Voluntary control of arm movement in athetotic patients

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SYNOPSIS Visual tracking tests have been employed to provide a quantitative description of voluntary control of arm movement in a group of patients suffering from athetoid cerebral palsy. Voluntary control was impaired in all patients in a characteristic manner. Maximum velocity and acceleration of arm movement were reduced to about 30-50% of their values in normal subjects and the time lag of the response to a visual stimulus was two or three times greater than in normals. Tracking transmission characteristics indicated a degree of underdamping which was not present in normal or spastic patients. This underdamping could be responsible for a low frequency (0.3-0.6 Hz) transient oscillation in elbow-angle movements associated with sudden voluntary movement. The maximum frequency at which patients could produce a coherent tracking response was only 50% that in normal subjects and the relationship between the electromyogram and muscle contraction indicated that the mechanical load on the biceps muscle was abnormal, possibly due to increased stiffness of joint movement caused by involuntary activity in agonist and antagonist muscles acting across the joint.

Athetosis is characterized by involuntary movements and abnormal postures which involve chiefly, but not exclusively, the distal musculature. Many authors have attempted to describe or to measure the involuntary movements which are considered important for the diagnosis and classification of athetosis (Hammond, 1871; Foerster, 1921; Wilson, 1925; Herz, 1931; Hoefer and Putnam, 1946; Carpenter, 1950; Bobath and Bobath, 1952; Koven and Lamm, 1954; Twitchell, 1961; Narabayashi *et al.*, 1960, 1965). Less attention has been given to measurement of voluntary control and interaction between voluntary and involuntary movement in athetosis. Most of the theories which have been proposed to explain the involuntary movements of athetosis imply that the ability to organize and execute purposive movements is also impaired.

Hoefer and Putnam (1940) studied electromyographic (EMG) patterns in athetotic muscles and decided that there are two types of voluntary

movement. The first is normal; the motor units act independently and asynchronously, fatigue is minimal and the movement is smooth and precise. The second type of movement is pathological. The motor units discharge synchronously because reflex mechanisms dominate the muscle contraction. The movements are weak and fatigue easily.

A frequently expressed view is that control of voluntary movement in athetosis is impaired because of the release of postural reflexes from inhibitory control of higher centres (Bucy, 1942; Bobath and Bobath, 1952; Stanley-Jones, 1956; Twitchell, 1961; Gillette, 1964). Bucy (1942) considered that involuntary movements were caused by interruption of a suppressor mechanism causing hyperactivity of motor and premotor cortex. Bobath and Bobath (1952) commented that patients display stereotyped patterns of voluntary movement which are dictated by a combination of hypersensitive postural reflexes. The patient is unable to contract an individual muscle without a reflex spread of activity to

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involve other muscles. Although the patient can initiate movement, voluntarily activating the appropriate motor paths, the movement itself is largely carried out by reflex action outside his control. Twitchell (1961) concluded that it is not the presence of involuntary activity which impairs the performance of voluntary movement in athetosis, but rather an inability to integrate postural reflexes. Mettler and Stern (1962) discussed the possibility that athetoid movements may be caused by forcing voluntary effort through a more diffuse circuitry than the normal corticospinal system. Such theories suggest that a distinctive pattern of impaired voluntary control may exist in athetosis.

Voluntary effort and emotional stress aggravate involuntary athetoid movement (Koven and Lamm, 1954). Indeed, involuntary movements are obvious in some patients only during voluntary activity when they often interfere with performance and in extreme cases may completely disrupt the intended movement. The physiological basis of the interaction between voluntary and involuntary movement is unknown. A necessary step in investigating the mechanism is to obtain an objective description of the voluntary control of movement in athetosis.

The tonic stretch reflex (TSR) in athetosis is functionally reorganized during voluntary activity (Neilson and Andrews, 1973). Neilson (1972c) suggested that the action TSR pathways might provide the feedback required in the

length follow-up servo theory for control of movement (Merton, 1953). If this suggestion is correct, an abnormal action TSR in athetosis should contribute to the deterioration of voluntary control. A correlation between characteristics of the action TSR and features of voluntary control could indicate which aspects of the movement disorder are caused directly by lesions in the basal ganglia or other higher centres and which are secondary to changes in muscle tone. The purpose of the experiments explained below is to provide an objective description of the control of voluntary movement about the elbow joint in patients suffering from athetotic cerebral palsy.

METHOD

Ten cerebral palsied patients presenting a clinical picture of athetosis were tested using the following experimental procedures:

FREE WHEEL TEST This test has been described elsewhere (Neilson, 1972a). The right elbow angle was measured by a goniometer strapped to the arm and the EMG activity of the right arm biceps muscle was recorded by surface electrodes attached 5 cm apart over the muscle belly. The EMG was amplified and displayed on a 17 in. oscilloscope for continuous monitoring to assure that the signal was not contaminated by movement artefact. It was also amplified in a Grass 5P3 preamplifier, integrated (time constant—0.16 sec) and recorded on a model 5 Grass 4-channel polygraph. The elbow-angle signal from the goniometer was recorded on the polygraph

FIG. 1. Section of a typical polygraph recording of elbow-angle, and EMG and IEMG from biceps brachii muscle recorded during a free wheel test. By inspection, the EMG can be seen to have a phase lead of 180° to 270° ahead of the flexion movement.

so that a downward pen deflection of 10 mm corresponded to a 10° extension change in joint angle. The subject lay supine on a couch. He was instructed to oscillate the right arm back and forth about a mean 90° joint angle position as rapidly as possible. Thus the maximum speed of voluntary arm movement could be assessed and compared with that measured previously by the same method in both normal subjects (Neilson, 1972a) and spastic patients (Neilson, 1972c). A statistical description of the EMG signal and arm movement was obtained by computing correlation functions and power spectra using computer programmes described previously (Neilson, 1972a). The elbow-angle power spectrum enabled the predominant free wheeling frequency to be measured and the amount of distortion or deviation from a sinusoidal motion to be appraised. Phase lag of elbow-angle movement behind EMG activity in biceps was evaluated from the real and quadrature components of the cross power spectrum. This technique has been explained elsewhere (Neilson, 1972c).

VISUAL TRACKING TEST The elbow-angle signal and the integrated EMG (IEMG) of the biceps muscle were recorded on the polygraph in the same manner as described above. Polygraph sensitivity was reduced so that a 10° change in elbow-angle corresponded to a 5 mm deflection of the pen. The elbow-angle signal from the goniometer was also used to control the vertical position of a horizontal line on an oscilloscope screen. Sensitivity was adjusted so that a 10° elbow-angle extension movement corresponded to a 1 cm downward deflection on the screen. A second not so bright and slightly defocused horizontal line on the screen was moved up and down in an irregular fashion by the experimenter. The position of this target line was also recorded on the polygraph. A 1 cm downward deflection on the screen corresponded to a 5 mm downward deflection of the pen. The oscilloscope was positioned so that it could be viewed comfortably by the patient. He was instructed to move the right arm so as to keep the two lines on the oscilloscope screen superimposed. This required him to perform a series of irregular arm movements at different speeds and angular displacements about the elbow. The exact nature of these movements was dictated by the motion of the target line which was controlled by the experimenter. It involved movement through the full range of flexion-extension about the elbow and a range of speeds up to and slightly exceeding the maximum speed at which the patient could track. The movements were performed in a closed loop fashion in the sense that the patient was required to use visual information to organize the movement and

then the motion influenced the visual information thereby closing the feedback path. Ability to perform voluntary movement was assessed by analysis of target, IEMG, and elbow-angle signals recorded on the polygraph. The misalignment or error between target and elbow position was computed by subtracting the elbow-angle signal from the target signal. The root mean square value of the error signal provided an index of tracking performance for each test. Correlation functions and power spectra were computed to provide a statistical description of the signals for each test (Jenkins and Watts, 1968). Coherence between target and elbow-angle signal was calculated for each test (Jenkins and Watts, 1968). Coherence can be interpreted in this case as the proportion of elbow-angle movement at any frequency which was correlated with the target. Coherence also provides an indication of the patient's tracking performance because the presence of involuntary movement at any frequency reduces the coherence at that frequency. Coherence between target and IEMG signals and between IEMG and elbow-angle signals was also computed. Finally the patient's tracking performance on each test was specified by computing the gain and phase frequency response characteristics describing the relationship between target and IEMG, IEMG and elbow-angle, and target and elbow-angle signals. These characteristics apply only to the coherent portions of the signals and therefore the target to joint angle relationship describes the closed loop voluntary control of arm movement. The involuntary fluctuations in:

FIG. 2. A typical power spectrum of the elbow-angle signal recorded during a free wheel test. The predominant peak in the spectrum and the absence of harmonically related peaks indicates that the movement was a good approximation to a sinusoid. For the patients tested the peak was always between 1.5 and 2 Hz.

elbow-angle which occurred during each test were not coherent with the target signal and so were not included in the frequency response characteristics. The gain in each case was defined as the ratio of the amplitude of the output sine wave to that of the input.

Each tracking test lasted three minutes and each patient was tested four times. A rest period was given between each test.

RESULTS

FREE WHEEL TEST Ability to free wheel the right arm varied between athetoid patients (Fig. 1).

FIG. 3. An illustration of the calculation of phase lag of elbow flexion movement behind the EMG of the biceps muscle for signals recorded during a free wheel test. The real component (a) and the quadrature or imaginary component (b) of the cross power spectrum between the IEMG and elbow-angle signals are computed. The magnitude of the peaks at the free wheeling frequency, 2 Hz in this case, are measured and plotted on an argand diagram (c). The phase lag angle θ of flexion movement behind the IEMG signal can be measured from the diagram. The phase lag introduced into the IEMG signal by the integrating filter is added to θ to obtain the phase lag of flexion movement behind the EMG signal. For all patients this was in the range 180° to 270° .

The movement was rhythmical in each case as verified by the predominant peak in the power spectrum of the elbow-angle signal (Fig. 2). The frequency of the predominant peak varied between patients in the range 1.5–2 Hz. The size of the peak also varied between patients but the mean peak to peak amplitude of the movement was always 10° to 20° .

The phase lag of elbow flexion movements behind EMG activity in the biceps muscle was determined by measuring the size of the peaks at the free wheel frequency in both the real and quadrature cross-power spectra relating IEMG to elbow-angle signals (Fig. 3). After correction for the phase lag introduced by the integrating filter it was found that the EMG to elbow flexion phase lag was always 180° to 270° .

VISUAL TRACKING TEST Satisfactory polygraph recordings of visual tracking test data were obtained for all patients (Fig. 4). Target, IEMG, elbow-angle, and error signals were described statistically by power spectral curves (Fig. 5). In spite of variations between patients, a number of spectral features were consistent for all tests.

The power spectra all contained a large predominant peak at low frequencies (<0.5 Hz) indicating that most tracking movements were in this range. The size of the low frequency peak in the elbow-angle spectrum was always less than half that in the spectrum of the target signal.

The target signal, which was controlled by the experimenter, contained no frequencies higher than about 1.5 Hz as indicated by the power spectrum reaching a negligible value for all frequencies greater than 1–2 Hz. The elbow-angle spectrum, on the other hand, did not reach negligible values until the higher frequency of 2–3 Hz because of high frequency involuntary movement. The power spectrum of the IEMG signal was spread across the entire band (0–5 Hz) and displayed secondary peaks between 1 and 4 Hz.

The proportion of the total arm movement variance at any frequency which was correlated with the target signal and therefore represented voluntary movement, was measured by the coherence between target and elbow-angle signals (Fig. 6a). For all patients the coherence started at a high value (0.6–0.8) at low frequencies (0.1–0.4 Hz) and then decreased with frequency

FIG. 4. Section of a typical polygraph tracing of elbow-angle, target, EMG, and IEMG of the biceps muscle recorded during a visual tracking test. 2.5 Hz tremor can be seen in the elbow-angle signal but it is most obvious in the EMG and IEMG recordings. The target motions were irregular.

reaching a negligible value between 1 and 1.5 Hz.

The coherence between target and IEMG signals was also computed (Fig. 6b). The IEMG provided a very poor measure of voluntary movement at low frequencies (<0.3 Hz). The coherence was very low (0.3) at these frequencies

but increased to a maximum value (0.4–0.8) by 0.3–0.5 Hz. It then decreased again to a negligible value at a frequency between 1 and 1.5 Hz.

The coherence between IEMG and elbow-angle signals (Fig. 6c) was also very poor at low frequencies (<0.3 Hz) but it increased to

FIG. 5. Power spectra of target, IEMG, elbow-angle, and error signals recorded during a visual tracking test. In each graph power is plotted in arbitrary units equal to mm of pen deflection squared. Frequency is plotted in Hz.

FIG. 6. Coherence function between (a) target and elbow-angle, (b) target and IEMG, and (c) IEMG and elbow-angle signals recorded during a visual tracking test. Coherence provides a measure of the proportion of the variance of the response signal at each frequency which is correlated with the input signal. Frequency has been plotted in Hz.

FIG. 7. Gain and phase frequency response curves describing the mathematical relationship between the coherent portions of the target and elbow-angle signals recorded during a visual tracking test. The phase characteristics have been approximated by those of a 0.4 sec time delay. Gain is plotted in db and phase in degrees. Frequency is plotted in Hz on a logarithmic axis.

maximum value (0.8) and then decreased with frequency not reaching a negligible value until about 3–4 Hz.

Visual tracking performance was specified by gain and phase frequency response curves (Fig. 7). These curves provide a graphical description of the mathematical relationship between the target signal and the elbow-angle response. A number of features of tracking performance were similar for every patient. Gain was always less than one, it increased to a maximum (0.6–0.8) at a frequency between 0.3 and 0.6 Hz and then decreased to a negligible value by 1–1.5 Hz. Thus there was always a peak in the gain curve at 0.3–0.6 Hz. Elbow-angle responses always lagged in phase behind the target signal. At low frequencies (0.1 Hz) the phase lag was 0°–30°. It increased with frequency reaching 180° between

FIG. 8. Phase characteristics describing the mathematical relationship between IEMG and elbow-angle signals recorded during visual tracking tests. The variation between individuals has been illustrated by the difference between characteristic of subject A and subject B. The phase relation between force and displacement of a pendulum has been graphed to illustrate how the phase characteristics could be altered by a change in resonant frequency. Phase is plotted in degrees. Frequency is plotted in Hz on a logarithmic axis.

1 and 1.5 Hz. Since coherence between target and elbow-angle signals decreased to a negligible value by this frequency the concept of gain and phase becomes meaningless for higher frequencies. The phase characteristic for frequencies up to 1 Hz could be approximated reasonably well by the phase lag characteristic of a 0.4 sec time delay as shown in Fig. 7b.

The phase characteristics between IEMG and elbow-angle signals were also calculated. A surprising result was the large variation between patients in these phase curves at low frequencies. This variation has been illustrated (Fig. 8). The phase lag of elbow-angle behind IEMG at 0.1 Hz varied between patients in the range 5°–230°.

DISCUSSION

FREE WHEEL TEST The free wheeling frequency of 1.5–2 Hz in athetoid patients reported here was considerably lower than the 4–6 Hz measured previously in normal subjects (Neilson, 1972a) and overlaps the range (0.6–2 Hz) of spastic patients (Neilson, 1972c). The phase lag between EMG activity and elbow flexion movement was similar (180°–270°) for each group of

subjects. As was argued previously (Neilson, 1972c), this large phase lag indicates that the maximum velocity and acceleration of arm movement is limited by the mechanical load on the muscle, since power of the biceps muscle was not clinically impaired.

Inspection of the phase curves (Fig. 8) which describe the relationship between IEMG and elbow-angle changes indicates that there is great variation between patients. The curves are similar to the force-displacement phase curves of a pendulum which drop through a 180° phase range at the resonant frequency of the pendulum (Fig. 8). The resonant frequency of the pendulum equals the square root of the ratio of stiffness to inertia where stiffness is defined as the slope of the static force-displacement graph. A change in stiffness would therefore change the resonant frequency at which the phase drops through 180° . The variation between patients in the IEMG to elbow-angle phase curves can therefore be interpreted as a change in the resonant frequency of the mechanical load on the biceps muscle.

It seems reasonable that the amount of applied force required to alter the joint angle is increased when opposing muscle groups acting across the joint contract simultaneously. The slope of the length-tension curve of muscle has been shown to increase with contraction level (Rack and Westbury, 1969; Rosenthal *et al.*, (1970), implying a greater muscle stiffness. The presence of involuntary activity in opposing muscle groups in athetosis will therefore increase the stiffness of the joint and change the mechanical load on the biceps muscle. Lack of reciprocal inhibition is an outstanding feature of athetosis (Hoefler and Putnam, 1940), and appears to be the main factor in increasing the stiffness of the joint, changing the resonant frequency of the mechanical load on the muscle and reducing the maximum speed of movement to about 50% of the value in normal subjects.

VISUAL TRACKING TEST The power spectra providing statistical description of target, IEMG, elbow-angle, and tracking error signals each contained a predominant low frequency peak (Fig. 5). Although the target signal was controlled by the experimenter so that the fastest movements were just beyond the patient's tracking ability

(1–2 Hz), both the elbow-angle and IEMG signals contained higher frequencies apparently caused by involuntary athetoid activity.

In spite of involuntary activity, the athetoid patients made less low frequency movement than required for ideal tracking in which target and elbow-angle signals would be identical. This is indicated by the low frequency peak in the elbow-angle power spectrum always being less than half the size of the corresponding peak in the target spectrum. It was previously reported that spastic patients make more low frequency movement than required for ideal tracking and it was suggested that the tracking test may have revealed an underlying athetoid component not apparent on clinical examination (Neilson, 1972c). The results here suggest that this was not the case and that excessive low frequency movement during voluntary effort is a feature of spasticity not observed in athetosis. In athetosis there are low frequency involuntary movements, but in spasticity there are excessive levels of inappropriate voluntary movement.

A reinspection of earlier results (Neilson 1972a) revealed a similar insufficiency of low frequency movement in the tracking performance of normal subjects. An examination of the polygraph recordings shows that the subjects did not respond to almost all the low frequency target changes. Perhaps the insufficiency of low frequency power in the elbow-angle signal is characteristic of the way in which the central nervous system (CNS) organizes voluntary movements. Stark *et al.* (1962) demonstrated that in tracking a slowly changing ramp signal a series of brief movements are executed which cause step-like changes in position at irregular intervals. The presence of intermittency in control of voluntary movement has been indicated by other workers (Bekey, 1962; Stark *et al.*, 1962; Navas and Stark, 1968). Such intermittency, as well as an attempt to correct for phase lag caused by reaction time, could be responsible for the low frequency variance in the elbow-angle signal being less than ideal.

The maximum frequency (1–1.5 Hz) at which athetoid patients could produce a coherent response to the visual stimulus was less than the maximum frequency (1.5–2 Hz) at which they could freewheel the arm. The maximum frequency of a coherent response to a visual stimu-

lus must therefore be limited by central processes rather than by muscle and limb mechanics. A similar conclusion was made previously for normal subjects (Neilson, 1972a). The limitation is most likely imposed by the time required for the CNS to decode sensory information and organize the appropriate motor response. The phase lag curve between target and elbow-angle response (Fig. 7) indicates that the time delay is about 0.4 sec in athetoid patients compared with about 0.17 sec in normal subjects.

The coherence functions (Fig. 6) demonstrate that the IEMG provides a very noisy measure of muscle contraction, particularly at low frequencies (<0.3 Hz) where about 60% of IEMG fluctuations are incoherent with elbow-angle changes. At higher frequencies (3–5 Hz) the amplitude of the elbow-angle signal decreases but that of the IEMG signal increases (Fig. 4). For these reasons the elbow-angle signal provides the best measure of low speed movements (<0.5 Hz) while the IEMG provides the best measure of high frequency activity (>0.5 Hz).

Apart from the disrupting influence of involuntary activity the voluntary control of movement in athetosis is impaired in a characteristic fashion. This is indicated by the gain and phase frequency response curves (Fig. 7). The bandwidth is reduced to about half and the phase lag is more than double its value at any frequency in a normal subject. There is also a peak in the gain curve at 0.3–0.6 Hz (Fig. 7) which is not present for normal subjects or spastic patients. This peak indicates that the voluntary control system is underdamped and suggests that sudden movement should excite transient oscillations of the arm at the frequency of the peak (0.3–0.6 Hz). The irregularities at this frequency in the error spectrum (Fig. 5) and the coherence function (Fig. 6a) support this suggestion. This finding predicts that spectral analysis of involuntary athetoid movement should reveal a rhythmical component at 0.3–0.6 Hz.

In spite of the large inter-patient variation in the IEMG to elbow-angle phase characteristics, only small differences were measured in the target to elbow-angle characteristics. This indicates that the timing of the neural signals dispatched to the muscles by the CNS must have been adjusted to compensate for the changed muscle contraction characteristics, thus implying

that the CNS includes a mechanism to compensate for changes in mechanical load on muscle.

The impairment of voluntary control of arm movement measured in this study suggests that, even if it were possible to eliminate abnormal tone and involuntary movement in athetosis, the patient would still have a degree of disability due to difficulty in organizing and controlling volitional movement.

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