Disturbances in the voluntary recruitment order of anterior tibial motor units in ataxia

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SYNOPSIS  The recruitment order of motor units was studied with an electromyographic technique for secure identification of single motor unit potentials. It has been shown in previous studies of normal subjects that the recruitment order in sustained voluntary contraction is predominantly stable, and that motor units which increase slowly in discharge rate with increasing contraction strength and which already attain regular discharge intervals at low frequencies are always recruited before motor units which increase more rapidly in discharge rate and which do not attain regular discharge intervals until at higher frequencies. In this study 15 patients with severe cerebellar ataxia were examined. It was shown that the recruitment order in sustained voluntary contraction in ataxia is unstable and that low- and high-frequency motor units may alternate as the unit of lowest threshold.

In electromyographic recordings, single motor units can be identified by the characteristic shape of their potentials. Consequently, their discharge patterns and recruitment order can be studied in voluntary contraction in man (Adrian and Bronk, 1929; Smith, 1934).

Different human motor units have different discharge patterns. Certain motor units increase slowly in discharge frequency on increasing contraction strength and already discharge at regular intervals even at low frequencies and have low maximum frequencies. Other motor units increase faster in discharge rate and do not discharge at regular intervals until higher frequencies and have higher maximum frequencies (Grimby and Hannerz, 1974a; Hannerz, 1974). It is likely that the differences in discharge pattern of the motor units reflect differences in gain of their motoneurones and contraction time of their muscle fibres (cf. Discussion).

Normal subjects are able to choose the recruitment order that seems optimal for the work intended. Where precise or sustained voluntary work is intended, the recruitment order is stable and low frequency units are recruited first. When rapid movement is intended, on the other hand, the recruitment order is unstable and high frequency units play a greater role.

A series of studies on voluntary contraction and patients with different neurological symptoms is in progress with a view to determine the patient's ability to select the proper recruitment order for the work intended.

In a previous paper (Grimby and Hannerz, 1974b) the starting difficulty of patients with severe bradykinesia of Parkinsonism was studied. It was shown that bradykinetic patients have difficulty in establishing 'tonic recruitment order' and have to depend on 'phasic recruitment order' in the initiation stage of sustained voluntary contraction. In another paper (Grimby et al., 1974) the decreased voluntary endurance of flexor muscles in spastic paraparesis was studied. It was shown that spastic patients have difficulty in maintaining 'tonic recruitment order' when fatigue appears and tend to change to 'phasic recruitment order'.

In this paper the decreased ability of ataxic patients to keep a stable contraction level in sustained voluntary contraction is studied. It is shown that the variations in contraction level are combined with variations in recruitment order in such a way that 'tonic recruitment order' is replaced intermittently by 'phasic recruitment order' during sustained voluntary contraction.
METHODS

The subjects studied were 15 patients who according to clinical signs had cerebellar ataxia and were unable to maintain a constant contraction strength in a weak sustained voluntary contraction of the anterior tibial muscle. Patients with such severe ataxia as this seldom have anatomically well-defined lesions in the nervous system. Patients with clinical signs of lesion of the upper motoneurone or peripheral nerve were excluded, however, since it has been shown in previous studies that the recruitment order of motor units is disturbed in spastic pareses (Grimby et al., 1974) as well as in peripheral neuropathy (Grimby and Hannerz, 1973).

Cases 1 and 2 had hereditary ataxia. Neuro-radiological investigations showed severe cerebellar atrophy. The conduction velocity in peripheral nerves was normal. Case 3 had ataxia after a complicated operation on an acoustic neuroma. Case 4 had a superior cerebellar artery syndrome. Cases 5 to 7 had multiple sclerosis. Cases 8 to 12 were alcoholics with cerebellar degeneration but without clinical signs of peripheral neuropathy at the time of the experiment. Cases 13 to 15 had ataxia the origin of which was not completely investigated.

The electromyographic recording techniques were the same as those used in previous studies (Grimby and Hannerz, 1974b). The electrodes were of low-impedance type (Disa No. 9013 K0802), recording approximately 10 to 20 different units in the anterior tibial muscle (Ashworth et al., 1968).

The amplifier was connected to a loudspeaker. The patients were instructed to contract the muscle in such a way that a continuous sound was emitted by the loudspeaker and also to moderate the contraction so that the sound became as weak as possible but remained continuous. The shoe of the patient was fixed to a myograph.

The first motor unit was recruited already before any muscle tension could be recorded. As the contractions studied were so weak, they were not purely isometric.

The discharge of a single motor unit was classified as regular if the single discharge intervals did not diverge more than 20% from the mean value.

RESULTS

NORMAL SUBJECTS In normal subjects the recruitment order of motor units in sustained voluntary contraction is predominantly stable as mentioned previously. In most recordings one motor unit constantly has the lowest threshold in a recording of five minutes’ duration, but in some experiments two motor units with neighbouring thresholds may alternate as the unit of lowest threshold. Units which appear as the unit of lowest threshold have the same discharge characteristics. They increase slowly in discharge rate on increasing contraction strength and attain regular discharge intervals at a frequency of seven per second. Units which are capable of faster increases in discharge rate and which do

FIG. 1. Unstable recruitment order of a ‘low frequency motor unit’ and a ‘high frequency motor unit’ in ataxia. Further description in text. Time bar: 100 ms.
not attain regular discharge intervals until frequencies above 10 per second normally never participate in a sustained voluntary contraction below 20% of maximum tension. For further data concerning the normal recruitment order, see Grimby and Hannerz (1968) and Hannerz (1974).

**ATAXIC SUBJECTS** Patients selected because they could not keep a constant contraction level in sustained voluntary contraction were also unable to keep a constant recruitment order.

In every five minutes recording three to six different motor units alternated as the unit of lowest threshold. Most of these units increased slowly in discharge rate on increasing contraction strength and attained regular discharge intervals by frequencies below 10 per second—that is, they had about the same discharge.

**FIG. 2.** Change in recruitment order of motor units when the ataxic subject tries to re-establish activity after an involuntary pause. Further description in text. Time bar: 100 ms.

**FIG. 3.** Change in recruitment order of motor units just before an involuntary pause. Each trace is a direct continuation of the trace above. Further description in text. Time bar: 100 ms.
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pattern as do low threshold units in normal subjects.

However, one or two out of six alternating units might have a diverging discharge pattern. In Fig. 1, two units with significantly different discharge pattern alternate as the unit of lowest threshold in sustained voluntary contraction. The unit that has the lowest threshold in Fig. 1A is called the I-unit and the unit that has the lowest threshold in Fig. 1B is called the II-unit. In Figs 1A and B, the units are driven at the lowest frequency that is compatible with regular discharge intervals. The I-unit is capable of discharging as slowly as 7/s. The II-unit, however, must be driven above 15/s. In Fig. 1C the I-unit has the lowest threshold and the voluntary drive is adjusted in such a way that the II-unit is activated only sporadically. Nevertheless it discharges at shorter intervals than does the I-unit. In Fig. 1D, the I-unit still has the lowest threshold and the contraction strength is rapidly increased. The II-unit increases faster in discharge rate than does the I-unit. The findings may be interpreted as meaning that the motoneurone of the II-unit has a higher gain than has the motoneurone of the I-unit (cf. Discussion).

During each experiment there was a close correlation between the degree of ataxia and the degree of instability of recruitment order. When the level of contraction was relatively stable, the recruitment order was also relatively stable. The unit of lowest threshold was then always of low-frequency type. When, however, the ataxic subject had great difficulty in keeping a stable contraction level, the recruitment order was also unstable. A high-frequency unit might then temporarily have the lowest threshold. Variations in recruitment order were particularly observable in connection with involuntary pauses.

When activity started again after a pause, the recruitment order was unstable. The harder the subject tried to reestablish contraction, the shorter was the pause, but the greater was the instability of the recruitment order. In Fig. 2A, the subject has no difficulty in keeping a steady contraction and one unit discharges at a low regular frequency. In Fig. 2B and C, the subject tries to reestablish contraction after an involuntary pause. Two new units alternate as the unit of lowest threshold and there is a marked tendency towards bursts of several units. In Fig. 2D the subject finally succeeds in maintaining a steady contraction and the original unit discharges, again at long, regular intervals.

Shifts in recruitment order also occurred just before involuntary pauses. A pause was often preceded by suddenly increased activity. One of those units which were recruited when activity was increased tended to be the last to discharge before the pause instead of the unit which originally had the lowest threshold. In Fig. 3A, one unit is continuously discharging at long, regular intervals. In Fig. 3B, an additional unit discharging at slightly shorter intervals is recruited. In Fig. 3C, the subject fails to maintain the contraction and just before the pause the recruitment order of the two units is reversed.

DISCUSSION

Normal subjects are able to maintain a constant contraction strength and a stable recruitment order in sustained voluntary contraction. The ataxic subjects of this study were selected because they could keep a constant contraction strength only for brief periods of time. It is shown that the variations in contraction strength are combined with variations in recruitment order. Thus, in ataxia not only the level of facilitation but also the mode of facilitation of the motoneurone pool is unstable.

It is also shown that the motor units which alternate as the unit of lowest threshold in ataxia have different discharge patterns. Most of them increase slowly in discharge rate on increasing contraction strength and already discharge at regular intervals at low frequencies. These units have the same discharge patterns as do units with low threshold in normal sustained voluntary contraction. However, some of the units appearing as the unit of the lowest threshold in ataxia increase faster in discharge rate and need higher discharge frequency to attain regular discharge intervals. Their discharge pattern is in accord-
majority of the low-threshold units retain their original pattern.

Phasic motoneurones have a higher gain than tonic motoneurones and thus greater ability to increase rapidly in discharge rate. Fast-twitch muscle fibres need a higher discharge frequency to fuse than do slow-twitch muscle fibres. We think it is reasonable to assume that the greater role played by high-frequency units in weak sustained voluntary contraction in ataxic than in normal subjects reflects a greater role of phasic motoneurones and fast-twitch muscle fibres.

The recruitment order disturbance found in ataxic subjects can also be evoked in normal subjects by blockades of the afferent inflow (Grimby and Hannerz, 1973, 1975). If the muscle nerve is partially blocked by Lidocaine or local pressure during sustained voluntary contraction the recruitment order of a low- and a high-frequency unit may be reversed. The same disturbance of recruitment order is present also in myopathy so severe that voluntary effort hardly produces any tension (Grimby, 1974). It seems likely that defects in the servo mechanisms play a part in the instability of the recruitment order in ataxia also.

The recruitment order of a low- and a high-frequency unit may normally be changed upon a change from sustained to twitch contraction also (Grimby and Hannerz, 1968; Hannerz, 1974). The recruitment order is particularly unstable when the ataxic subject tries to reestablish contraction after involuntary pauses. It seems as though the ataxic subject has difficulty in keeping the mode of facilitation of the motoneurone pool that is normally used for sustained voluntary contraction and so has to depend intermittently on the mode of facilitation which normally is used only for voluntary twitches and which presumably is less linked to the gamma loop. Changes in recruitment order also appear after involuntary increases of the contraction strength. These changes may be due to inhibiting impulses from supraspinal centres but may be explained also by insufficient muscle spindle adaptability.

As all the subjects had severe cerebellar ataxia and as there was a close correlation between the degree of instability of recruitment order and the degree of ataxia, we assume that the instability of the recruitment order found in this study is caused by cerebellar dysfunction.

As the disturbance of recruitment order can also be evoked in normal subjects by blockade of the afferent inflow and as most of the cases studied had diseases which were not restricted to the cerebellum it can, however, be objected that the cause may be peripheral neuropathy which in other respects is subclinical. This interpretation is, however, unlikely, since it would imply that ataxia which according to clinical signs is of cerebellar origin would, in fact, be due to peripheral neuropathy selectively affecting afferent nerve fibres from muscle. Moreover, in cases 1 to 4 significant peripheral neuropathy can be ruled out.

The similarity between the disturbance of recruitment order found in patients with cerebellar ataxia and that evoked by blockade of the proprioceptive activity in normal subjects may be due to the fact that the cerebellum is a co-ordinator of the proprioceptive inflow and the alpha and gamma motoneurone innervation. It is likely that the changes in voluntary recruitment order obtained by blockade in normal subjects are mediated not only by spinal reflex arcs but also by the cerebellum and other supraspinal centres.

REFERENCES


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