Sensory and motor residual latency measurements in healthy patients and patients with neuropathy—Part 1

PAUL E. KAPLAN

From the Rehabilitation Institute of Chicago, Chicago, Illinois, U.S.A.

SYNOPSIS Median and ulnar nerves were evaluated using established nerve conduction techniques in a control population and in a population of patients with neuropathy. Motor and sensory residual latencies were obtained. The main contributor to the latencies was slowing of the nerve conduction in the smaller attenuated nerve fibres of the hand and fingers.

In 1948 Hodes et al. referred to a ‘latency of response’ while doing nerve conduction measurements across the human wrist. They noted that the observed conduction time of the ulnar nerve across the wrist was longer than the expected time based upon the application of the obtained nerve conduction velocity to the distal distance involved. This gap between the calculated and observed terminal conduction latencies was called a ‘residual latency’. From 1964 to 1966, important research was done that had the effect of standardizing motor and sensory nerve conduction velocity techniques (Trojaborg, 1964; Buchthal and Rosenfalck, 1966; Melvin et al., 1966). The original residual latency measurement was obtained using only motor conduction velocity techniques. The purpose of this paper is to apply sensory nerve conduction velocity techniques to the residual latency measurement.

METHODS

Twenty patients with an age range of 21 to 71 years, and a mean age of 45 years, were chosen for the control group. None of the control group patients had an abnormal glucose tolerance test; and none of the control group patients had a history of alcoholism or diabetes, or a family history of diabetes. They were compared with a group of 20 randomly selected patients with neuropathies. The age range of the group of patients with neuropathies was 28 to 73 years, and the mean age was 50 years. These 20 patients with neuropathies of the median and ulnar nerves were selected completely at random from a much larger population of patients with neuropathies. In all 40 subjects, the temperature of the forearm was determined using a skin surface thermistor thermometer technique, using the (Rochester Electro-Medical, Inc.) thermistor with surface probe. All forearm temperatures equalled or exceeded 34°C.

Sensory and motor conduction velocities in the median and ulnar nerves were determined using a standardized technique (Melvin et al., 1966). Residual latency was determined using techniques previously described by Mavor et al. (1962). The residual latency for sensory nerve fibres was determined as follows. The sensory nerve conduction velocity was calculated from the nerve using stimulating and recording sites at above the elbow and at the wrist respectively. From this elbow–wrist conduction velocity an expected terminal latency from wrist to finger was calculated and compared with the measured sensory terminal latency. The difference between these values was termed the residual latency for the sensory fibres.

RESULTS

Control and neuropathic groups are compared in the Table. There was no significant difference between the control and neuropathic groups (median and ulnar) as far as sensory and motor nerve conduction velocities from above-elbow to wrist positions were concerned. However, the ranges and standard deviations of the neuropathy group of nerve conduction velocity measurements were increased over those of the control population. There was a significant prolongation in the neuropathic group compared

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with the control group of all terminal and residual latencies for both sensory and motor conduction. With regard to the control population, the standard deviation (SD) of the residual latency measurements compared favourably with the standard deviation for the terminal latencies for both sensory and motor conduction, neither SD exceeding 0.6 ms. The values for the residual latency measurements in the control population in the sensory conduction were virtually identical with the residual latency measurements of the motor conduction in the same nerve, and also from nerve to nerve.

**DISCUSSION**

In 1948 Hodes et al. wrote that the residual latency seemed to be due to two factors: (1) the slowing of the conduction velocity in the smaller terminal fibres of the nerve, and (2) neuromuscular transmission delay. In 1966 Buchthal and Rosenfalck observed the slowing in the sensory conduction velocity distal to the wrist, and discussed the possibilities and probabilities of the slowing being due to either a gradual tapering of the sensory fibres or to a ‘distal delay’. The difference between the two possibilities was evidently whether the tapering occurred in the palm of the hand or in the digits. In the study of Lamontagne and Buchthal (1970) on diabetic neuropathy, residual latencies were not measured. On the other hand, Carpendale (1956) did measure residual latencies in normal subjects and patients with neuropathies and found abnormal residual latencies in neuropathies. Norris et al. (1953) noted that age change did not change the value of the residual latency by itself. Norris et al. (1953), Carpendale (1956), and Thomas and Lambert (1960) quoted the original thoughts of Hodes et al. (1948) on the aetiology of the residual latency as being a mixture of the effects of the nerve tapering and of the neuromuscular delay. Loewenstein (1961) has described the anatomy of the sensory nerve ending in detail. It is evident from his description that there is no equivalent of a neuromuscular junction, but that the nerve ending itself has adapted to its function. The values given here for residual latency measurements compare favourably with those given by Mavor and Libman (1962) in their control population. They did not document residual latency values for their

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**TABLE**

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Nerve conduction velocity above elbow to wrist</th>
<th>Terminal latency</th>
<th>Residual latency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control (m/s)</td>
<td>Neuropathy (m/s)</td>
<td>Control (ms)</td>
</tr>
<tr>
<td>Ulnar</td>
<td>Control (m/s)</td>
<td>Neuropathy (m/s)</td>
<td>Control (ms)</td>
</tr>
<tr>
<td>Sensory</td>
<td>55-61</td>
<td>36-64</td>
<td>3.2-3.6</td>
</tr>
<tr>
<td></td>
<td>59 (10)</td>
<td>51 (12)</td>
<td>±0.3</td>
</tr>
<tr>
<td></td>
<td>(P &gt; 0.20)</td>
<td>(P &gt; 0.20)</td>
<td>(P &lt; 0.01)</td>
</tr>
<tr>
<td>Motor</td>
<td>Motor</td>
<td>39-61</td>
<td>3.0-3.7</td>
</tr>
<tr>
<td></td>
<td>60 (12)</td>
<td>51 (12)</td>
<td>±0.6</td>
</tr>
<tr>
<td></td>
<td>(P &gt; 0.20)</td>
<td>(P &gt; 0.20)</td>
<td>(P &lt; 0.01)</td>
</tr>
<tr>
<td>Median</td>
<td>Median</td>
<td>39-60</td>
<td>3.2-3.6</td>
</tr>
<tr>
<td>Sensory</td>
<td>60 (15)</td>
<td>54 (11)</td>
<td>±0.3</td>
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<td></td>
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<td>(P &gt; 0.20)</td>
<td>(P &lt; 0.01)</td>
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<tr>
<td>Motor</td>
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<td>30-60</td>
<td>3.2-3.8</td>
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<tr>
<td></td>
<td>58 (12)</td>
<td>52 (12)</td>
<td>±0.4</td>
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<td>(P &gt; 0.20)</td>
<td>(P &gt; 0.20)</td>
<td>(P &lt; 0.01)</td>
</tr>
</tbody>
</table>

*Key:*
B. Age (yr). Control: range 21-71, mean 45; Neuropathy: range 27-73, mean 50.
C. All forearm temperatures equalled or exceeded 34°C.
patients with neuropathy. Mavor and Libman mentioned neuromuscular delay as being a factor, but thought that slowing of the nerve conduction due to temperature changes or decrease of fibre diameter probably also contributed to the residual latency. They did not study sensory nerve conduction velocities.

Temperatures were all standardized in the present study. Residual latency values for a normal nerve were virtually identical whether sensory or motor techniques were used. These results would be consistent with the theory that the residual latency was entirely due to tapering of the nerve distal to the wrist. The results of this study also indicate that residual latency measurements were as effective and as accurate as terminal latency measurements in determining the presence of a neuropathy distal to the wrist. Residual latency techniques would seem to be an accurate way of measuring the state of the delicately tapering nerve endings distal to the wrist.

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REFERENCES


Sensory and motor residual latency measurements in healthy patients and patients with neuropathy-part 1.

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