F tacheodispersion

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Abstract

F tacheodispersion is defined as the distribution of the conduction velocities of individual or small groups of nerve fibres estimated from significant numbers of consecutively recorded F waves. The ulnar and peroneal nerves in 18 healthy subjects were studied using this method and histograms of motor fibre conduction velocities for the control nerves were created. F tacheodispersion was applied in nine patients with neuropathies and radiculopathies selected on the basis that at least one nerve was 'normal' as measured by conventional techniques (M response, F wave minimum latency to height). In the patient group it was demonstrated that a significant proportion of motor nerve fibres had F tacheodispersion conduction velocities below normal limits despite normal conventional findings. It is concluded that F tacheodispersion should be considered in routine neurophysiological investigation when conventional methods had failed to reveal a suspected normal lesion.

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Conventional electrophysiological evaluation of motor nerve conduction velocity depends on measurements of response latency of the fastest fibres, either distally (compound muscle action potential–CMAP) or along the entire length of the nerve (minimum F wave latency–FLmin). Such methods provide no information about fibres with submaximal conduction velocities and may fail to detect lesions which spare fast motor nerve fibres.

Latency measurements of statistically significant numbers of F waves (F tacheodispersion),1,2 detect mild neuropathies more sensitively than measurements of CMAP and FLmin alone. Although described in 1976, F tacheodispersion1,2 has only recently been exploited by other groups3,4 although simplified to measurement of the difference in latency between FLmax and FLmin (F chronodispersion range). Reference F latencies to the FLmin has serious disadvantages. Clustering of slow conducting fibres or inadequate numbers of F waves in severely affected nerves may erroneously produce normal F chronodispersion. Furthermore, F chronodispersion latency measurements are not corrected for patient height and nerve length, although distance dependent. Converting latency measurements to conduction velocities eliminates these sources of error and provides data that can be quantitatively manipulated.

F tacheodispersion is defined as the distribution of conduction velocities of individual or small groups of nerve fibres estimated from significant numbers of consecutively recorded F waves.

We report the results of the application of F tacheodispersion in control subjects and in 'neurophysiologically normal nerves' of patients with neuropathy—that is, nerves in which conventional methods (CMAP and FLmin) were normal.

Materials and methods

There were 18 healthy, control subjects (9 men and 9 women, age range 20–64 years, mean 39). In 17 of them the ulnar nerve was studied (seven bilaterally), and in 10 the peroneal nerve was selected (six bilaterally).

Nine patients (seven men and two women, aged 25–75, mean age 50) with polyneuropathy (seven patients), mononeuropathy (one) and radiculopathy (one) (table 1) were selected as having at least one 'normal' nerve measured by conventional methods. Six patients had their ulnar nerve studied and in seven a peroneal nerve was used. A patient with Guillain–Barré syndrome was analysed separately because the peroneal nerve was stimulated at the ankle only.

All studies were performed in a Nicolet Viking 2 electromyographic apparatus and

Table 1 Clinical syndromes and electrophysiological findings in the patient group

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Aetiology</th>
<th>Type of neuropathy</th>
<th>SNS</th>
<th>MNS</th>
<th>EMG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mitochondrial disease</td>
<td>Polynuropathy and ocular myopathy</td>
<td>+ amp</td>
<td>normal</td>
<td>neut/myop</td>
</tr>
<tr>
<td>2</td>
<td>Diabetes</td>
<td>Polynuropathy</td>
<td>+ amp</td>
<td>+ amp</td>
<td>neut</td>
</tr>
<tr>
<td>3</td>
<td>Ethanol abuse</td>
<td>Polynuropathy</td>
<td>+ amp</td>
<td>+ amp</td>
<td>neut</td>
</tr>
<tr>
<td>4</td>
<td>B-12 deficiency</td>
<td>Polynuropathy</td>
<td>+ amp</td>
<td>+ amp</td>
<td>neut</td>
</tr>
<tr>
<td>5</td>
<td>Herniated lumbar disc</td>
<td>Radiculopathy</td>
<td>+ amp</td>
<td>+ amp/MCV</td>
<td>neut</td>
</tr>
<tr>
<td>6</td>
<td>Sarcoidosis</td>
<td>Mononeuropathy multiplex</td>
<td>+ amp</td>
<td>+ amp</td>
<td>neut</td>
</tr>
<tr>
<td>7</td>
<td>Unknown</td>
<td>Proximal muscle weakness &amp; wasting</td>
<td>+ amp</td>
<td>normal</td>
<td>neut</td>
</tr>
<tr>
<td>8</td>
<td>Unknown</td>
<td>Polynuropathy</td>
<td>+ amp</td>
<td>+ amp</td>
<td>neut</td>
</tr>
<tr>
<td>9</td>
<td>Unknown</td>
<td>Compressive ulnar neuropathy?</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
</tr>
</tbody>
</table>

SNS = sensory nerve study; MNS = motor nerve study in nerves other than those studied with F tacheodispersion; EMG = electromyography; + amp = response of low amplitude or absent; + MCV = slow motor conduction velocity; neut = neurogenic EMG pattern; myop = myopathic EMG pattern.
Figure 1. Recordings from a patient with old poliomyelitis. Upper trace: M response and F wave were recorded over the extensor digitorum brevis muscle following supramaximal stimulation of the peroneal nerve at the knee. Lower trace: by reducing the stimulus level identical orthodromic (M response) and antidromic (F wave) action potentials were obtained. Calibration: M response sensitivity 0.2 mV per division, F wave sensitivity 0.2 mV per division, sweep speed 10 ms per division.

![Figure 1: Recordings from a patient with old poliomyelitis.](image)

The recordings were stored for subsequent analysis. The subjects were tested while reclining and relaxed. The skin temperature of the limb under study was monitored with a thermistor and conduction velocities corrected to a nominal temperature of 35°C using a correction factor of 1.9 m s⁻¹ per degree centigrade. The ulnar and peroneal nerves were stimulated percutaneously at wrist/elbow and ankle/knee using a square current 0.1 to 0.2 ms in duration. Belly-tendon recordings were made over the abductor digiti minimi and extensor digitorum brevis muscle respectively. To obtain at least 30 F waves from each nerve, a series of 32–130 supramaximal stimuli (intensity 1.2 times that producing maximal M response) with a frequency of 1 Hz was applied to the ulnar nerve at the wrist and to the peroneal nerve at the knee. A band-pass from 2 Hz to 10 kHz, a sweep speed of 5 to 20 ms per division (20 ms was used for recordings of delayed F waves) and a sensitivity of 0.1–0.5 mV per division for the F waves and 0.5–5 mV for the M response were used. F waves were also reviewed at higher sensitivity or longer sweep times allowed by the EMG apparatus.

The CMAP latency to onset, peak to peak amplitude, duration from onset to the final return to baseline and area were measured, as was the onset latency of each F wave complex. For each nerve the F wave persistence (percentage of stimuli evoking an F wave) was calculated.

The conduction velocity of the fastest fibres in a nerve (FCV_max) was calculated by the formula:

\[ D/([FL_{max} - M - 1]/2) \]

For the calculation of conduction velocity of fibres other than the fastest (FCVs) the above formula was modified as:

\[ D/([FL_{max} - M - 1]/2) \times (FL_{max} - 1)/(FL_L - 1) \]

where D = distance from stimulus point to C7 or L1 (mm), FL_{max} = minimum F wave latency (ms), M = M response latency (ms), FL_L = an F wave latency other than the FL_{max}. This modification was based on the assumption that the ratio of the latency of an F wave to the minimum F wave latency is inversely proportional to the ratio of the corresponding conduction velocities. The reliability of the modified formula was verified by application in identical orthodromic and antidromic responses (fig 1). The use of Kimura’s formula, which in this case is justified because the onset latency of M response and F wave represent the same fibre group, gave FCV, 41.1 m s⁻¹. Application of our formula gave a similar result (41.2 m s⁻¹).

Calculation of conduction velocity corresponding to each F wave latency measurement and creation of distribution histograms were performed by a personal computer using a specially designed program. For the presentation of the distribution of motor fibre conduction velocities (F tachoeispersion) on a graph, the FCV range was divided in bins of 2 m s⁻¹. The proportion of fibres of each subject in a bin was used to calculate the group mean for that particular, 2 m s⁻¹, bin.

F tachoeispersion procedure for each nerve (recording of F waves, mathematical and statistical calculations and graphical presentation) takes on average 10–15 minutes.

The unpaired Student’s t test was used for comparison of the means.

Results

Control subjects

Clear, usually biphasic, F waves were obtained without facilitation in all subjects. Persistence ranged from 70 to 99% for the ulnar nerve and from 24 to 88% for the peroneal nerve. Mean F chronodispersion (SD) was 3.3 (0.6) (range 2.0–4.7) ms for the ulnar and 5.1 (1.4) (2.1–6.8) ms for the peroneal.

F tachoeispersion distribution is shown in figs 2 and 3. The difference between maxi-
velocity ($FCV_{max}$) 63.9 (3.7) (57.0–69.6) m s$^{-1}$. For the peroneal nerve the $FCV_{max}$ was 46.0 (3.3) (55.7–65.2) m s$^{-1}$, the $FCV_{min}$ 52.9 (3.7) (48.0–61.3) m s$^{-1}$ and $FCV_{velo}$ 56.8 (3.0) (52.8–62.8) m s$^{-1}$. Where nerves were studied bilaterally there was no significant difference for any measurement between right and left (unpaired Student’s t test, $p > 0.2$).

We examined the possibility that the small portion of control subjects aged over 50 years may bias the fast conducting fibres in the distribution of the control group. Our data were re-analysed by applying Kimura’s formula for the calculation of $FCV$ to pooled data and compared with the results of an unpublished study performed, 12 years ago, by one of us (CPP) which referred to 25 healthy subjects of a mean age of 50 years. The differences between the distribution of motor fibre conduction velocities in the two groups of healthy subjects was negligible ($p > 0.2$) (fig 4).

**Patients**

The M response latency, amplitude, and conduction velocity of all nerves included in this group were, by virtue of selection criteria, within normal limits (table 2). M response duration and area were also normal. Easily measurable F waves were obtained from all nerves without facilitation; often two clearly separated or polyphasic F waves were recorded (fig 5). F wave minimum latency corrected for patient height was normal (table 2) and F wave persistence was similar to that of the control group (63–98% for the ulnar nerve and 40–88% for the peroneal nerve). In contrast, F chronodispersion range was prolonged in all nerves but one, 7.0 (2.0) (4.0–9.8) ms for the ulnar (only one nerve had normal value, 4 ms) and 19.3 (3.9) (10.9–22.9) ms for the peroneal. There was a corresponding increase in the range of calculated conduction velocities. The $FCV_{velo}$ in this group was 11.2 (3.6) (6.0–17.3) m s$^{-1}$ and 17.6 (4.3) (10.3–22.5) m s$^{-1}$ for the ulnar and peroneal respectively. For the ulnar nerve $FCV_{max}$ was 58.9 (3.9) (54.7–65.8) m s$^{-1}$, $FCV_{min}$ 47.6 (2.4) (42.8–50.0) m s$^{-1}$ and $FCV_{velo}$ 54.3 (3.2) (50.6–60.2) m s$^{-1}$. For the peroneal nerve $FCV_{max}$ was 55.2 (4.4) (47.3–59.6) m s$^{-1}$, $FCV_{min}$ 37.6 (1.8) (35.2–40.8) m s$^{-1}$ and $FCV_{velo}$ 47.4 (3.0) (42.3–52.0) m s$^{-1}$. Statistical analysis showed $p < 0.001$ for all the above parameters except for $FCV_{min}$ for the ulnar nerve where $p < 0.01$ and $FCV_{max}$ for the peroneal nerve where $p < 0.02$.

Figures 2 and 3 show the distribution of motor fibre conduction velocities, estimated by F tachoeispersion ($FCV$s) of the ulnar and peroneal nerves in patients compared with control subjects. F tachoeispersion allows estimation of the proportion of fibres in each nerve with conduction velocity below normal limits (table 3). The conduction velocity of 90% of the fibres in the ulnar nerve of patient 2 were slower than the mean of $FCV_{velo}$ in the control group by more than 2 SDs, despite the normal findings of conven-
tional techniques.

One patient (3) had normal F chronodispersion (4 ms) because of clustering of conducting fibres; F tacheodispersion in the same nerve was severely abnormal (fig 6).

Serial measurements of F tacheodispersion quantitatively demonstrated the improvement of a patient with Guillain-Barré syndrome (fig 7).

Discussion

Supramaximal electrical stimulation of a peripheral nerve produces an orthodromic (CMAP) and an antidromic (F wave) muscle response which are the basis for the investigation of peripheral motor neuropathies in clinical neurophysiology. Under identical conditions of stimulation and recording, consecutively recorded F waves vary in latency, amplitude, area, and morphology, because individual F waves represent different, individual, or small groups of motor units of the same nerve. F waves provide data that describe the characteristics of a population of motor units (compound F wave population).

Current practice is limited to the assessment of the Flmin with or without the corresponding FCVmax, ignoring valuable information provided by latency and other measurements of the remaining CFP. The profile of CFP regarding amplitude, latency, area, duration, and persistence may accurately reflect changes of the motor neurons providing that a statistically significant sample is studied.

F chronodispersion describes the latency characteristics of the compound F wave population and is defined as 'the scatter or dispersion of the relative latencies of statistically significant numbers of consecutively recorded F waves'. It has been shown to be more sensitive than conventional neurophysiological methods in detecting mild neuropathies where affected fibres do not influence the CMAP or the Flmin measurements but appear delayed (prolonged F chronodispersion range) in relation to the main bulk of unaffected nerve fibres.9

F chronodispersion data, which may be statistically manipulated like any other population data, is most often simplified to F chronodispersion range (Flmin–Fmax). Such presentation of F wave data relative to Flmin may result in spurious normal values when inadequate numbers of F waves are obtained (either because of severe functional depletion of nerve fibres or because of erroneous methodological practice),9 or the range of F latencies is unchanged by the neuropathic process (such as in patient 3).

Comparison of F chronodispersion latency distribution between subjects and its statistical manipulation cannot be performed as data are not corrected for height or nerve length. F tacheodispersion conduction velocity distrib-

Table 3  Patients: proportion of fibres with conduction velocity below normal limits

<table>
<thead>
<tr>
<th>Patient no</th>
<th>Ulnar nerve</th>
<th>% &lt; cut off point*</th>
<th>Peroneal nerve</th>
<th>% &lt; cut off point*</th>
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<tr>
<td>2</td>
<td>90-3</td>
<td>1</td>
<td>18-6</td>
<td>2</td>
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<td>23-6</td>
<td>2</td>
<td>34-4</td>
<td>3</td>
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<tr>
<td>6</td>
<td>34-4</td>
<td>3</td>
<td>67-8</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>47</td>
<td>4</td>
<td>12-8</td>
<td>5</td>
</tr>
<tr>
<td>8</td>
<td>34-7</td>
<td>5</td>
<td>29-9</td>
<td>6</td>
</tr>
<tr>
<td>9</td>
<td>20-7</td>
<td>7</td>
<td>14-5</td>
<td>8</td>
</tr>
</tbody>
</table>

Cut off point = mean of FCVmax in the control group – 2 SDs.
Figure 6  Distribution of FCVs of the ulnar nerve in a patient (3) with alcoholic polyneuropathy compared with control subjects. Although F chronodispersion range is normal, there is a clear shift of the patient's FCVs towards slower values.

Figure 7  Distribution of FCVs of the peroneal nerve in the patient with Guillain-Barré syndrome. Note the increased range of F tachodispersion in the first study, 1 month after onset of the symptoms. One year later, following the complete clinical recovery of the patient, the abnormally slow conducting fibres have recovered.

The calculation of conduction velocity was based on a widely accepted formula. The M response and F wave may represent different fibre populations, however, this formula was modified to attribute latency differences between F waves to conduction velocity differences. Although apparently complex, the required calculations were performed rapidly by a personal computer. Some authors have expressed concern that calculation of conduction velocities from F wave data introduces error from inaccurate measurement of the distance travelled by the propagating impulse and the formula used for this calculation. This is only of theoretical interest as previously detailed. In practice any method of comparison of conduction properties between nerves requires some measurement of distance, and errors introduced by the formula are likely to be similar between patients.

In the present study, sensitivity of F tachodispersion did not differ significantly from F chronodispersion range as 12 out of 13 nerves were found to be abnormal with both methods. This is attributable to selection of mildly affected nerves—that is, those with normal conventional measurements. Unlike F chronodispersion, F tachodispersion provided quantitative comparisons with nerves of healthy subjects allowing assessment of the proportion of abnormally conducting fibres. Such data also serve as basis for longitudinal studies.

Because F tachodispersion data describe the characteristics of a population of fibres in a nerve, sufficient F waves must be recorded in order to reflect accurately the population profile. This number has been estimated for F chronodispersion as at least 20, and should be similar for F tachodispersion.

Other techniques designed for estimating the conduction velocity distribution, have employed computer simulation programs or used collision of paired stimuli. All depend on CMAP and are restricted to measurement of the distal nerve segment. The higher nerve conduction velocities estimated by F tachodispersion are the result of inclusion of proximal segments of the nerve in the measurement, which are faster than the distal segments. For example the mean values (SD) of FCV_max and FCV_min of the ulnar nerve in our control group were 67-7 (4-3) m s⁻¹ and 59-8 (3-5) m s⁻¹ respectively. Using other techniques the value for FCV_max ranged from 57-2 (4-7) m s⁻¹ to 61-9 m s⁻¹, and for FCV_min from 43-4 (2-9) m s⁻¹ to 55 m s⁻¹.

It has been suggested that F wave methods may sample a neuronal subpopulation (especially when small numbers of F waves are used) because of the preferential generation of F waves by large neurons. The difference between FCV_max and FCV_min (FCV_dif) in this study was 7-9 (1-6) m s⁻¹, which agrees with other reports using M response techniques, [5-3 (0-9) m s⁻¹] to 17-9 (3-5) m s⁻¹]. The similarity of FCV_dif estimations and the normal distribution of FCVs in our study indicates that neuronal subpopulations are represented to the same extent as in M response studies.

In conclusion, F tachodispersion is a quantitative method which could be routinely applied in the examination of patients with suspected neuropathies, particularly when conventional neurophysiological findings are normal or borderline.

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