). Since sensory conduction velocity in the median nerve decreases with age, each value in uraemics was compared to the estimated normal value as obtained from the regression equation between age and conduction velocity in the normal material. A Students t-test was applied on the average age-adjusted differences.

Results

The average sensory nerve conduction velocity was decreased in all segments of the nerve in patients with and without neuropathy (Figs. 2 and 3).

In patients with neuropathy the average age-adjusted deviation from the normal material was highly significant in distal as well as proximal segments of the nerve (P < 0.001). The slowing was significantly more pronounced in patients below the age of 35, than in patients above this age (P < 0.05).

Conduction velocity was significantly reduced too in all segments in young patients without clinical evidence of neuropathy (P < 0.05). In patients above the age of 35, however, only conduction velocities between wrist and elbow showed a significant reduction. The difference between the two age groups was insignificant.

Fig. 2. Sensory nerve conduction velocity (median nerve) in uraemic patients plotted against age. X = Patients without clinical evidence of neuropathy. ○ = Patients with neuropathy.

The central and outer lines indicate the regression ± two standard deviations of conduction velocity on age in the normal material.

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Fig. 3. Sensory nerve conduction velocity (median nerve) in uraemic patients plotted against age.
(Ref. Fig. 2).

Slowing of sensory conduction was generally more pronounced in patients with clinical signs of neuropathy, but the difference between patients with and without neuropathy was only significant below the age of 35 (P < 0.05).

Fig. 4. Duration in msec. of action potentials recorded at wrist.

\[
\begin{array}{ll}
\triangle & \text{Normal persons.} \\
\bullet & \text{Uraemic patients with neuropathy.} \\
\times & \text{Uraemic patients without neuropathy.} \\
\end{array}
\]

The height of the columns indicates mean values.
By measuring the latencies to the first positive peak in the potential, the study of conduction velocity is confined to the fastest fibres in the nerve.

An evaluation of slower conducting fibres is possible by measurement of the duration of the recorded potential. Only potentials recorded at the wrist are suitable for this study.

Action potentials were recorded with high sweep velocity after stimulation of finger I and III separately. The statistical analysis showed a good correlation between the two series of potentials in both groups of patients (Fig. 4). The increase of potential duration was significant in patients with neuropathy as well as patients without ($P < 0.001$ and $P < 0.05$ respectively). The difference between the two groups of patients was insignificant.

The increased potential duration represents the most prominent variation from the normal smooth, narrow, and triphasic potential (Fig. 5). A tendency towards rounding off the spikes and a poorly developed third phase seen in some potentials are probably inherent in the increased duration. Nearly all potentials retained the smooth well-synchronized shape.

All conditions being equal the amplitude of the action potential is a function of the number of activated fibres. If slowing of conduction velocity were mainly due to a loss of

![Diagram of sensory action potentials recorded at wrist. Figures on the potentials give conduction velocities (metres per sec.) between finger and wrist.](image)

Fig. 5. Sensory action potentials recorded at wrist. Figures on the potentials give conduction velocities (metres per sec.) between finger and wrist.
nerve fibres, the amplitudes might be expected to be considerably decreased in the most affected patients. However, no clear-cut tendency was ascertained, and comparison is further invalidated by a considerable variation in the normal material.

**Conclusion**

In the majority of uraemic patients the clinical picture of peripheral neuropathy is predominantly that of a sensory neuropathy.

Even in cases of protracted and severe neuropathy it was usually not possible to detect sensory defects in the upper extremity clinically. On this background the electrophysiological examination of the median nerve has yielded valuable supplementary information, which may be summarized in the following conclusions:

I. Statistical analysis of observations obtained in a group of uraemic patients yielded evidence of an affection of the sensory nerve conduction, regardless of the presence of clinical signs of neuropathy elsewhere.

II. Slowing of sensory nerve conduction was most pronounced in patients with clinical signs of neuropathy and most evident in patients below the age of 35.

III. Slowing of sensory nerve conduction was present in distal as well as proximal segments of the nerve in patients with and without clinical neuropathy. This is incompatible with the assumption of a purely distal affection of the nerve, even in the early stage of the disorder.

IV. The results of conduction velocity corresponded well to those of potential duration and leads to the conclusion that the pathophysiological changes involve fast as well as slow conducting fibres.

V. The uniform slowing of conduction velocity and the relatively well preserved potential amplitudes indicate a disturbance of the conduction mechanism rather than a loss of nerve fibres as the dominant pathogenic factor.

**REFERENCE**