F-wave and Cervical Somatosensory Response
Conduction from the Seventh Cervical Spinous Process to Cortex in Multiple Sclerosis

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SUMMARY: Transit (conduction) times from the wrist to the seventh cervical spinous process (C7) were measured by using the F-wave response (TA) and the cervical somatosensory response (SEPC). The mean values obtained in 25 controls were 10.1 ± 0.9 s⁻¹ and 10.9 ± 1.0 s⁻¹ respectively. The 0.8 ± 1.0 s⁻¹ difference between the two methods represents central delay of SEPC, possibly at the level of the dorsal root ganglion. The mean onset latency of the cortical somatosensory response (SEPA) evoked by median nerve stimulation in the same subjects was 15.3 ± 1.0 s⁻¹. Transit time from C7 to the cortex (TB) was given by either SEPA - TA or SEPA - SEPC - 0.8, where 0.8 is the necessary corrective factor for central delay of SEPC. This technique was applied to 10 patients suspected of having multiple sclerosis, but without clinical evidence of involvement above C7. Six of the 10 cases showed prolongation of TB. In 4 of these, this could only be calculated by the F-wave method since SEPC was absent. It is concluded that transit times derived from either the F-wave or SEPC are equally valid and interchangeable. The absence of one response allows for its replacement by the other.

INTRODUCTION
In a previous study from this laboratory (Eisen and Nudleman, In Press), a technique was used to measure indirectly spinal cord conduction employing F-wave and somatosensory responses. It was shown that spinal cord transit time and conduction velocity were slowed in a significant number of patients with confirmed multiple sclerosis. The method, in essence, depends upon the difference between the transit time from the wrist to the cortex recorded from the cortically evoked somatosensory response, and the transit time from the wrist to the spinal cord using the F-wave technique (Kimura, 1974). The same principles are then applied to the leg. By subtracting the cervical cord to cortex transit time from the lumbar cord to cortex transit time, it is possible to arrive at a transit time from L1 to C7 (Dorfman, 1977).

The technique, although useful, had a number of limitations. In particular, it was assumed that the velocity through the peripheral nervous system measured through the F-wave (motor fibers) could be equated with velocity through the central nervous system recorded via somatosensory evoked responses (sensory fibers). In addition, seven independent measurements are required to calculate cord conduction velocity. Accurate onset latency of the somatosensory responses is crucial to the final calculation. Although this was not a serious problem when measuring the response evoked by stimulating the median nerve at the wrist, the response recorded from stimulating the peroneal nerve (or posterior tibial nerve) at the knee is such that its onset is not as easily definable (Tsumoto et al., 1972).

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The present study was undertaken to answer the following questions: 1. Is it valid to equate conduction time and velocity from the wrist to the C7 spinous process using the F-wave method with values over exactly the same segment of nerve obtained by recording the cervical somatosensory response? 2. Is there a difference in the transmission time between C7 and the contralateral cortex, which can be derived by subtracting either of the above from the onset latency of the cortically evoked somatosensory response? 3. Would this measurement be of value in indicating disease above the level of C7?

METHODS

Electrophysiological studies were conducted on 25 healthy volunteers of both sexes aged between 15 and 71 years (mean 37.85 years). The experiments were performed with the subject lying comfortably supine, in a semidarkened air conditioned room, in which the ambient temperature was maintained at between 20 and 22°C. Subjects were encouraged to relax without becoming drowsy or sleepless.

Stimulation:

The median nerve was stimulated at the wrist using surface electrodes. For M and F-wave recordings, the stimulus intensity was supramaximal, usually being about 10 to 15 mA. Stimuli were delivered at a rate of 1 Hz, with a duration of 0.2 s. When recording the somatosensory responses and mixed nerve action potential, the stimulus intensity was reduced enough to be without discomfort, but still sufficient to produce a clearly visible twitch of the thenar muscle group.

Recording:

M and F-wave responses were recorded from the thenar muscle using gold disc surface electrodes. The active electrode was placed over the muscle belly and the indifferent electrode over its tendon. The latency of the F-wave response was taken from the wave form having the shortest latency out of 4 to 8 trials. In the majority of cases, no difficulty was encountered in eliciting F-waves. However, when necessary, responses could be augmented by slight voluntary contraction of the muscle under study, and/or by making a fist with the contralateral hand (Eisen et al., 1977 a and b).

The latency of the mixed nerve action potential recorded from Erb's point was measured as the initial negative deflection of this response, which was recorded using surface disc electrodes. The active electrode was placed just behind the posterior border of the sternocleidomastoid muscle and 2 cm. above the clavicle. The indifferent electrode was positioned on the sterno-manubrial joint.

Cervical somatosensory evoked responses were recorded by surface electrodes placed just rostral to the C7 spinous process (active electrode) and on the mid-forehead (indifferent electrode).

The cortical somatosensory evoked response was recorded through a surface electrode positioned over C3 or C4 (international 10-20 system) contralateral to the side of stimulation. The differential electrode was placed on the forehead 2-3 cm. above the one employed for recording the cervical somatosensory response.

The cortical somatosensory response was recorded utilizing differential amplifiers having a flat frequency of 0.2 Hz to 500 Hz, this bandwidth being broadened to between 0.2 Hz and 2000 Hz when recording the cervical somatosensory response and mixed nerve action potential recorded at Erb's point. Each response was averaged 128 times. This procedure was repeated two or three times to insure accuracy of the time locked response.

RESULTS

The mean transit (conduction) times and mean conduction velocities from the wrist to the C7 spinous process, calculated from the F-wave, versus the cervical somatosensory response (SEPC) are compared in Table 1. The transit time computed from SEPC was 0.77 ± 1.0 s-3 longer and the velocity was 4.6 ± 6.2 m·1 slower than those computed from the F-wave response.

The mean conduction velocity from the wrist to Erb's point, when recording the mixed nerve action potential was 69.7 ± 6.1 ms-1, a value not significantly different from the velocity measured from the wrist to C7 computed by the F-wave method (65.0 ± 5.6 ms-1), but significantly faster than the velocity (60.4 ± 3.5 ms-1) from the wrist to C7 using SEPC (p<0.01). The mean transit time from Erb's point to C7 using the F-wave method (TA - latency of response of mixed nerve action potential recorded over Erb's point) was 2.5 ± 0.6 s-3 compared to a value of 3.3 ± 0.6 s-3 obtained by SEPC - latency of the response

Calculations:

1. Using the F-wave response the time from the wrist to C7 designated as TA is given by:

\[ \text{TA} = \left( \frac{\text{F-wave latency (s-3)} - \text{M latency (s-3)}}{2} \right) - 1 \]

assuming a central delay of 1 s-3 (Renshaw, 1941).

F-wave impulse velocity (ms-1) =

\[ \text{Distance between stimulation site and C7 spinous process (m-1)} \times \frac{1}{\text{TA (s-3)}} \]

2. Recording the cervical somatosensory evoked response, the transit time over the same segment of nerve (wrist to C7) is designated as SEPC (s-3), and the conduction velocity between these two points is given by:

\[ \text{Distance between stimulation site and C7 spinous process (m-1)} \times \frac{1}{\text{SEPC (s-3)}} \]

The distance (m-1) between the wrist and the C7 spinous process was measured with the subject sitting up and the arm outstretched horizontally.

3. Transit time to the contralateral cortex (s-3) designated as TB is given by:

\[ \text{Onset latency (s-3)} \times \frac{1}{\text{TA} - \text{F-wave response}} \]

or

\[ \text{SEPC (when recording the cervical somatosensory response).} \]

Ten patients aged between 23 and 47 years (mean 37.5 years), suspected of having multiple sclerosis but in whom the clinical deficit was limited to the lower limbs or below the C7 spinous segment, gave informed consent to undergo the same electrophysiological testing as the control group. They were examined clinically at the time of testing.
TABLE 1

Transit times and conduction velocities derived from F-wave and cervical somatosensory responses in 25 normal subjects

<table>
<thead>
<tr>
<th>Utilizing</th>
<th>Utilizing</th>
<th>Difference</th>
<th>p value (t value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>F-wave</td>
<td>Cervical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Response</td>
<td>Somatosensory</td>
<td>response</td>
<td></td>
</tr>
<tr>
<td>Transit time</td>
<td>from wrist to C7 (s⁻³)</td>
<td>10.1 ± 0.93 (8.6 - 12.1)</td>
<td>10.9 ± 1.0 (8.9 - 13.1)</td>
</tr>
<tr>
<td>Conduction velocity</td>
<td>from wrist to C7 (m⁻¹)</td>
<td>65.0 ± 5.6 (54.7 - 75.7)</td>
<td>60.4 ± 3.5 (53.6 - 68.5)</td>
</tr>
<tr>
<td>Transit time</td>
<td>from C7 to C7 (s⁻³)</td>
<td>5.3 ± 1.0 (4.05 - 7.5)</td>
<td>4.6 ± 0.84 (3.6 - 6.2)</td>
</tr>
</tbody>
</table>

Values given as mean ± 1 SD, ranges within the brackets.

Table 2 summarizes the electrophysiological data from 10 patients with suspected “spinal” multiple sclerosis.

TABLE 2

Somatosensory Studies in Patients with “Spinal Form” of Multiple Sclerosis

<table>
<thead>
<tr>
<th>Case Age</th>
<th>Velocity from wrist to C7 (m⁻¹)</th>
<th>SEPA (s⁻³)</th>
<th>SEPC (s⁻³)</th>
<th>TB (s⁻³)</th>
<th>Difference</th>
<th>p value (t value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. J.H. (F) 40</td>
<td>61.4</td>
<td>23.85</td>
<td>10.5</td>
<td>18.4 (18.6)</td>
<td>15.3 (4.0)</td>
<td>&lt;0.01 (t = 2.52)</td>
</tr>
<tr>
<td>2. J.L. (F) 42</td>
<td>54.7</td>
<td>24.6</td>
<td>No</td>
<td>13.7</td>
<td>10.5 (10.3)</td>
<td>&lt;0.01 (t = 2.52)</td>
</tr>
<tr>
<td>3. F.A. (F) 46</td>
<td>57.8</td>
<td>16.0*</td>
<td>No</td>
<td>5.7</td>
<td>10.2 (10.2)</td>
<td>&lt;0.01 (t = 2.52)</td>
</tr>
<tr>
<td>4. E.A. (F) 47</td>
<td>58.1</td>
<td>26.9</td>
<td>No</td>
<td>16.4</td>
<td>11.3 (11.3)</td>
<td>&lt;0.01 (t = 2.52)</td>
</tr>
<tr>
<td>5. T.S. (M) 29</td>
<td>57.0</td>
<td>23.8</td>
<td>No</td>
<td>13.1</td>
<td>11.1 (11.1)</td>
<td>&lt;0.01 (t = 2.52)</td>
</tr>
<tr>
<td>6. A.L. (F) 23</td>
<td>70.8</td>
<td>15.8*</td>
<td>8.9</td>
<td>6.35 (7.5)</td>
<td>&lt;0.01 (t = 2.52)</td>
<td></td>
</tr>
<tr>
<td>7. M.S. (M) 39</td>
<td>61.1</td>
<td>16.9*</td>
<td>12.1</td>
<td>5.3 (4.0)</td>
<td>&lt;0.01 (t = 2.52)</td>
<td></td>
</tr>
<tr>
<td>8. F.B. (F) 32</td>
<td>65.6</td>
<td>14.7</td>
<td>7.5</td>
<td>5.6 (6.4)</td>
<td>&lt;0.01 (t = 2.52)</td>
<td></td>
</tr>
<tr>
<td>9. M.S. (F) 42</td>
<td>63.3</td>
<td>16.9*</td>
<td>11.6</td>
<td>7.1 (4.5)</td>
<td>&lt;0.01 (t = 2.52)</td>
<td></td>
</tr>
<tr>
<td>10. B.C. (F) 36</td>
<td>59.4</td>
<td>26.8</td>
<td>No</td>
<td>10.1</td>
<td>11.3 (11.3)</td>
<td>&lt;0.01 (t = 2.52)</td>
</tr>
</tbody>
</table>

| 5.6 SD | > | < | < | < |
| Normal | 51.65 | 18.0 | 13.4 | 7.4 (6.7) | < |

No = not obtained. The values of TB (in brackets) were calculated from the corrected values of SEPC i.e. [SEPA - (SEPC - 0.8)].

* designates a markedly dispersed abnormal wave form.

recorded at Erb’s point. This numerically small difference 0.8 s⁻³ is significant (p < 0.01).

The mean onset latency of the somatosensory response recorded over the contralateral cortex (SEPA) was 15.5 ± 0.1 s⁻³. Thus the mean transit time from C7 to the contralateral cortex using the F-wave method (SEPA - TA) was 5.3 ± 1.0 s⁻³ compared to 4.6 ± 0.84 s⁻³ when calculated from SEPC (SEPA - SEPC). The mean difference of 0.74 ± 1.1 s⁻³ is again significant (p < 0.02) see Table 1. The histogram (Fig. 1) compares in graphical form the transit time (s⁻³) from wrist to C7, Erb’s point to C7, and C7 to the contralateral sensory cortex, as computed from the F-wave response and SEPC. The transit time from C7 to the cortex (TB) is significantly slower using the F-wave method as compared to TB calculated from SEPC (p < 0.02). Figure 2 is an example from a normal subject of the somatosensory evoked responses namely; cervical (SEPC) and cortical (SEPA), and also the mixed nerve action potential recorded from Erb’s point. The arrows indicate the onset latency of each response.

The rationale for recording the response over Erb’s point was to aid in deciding the onset latency of SEPC. The two recording sites are so close (about 14 cm.) that the possibility of a volume conducted response from Erb’s point contaminating SEPC had to be considered. In the example shown, the mixed nerve action potential is virtually over before the onset of SEPC. The cervical somatosensory response is, however, not always so clearly delineated as in the example. Matthews et al. (1974) have shown that the main negative (upgoing) wave is often “indented” by one and sometimes two small positive waves. We believe that the first of these small positive waves (present in about one third of normal subjects) is volume conducted from Erb’s point. Although there is presently no proof for this argument in man, we have temporarily decided it is more correct to take the onset of SEPC from the second small positive dip, if present. The same concern of some volume conducted contamination of SEPA by SEPC has to be considered in measuring the onset latency of SEPA. It will be seen (Fig. 2) that the cortical response begins (usually as a small positivity) after the cervical response has been completed (Cracco and Cracco, 1976). To achieve a more accurate measurement of the onset latencies, a fast sweep speed (5 s⁻³) with a 5 s⁻³ pre-analysis delay was utilized in the normal subjects. Clearly, a large part of the cortically evoked response will not be visualized as is shown in Fig. 2. This is of same relevance when considering the abnormal state (see below).

A good linear relationship (r = 0.84) was found between the onset latency of SEPC and arm length (Fig. 3). A similar relationship was found between TA and arm length, but the correlation coefficient dropped to 0.71.

Table 2 summarizes the electrophysiological data from 10 patients with suspected “spinal” multiple sclerosis (see discussion). Their clinical details are briefly outlined in the appendix.

In 5 patients, no convincing cervical somatosensory response was obtained. This is considered abnormal, since SEPC can invariably be obtained in normal subjects. In the remaining 5 patients, the response was of normal latency. The onset latency of SEPA was prolonged significantly in 5 patients. In four others (cases 3, 6, 7 and 9), the response, although of normal latency, was markedly dispersed in shape (see Fig. 4). No attempt has been made to measure the duration of SEPA.
Figure 2—Example of normal mixed nerve action potential recorded at Erb’s point (top), cervical somatosensory response (SEPC) - middle, and cortical somatosensory response (SEPA) - third trace. This is also shown at a slower sweep speed and higher magnification - bottom trace. Two separate trials of SEPC and SEPA are shown. A pre-analysis delay of 5 ms was used in evoking these.

systematically in normal subjects, but this would be a valuable characteristic. TB calculated by (SEPA - TA) was abnormally prolonged in 5 patients in whom SEPC was obtainable. Of the 5 patients in whom SEPC could be recorded, two (cases 1 and 6) also had prolongation of TB as measured by (SEPA - SEPC). Thus 6 out of the 10 patients studied showed evidence for a lesion between C7 and the cortex, i.e. prolongation of TB.

DISCUSSION

In this study, the transit time from the wrist to the C7 spinous process has been determined by two different methods. Using the F-wave response, the mean transit time of 10.1 s⁻¹ compared favorably with our previous mean value of 10.4 s⁻¹ (Eisen et al., 1977a). This would indicate that despite the variability of the F-wave response when recorded from a given muscle in a given individual (Upton et al., 1971), the technique in any one laboratory can give reproducible results. The onset latency of the cervical somatosensory response, a measure of the transit time over the same segment of nerve, was significantly slower. The mean value obtained (10.9 s⁻¹) can be compared to the average of 10.6 s⁻¹ reported by Matthews et al. (1974), who made the first systematic study of the cervical somatosensory response. Our mean onset latency of 15.5 s⁻¹ for the cortically evoked somatosensory response obtained with median nerve stimulation at the wrist (SEPA) is also in keeping with that originally described by Dawson (1956) and Goff et al. (1962), as well as that recently described by Dorfman (1977).

There is now ample evidence that the F-wave is mediated entirely through motor fibers (Magladery and McDougald, 1950; Gassel and Weisendanger, 1965; Thorne, 1965; McLeod and Wray, 1966; Mayer and Feldman, 1967; Miglietta, 1973). Conversely, the cervical somatosensory response is mediated, for the most part if not entirely, via sensory fibers (Matthews et al., 1974). Conduction mediated through sensory fibers in man is known to be faster than motor fibers (Dawson, 1956), but the magnitude of the difference is disputable (Magladery et al., 1951; Eisen et al., 1977b). Therefore, it was surprising that the transit time from the wrist to C7 obtained by measuring the onset latency of SEPC ("sensory fibers") was slower rather than faster than that determined by using the F-wave method ("motor fibers").

Figure 3—The regression line relates arm length to onset latency of cervical somatosensory response (SEPC). A similar linear relationship holds true for arm length and TA, calculated from the F-wave response in the same control population and is given by $y = 0.15x + 4.02$ ($r = 0.71$).
**Case**

Figure 4—Examples of traces from the first 4 cases of patients with suspected multiple sclerosis (see table 2). SEPC was obtained from case 1 only and is of normal latency (arrow). SEPA in case 1, 2, and 4, are of prolonged latency and dispersed shape (compare to fig. 2). The apparent negative wave seen in SEPA of case 1 was not time locked. In case 3, SEPA is of normal latency, but again the wave form is very dispersed. The upper 4 traces are at twice the sweep speed of the lower three traces.

The mean difference between the two transit times of 0.77 s⁻¹ (Table 1) may be explained by a single synaptic delay of approximately one millisecond. Possible sites for this delay of the latency of SEPC could be the dorsal column nuclei, spinal interneurones, or even the dorsal root ganglia.

If one takes into account this delay at or near the dorsal root ganglion, it becomes necessary to subtract 0.8 millisecond (Table 1) from SEPC to include the central delay. If this is done, the mean transit time from the wrist to C₇ would be the same whether using the F-wave or cervical somatosensory response, which become interchangeable. Similarly, TB becomes equivalent whichever method is used. Validating the equality of both methods by the necessary corrective factor is not merely of academical interest. The F-wave response is technically easier to obtain. Indeed, in confirmed multiple sclerosis as well as other diseases associated with cortico-spinal tract involvement this response is, in our experience, often of higher amplitude than normal. Presumably, this reflects release of anterior horn cell inhibition from higher centers. In contrast, the cervical somatosensory response may be abnormal in almost 90 percent of patients with confirmed multiple sclerosis (Small et al., 1977). Abnormalities of SEPC were also found in 40 percent of patients with purely ocular manifestations of multiple sclerosis by Small et al., (1977). However, it is not stated whether this represented a delay in latency or an absent response, which occurred in 4 of our 6 patients. As such, TB could still be calculated using the F-wave method.

Approximately 10 to 15 percent of patients with presumed multiple sclerosis run a lengthy and relatively benign course, which is usually progressive. In these patients, the clinical neurological deficit appears to be limited to evidence for a myelopathy. This “progressive spinal syndrome” (McAlpine and Compston, 1952; Leibowitz et al., 1967; Cárde and Kjellin, 1971; Leibowitz and Alter, 1973; Link et al., 1976) may initially be manifested by little more than hyperreflexia in the lower limbs. With time, sustained ankle clonus, extensor plantar responses, absent abdominal reflexes, and an objective sensory deficit usually develop as isolated phenomena or in combination. Although the appearance of these signs satisfies the physician as to the existence of true pathology, absence of signs above the neck still makes the diagnosis of multiple sclerosis suspect. Objective evidence for disease above the level of the lower cervical spinal cord, as indicated by slowing of conduction time from C₇ to the contralateral cortex (TB), would support the clinical suspicion of multiple sclerosis.

Sixty percent of our admittedly small group of patients, with clinical features indicating only “spinal pathology” (see appendix), demonstrated a slowing of TB, thus confirming the presence of a lesion at more than one anatomical site. Clearly, this type of physiological investigation must be extended to a larger group of similar patients before its real value can be substantiated. Bynke et al., (1977) have recently shown an abnormality of the visual evoked response in 76 percent of 25 patients with the “progressive spinal form” of multiple sclerosis. A combination of this technique and measurement of TB might give an even higher diagnostic yield than either technique alone.
The type of approach employed in the present study should also complement other physiological techniques that are providing valuable support in the diagnosis of multiple sclerosis. These have recently been reviewed by McDonald and Halliday (1977). In addition, this technique should prove useful in following the natural history of multiple sclerosis and its response to future therapy.

The present study has shown that the transit time from wrist to C7 derived from the F-wave response can be equated with the transit time over the same segment of nerve using the cervical somatosensory response, providing a corrective factor of 0.8 is applied to the latter. Thus the transit time from wrist to C7 (s⁻¹) = (SEP - 0.8) = TA, which is the transit time over the same segment employing the F-wave response.

APPENDIX

Case 1 (J. H. female 40) Four year prior to testing, weakness and numbness of both legs. Since then, fleeting parathesiae of both hands and recurrent weakness of legs, more marked on the right.

**Examination at time of testing:** Hyper-reflexia in the lower limbs, unsustained ankle clonus on the right. Reflexes in the upper limbs brisk. Toes downgoing, abdominal reflexes present. Sensory examination normal, but subjective tingling right hand. Cranial nerves normal. IGG 15 mg%, myelogram normal.

Case 2 (J.L. female 42) Ten years slowly progressive weakness and spasticity of legs, more marked on right.

**Examination at time of testing:** Marked spasticity of both legs, with hyper-reflexia and bilateral extensor clonus. Bilateral extensor plantar responses and absent abdominal reflexes. Reduced vibration in the ankles. Abdominal reflexes present. No abnormalities of cranial nerves. IGG 5 mg%, myelogram normal.

Case 3 (F.A. female 46) Slowly progressive weakness of both legs for 6 years prior to testing, associated with “ascending” numbness to level of the mid-trunk. Urinary urgency and stress incontinence.

**Examination at time of testing:** Moderate spasticity of the legs, marked hyper-reflexia. Bilateral extensor plantar and absent abdominal reflexes. Brisk finger flexion bilaterally. Diminution to pinprick with a fading level at T4. Vibration markedly diminished in the legs but normal position sense. Normal sphincter function, and no abnormality of cranial nerves. IGG 33 mg%, myelogram normal.

Case 4 (E.A. female 47) Two years prior to testing, “giving way” of left leg, and urgency incontinence.

**Examination at time of testing:** Mild spasticity of both legs, more so on the left. Hyper-reflexia with unsustained ankle clonus on the left. Bilateral extensor plantar responses, and absent abdominal reflexes. Brisk finger flexion. No abnormality of the cranial nerves. IGG 10 mg%, myelogram normal.

Case 5 (T.S. male 29) One year prior to testing, an episode of numbness left leg which recurred 4 months ago. Occasional “stiffness” of both legs.

**Examination at time of testing:** Hyper-reflexia in the lower limbs. Toes downgoing, and abdominal reflexes present. No abnormality of the cranial nerves. IGG 5 mg% (myelogram not done).

Case 6 (A.L. female 23) One week prior to testing, an acute episode of ascending numbness beginning in the legs which within 48 hours reached and remained at a segmental level of T8.

**Examination at time of testing:** Mild weakness of the legs, with hyper-reflexia and bilateral extensor plantar responses. Abdominal reflexes present. Diminished pinprick to a fading level at about T8. No sphincter disturbance and no abnormality of cranial nerves. IGG 15 mg%, myelogram normal.

Case 7 (M.S. male 38) Numbness of legs intermittently for 6 years. Two years progressive difficulty walking and recently clumsy left hand.

**Examination at time of testing:** Spasticity of both legs with marked hyper-reflexia and left extensor plantar response. Brisk finger flexion bilaterally. Diminished vibration and position sense both feet and left hand. No abnormality of cranial nerves. IGG 11 mg%, myelogram normal.

Case 8 (F.B. female 32) Numbness of left hand and both thighs intermittently since age 19. Legs fatigue with exercise, a new symptom has remained the same. Reflexes in the upper limbs brisk, and very brisk in the lower limbs. Planter responses equivocal bilaterally. Sensory exam did not reveal an objective level, and proprioception and vibration sensation were normal. IGG 11 mg%, myelogram normal.

Case 9 (B.C. female 36) Two years steadily progressive difficulty walking - legs feel stiff. Urinary frequency and urgency. 6 weeks intermittent numbness of both hands. No visual complaints or dysarthria.

**Examination at time of testing:** Evidence for a spastic paraparesis, with marked hyper-reflexia, bilateral ankle clonus, and a right extensor plantar response. Loss of vibration in the feet and poor position sense right foot. Reflexes in the upper limbs also very brisk, abdominal reflexes absent. No abnormalities of the cranial nerves. IGG 7 mg% (done 6 months prior to test), myelogram normal.

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Andrew Eisen and Kenneth Nudelman