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Early stabilization of human posture after a sudden disturbance: influence of rate and amplitude of displacement*

H.C. Diener, J. Dichgans, F. Bootz, and M. Bacher

Neurologische Universitätsklinik, Abteilung Allgemeine Neurologie, Liebermeisterstr. 18–20, D-7400 Tübingen, Federal Republic of Germany

Summary. The functional role of short-, medium- and long-latency responses for the maintenance of upright posture was investigated in twenty healthy subjects standing on a platform which could be rotated in pitch around the subject's ankle joints. Tilting the platform toe-up evokes a stretch reflex in the triceps surae muscle (TS, latency 55–65 ms) and at higher speeds and amplitudes of platform displacement a medium-latency response (latency 108–123 ms). Both responses functionally destabilize posture, since they enforce the induced backward displacement of the body. Compensation of body displacement in this situation is achieved by a long-latency EMG response in the anterior tibial muscle (TA 130–145 ms). Platform movement toe-down elicits a rather small medium-latency response in TA (103–118 ms), but no short-latency response. A late compensatory response occurs in the triceps surae muscle (latency 139–170 ms). The mean latency of the late antagonistic EMG response was significantly shorter than that of a voluntary movement triggered by a somatosensory stimulus. Integrals of rectified EMG responses from the two muscles were linearly related to the amplitude and to a smaller degree to the velocity of platform displacement. The slope of this function (gain) varied depending on the direction of ankle displacement and the functional importance of the subsequent EMG responses. Destabilizing short- and medium-latency responses of the stretched muscle had a lower gain relative to amplitude than the late stabilizing response of the antagonist. This functionally adaptive modulation of gain was not seen in relation to the rate of platform displacement.

Key words: Human posture – Short-, medium-, long-latency responses

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Offprint requests to: H.C. Diener (address see above)

Introduction

The maintenance of upright human posture after an external disturbance requires the integration of afferent information from proprioceptors, vision, and the vestibular system, and the coordination of short-, medium- and long-latency responses with subcortical and voluntary postural control movements. In the case of minor disturbances applied to the body or the extremities the visco-elastic properties of the muscles, ligaments, and tendons may be sufficient to compensate for most of the imposed disturbance (Gurfinkel et al. 1974; Bizzi et al. 1978; Allum et al. 1982a). In addition, body inertia plays a major role, particularly at high frequencies. When the amplitude of an imposed displacement is increased, however, muscular activity then becomes increasingly more important for restoring the initial position.

Sudden disturbances through tilt about the ankle in a *standing subject* cause a number of clearly separable EMG responses of varying latencies in flexors and extensors of the lower leg (Nashner 1976; Allum and Büdingen 1979; Gottlieb and Agarwal 1979, 1980a; Allum et al. 1982b). The relationship of the latency and amplitude of these responses to the angle and velocity of the displacement of the supporting platform, although determining their functional significance has up to date not been examined. In the *sitting* man sudden torques about the ankle cause similar EMG responses of short (myotatic reflex) and medium latency (postmyotatic reaction) in the stretched muscle (Gottlieb and Agarwal 1979, 1980a, b; Gottlieb et al. 1981; Allum et al. 1982a). The "myotatic reflex" in the triceps surae and the later responses in both the triceps surae and the anterior tibial muscle demonstrate a linear relation to and high correlation with the rate of muscle stretch.

Tilt of the supporting platform toe-up stretches the triceps surae. Myotatic and postmyotatic

responses to stretch through tilt further destabilize posture whereas activity of the anterior tibial muscle stabilizes. The time-locked response in the anterior tibial muscle, although occurring somewhat later, tends to counterbalance the destabilizing medium-latency response in the triceps surae muscle (Allum and Büdingen 1979). Our experiments were conducted in an attempt to elucidate to what degree a setting according to functional demands facilitates or inhibits segmental and suprasegmental responses to a sudden external disturbance in a subject standing at rest. Our working hypothesis was that a high positive relationship in the amount of destabilizing EMG responses to the rate or amplitude of the imposed stretch, as in the experiments of Gottlieb and Agarwal, would be extremely unfavorable in that it would increasingly destabilize posture along with the increasing rate and amplitude of the rotating support. Functional demands in the standing subject would require a proportionally lower gain of destabilizing EMG responses in the stretched muscle and a higher gain of the EMG responses in the shortened antagonist, the functionally stabilizing muscle.

Methods

Subjects stood on a moveable force measuring platform. Strain gauges at the four corners measured forces perpendicular to the platform. The displacement of the *center of foot pressure* (CFP) in the anterior-posterior direction was computed from the differences of forces acting on the two anterior and the two posterior corners of the platform.[1] The subject's weight, which influences the static and dynamic portion of the measurement, was electronically compensated. The servo-controlled platform could be rotated suddenly in pitch about an axis aligned with the axis of the subject's ankle joint. The rate and amplitude of the platform movement could be varied independently; the rate between 5 and 100°/s, the amplitude up to 8°. The rate of motion of the platform was controlled by integrating the output of an accelerometer attached to the platform.

The EMGs of the anterior tibial (TA) and the triceps surae (TS = gastrocnemius + soleus) muscles were recorded with two surface electrodes placed 5.3 cm apart between the two heads of the gastrocnemius muscle. EMG signals were full wave rectified and bandpass filtered (1.6 Hz – 1 kHz). The platform movements, displacements of the CFP in the anterior-posterior direction, and angular displacements of head and hip were recorded for later analysis after multiplexing (rate 263 Hz/channel) through the direct channel of a tape recorder (Tandberg 115 G). EMG signals were stored on the two FM-channels.

Ten healthy subjects (aged 22–32) participated in the first two experiments. Subjects were instructed to maintain a comfortable upright posture with open eyes during the entire experiment. Their feet were positioned parallel to each other 4 cm apart. Subjects were not informed beforehand as to the time of onset, the direction, rate, and amplitude of a forthcoming platform displacement. In the first experiment the *rate of ramp movement* of the platform was varied between 10 and 100°/s, while the amplitude remained constant at 4°. The sequence of the different angular velocities of platform movements was randomized, as was also the direction of the platform displacement, i.e. 'toe-up' or 'toe-down'. Every combination of platform rate and direction was recorded at least eight times in order to make reliable averaging possible.

The *amplitude of platform movement* was varied in the second experiment between 2 and 8°. The rate of ramp movement remained constant throughout at 80°/s.

The displacements of CFP, head, and hip were averaged over eight runs (Nicolet 1070) and displayed on an X-Y-plotter. Latencies indicating the onset of EMG activity after the start of platform displacement were separately measured for each of the eight runs by visual identification with a digital cursor on the oscilloscope display of the averager.

In the third experiment we tried to investigate whether or not the late antagonistic EMG response in the anterior tibial muscle after a platform tilt toe-up is a voluntary response triggered by the sensory input to the foot when the platform is tilted. Ten healthy subjects participated in this experiment. The platform was tilted toe-down with an amplitude of 0.5°, a condition which in none of the cases evoked a short- or medium-latency response in the anterior tibial muscle. Subjects were instructed to perform a voluntary dorsiflection of the foot as rapidly as possible when they perceived the beginning of the platform tilt. After a training period with four platform tilts eight runs were performed. The mean latencies and intraindividual standard deviations indicating the beginning of the EMG activity in the anterior tibial muscle were measured and compared to the latency of the antagonistic long-latency response of this muscle after the platform was tilted 'toe-up' (4°; 50°/s).

In the fourth experiment we measured the integrated rectified EMG (IEMG) activity of both muscles. Twenty subjects participated in this experiment. Platform movements were controlled by a microprocessor system which varied randomly the velocity, the amplitude and the direction of platform movements. Time intervals between individual trials also varied randomly between 2 and 7 s. For each stimulus condition the four corresponding runs were averaged. Each trial was repeated with another sequence of platform rates and amplitudes. The average EMGs from each stimulus condition and subject were shown on the screen of the interactive graphics terminal of the computer (IN 110). The latencies of the short-, medium- and long-latency responses were manually marked by a cursor. The computer calculated the integrated EMG within the next 40 ms of activity for the short- and medium-latency response. The IEMG of the long-latency response was calculated within a time interval of 150 ms and then divided by 3.75 in order to allow a comparison with the earlier responses.

Data were then normalized in order to allow for a comparison between subjects. For this purpose we separately averaged all of the integrated EMG activities of each muscle across the entire experiment for each subject. This mean was given an arbitrary value of 50%. All the individual EMG integrals were then recalculated on this basis.

We performed a regression analysis in order to obtain the mean slopes of the functions relating IEMG to the rate of displacement as well as to the amplitude of platform displacement. This calculation was separately done for each EMG response. From the 200 individual measurements (20 subjects × 2 trials × 5 stimulus velocities respectively amplitudes) we additionally calculated the coefficient of correlation.

In order to get an estimate of the torque about the ankle joint produced by a certain amount of IEMG we performed an additional experiment. In this experiment subjects were asked to lean actively forward and backward. Subjects had to adjust their body position such that the CFP which was displayed on an

1 The CFP is not equivalent to the center of gravity, as it includes dynamic forces, the contribution of which increases with frequency

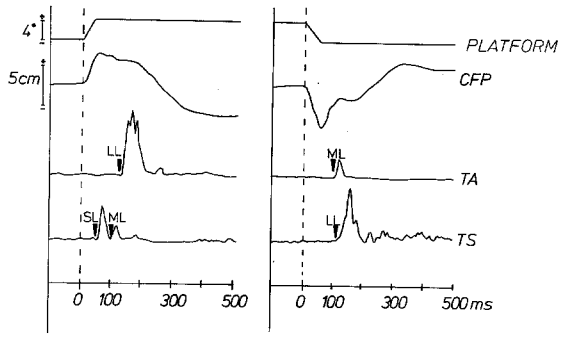

Fig. 1. Average displacement of platform and CFP after a sudden displacement of the measuring platform toe-up (left) or toe-down (right) with a velocity of 60°/s. EMG responses in the anterior tibial (TA) or triceps surae muscle (TS) are indicated by arrows. SL = short-latency response, ML = medium-latency response, LL = long-latency response. Average from eight runs. Plus means a forward shift of the CFP

oscilloscope in front of the subject, matched a target signal set, for example, to an equivalent of 2 cm displacement of the CFP. The same was repeated for different target signals equivalent to displacements ranging between 0.5 and 4 cm. We then calculated the integrated EMG of the actively shortened muscle within a time interval of 250 ms. This was only done if the antagonist muscle was relaxed.

The integrated EMG of the triceps surae and the anterior tibial muscle was then related to the torque acting on the ankle joint (which was also measured by the platform). The coefficient of correlation for torque versus integrated EMG was 0.925 for the triceps surae and 0.818 for the anterior tibial muscle. The slope of the function was 0.159 for TA and 0.191 for the TS (Y intercept –0.145 and –0.111, respectively). We had evidence indicating that the assumption of a linear relationship between IEMG and torque at the ankle joint was valid (Hof and van den Berg 1977). The two slope coefficients indicate that a unit change in EMG for each muscle produces roughly the same unit change in torque about the ankle, but in opposite directions.

Results

a) EMG responses

Sudden upward displacements of the platform, which stretch the triceps surae muscle, caused a short-latency reflex in this muscle with a latency of between 55 and 65 ms (Fig. 1, left side). The second medium-latency response in the triceps surae had a latency of 108–123 ms. In the antagonistic anterior tibial muscle the first activity appeared after 130–145 ms. It was sometimes followed by a tonic contraction of the anterior tibial muscle at a lower level of activity. During the first part of the platform movement the pressure on the forefoot increases and leads to a forward displacement of the center of foot pressure (CFP). This first segment is mainly due to the inertia of the foot and to the passive viscoeleastic resistance of the muscles and ligaments stretched. Correlated with the activity of the anterior tibial muscle, the CFP shifts backwards, since this muscle activity increases the pressure of the heels on the measuring platform.

Platform displacements 'toe-down' failed to evoke a short-latency reflex in the anterior tibial muscle, a phenomenon which was earlier observed by others (Allum and Büdingen 1979; Gottlieb and Agarwal 1979). A rather small medium-latency response in this muscle could be seen at a latency of 103–118 ms and again a stabilizing long-latency response in the antagonist muscle after 139–170 ms (Fig. 1, on the right). The displacement of the CFP differs from that after platform movement toe-up. The "plateau" of the CFP in the latter situation was not observed after platform movements toe-down. The forward shift of the CFP after 150 ms is due to the activity of the triceps surae muscle.

b) EMG latency in relation to rate and amplitude of platform displacement

The onset-latency of the stretch reflex in the triceps surae muscle depends on the rate of the platform displacement below 20°/s. Above 20°/s and up to 100°/s, the highest value tested, the short-latency response of the triceps surae muscle occurred at an average of 55 ms (Fig. 2A). At a rate of 80°/s the latency was independent of the amplitude of the stretch between 1 and 8° (Fig. 2C). With a rate of 10 and 20°/s the reflex response occurred only in 50–70% of our subjects. With higher angular velocities subjects invariably showed a short-latency response independent of amplitude. With increasing stimulus velocity and amplitude the medium-latency response in the triceps surae muscle appeared in an increasing number of subjects (max. 70%). The velocity threshold for this response was much higher (60°/s) than that for the stretch reflex (10°/s, Fig. 2A).

Rotating the platform toe-down evoked a medium-latency response in the anterior tibial muscle in 80% of the subjects when the rate of platform movement exceeded 40°/s. The frequency of this response was further increased whenever the amplitude of displacement was increased. The latency of this response was independent of the rate at rates greater than 20°/s and independent of the amplitude of platform rotation at 80°/s (Fig. 2B, D). The mean latency of the late activity in the shortened triceps surae muscle was also independent of the amplitude of the platform displacement (Fig. 2D). The latency seemed to be a monotonically decreasing

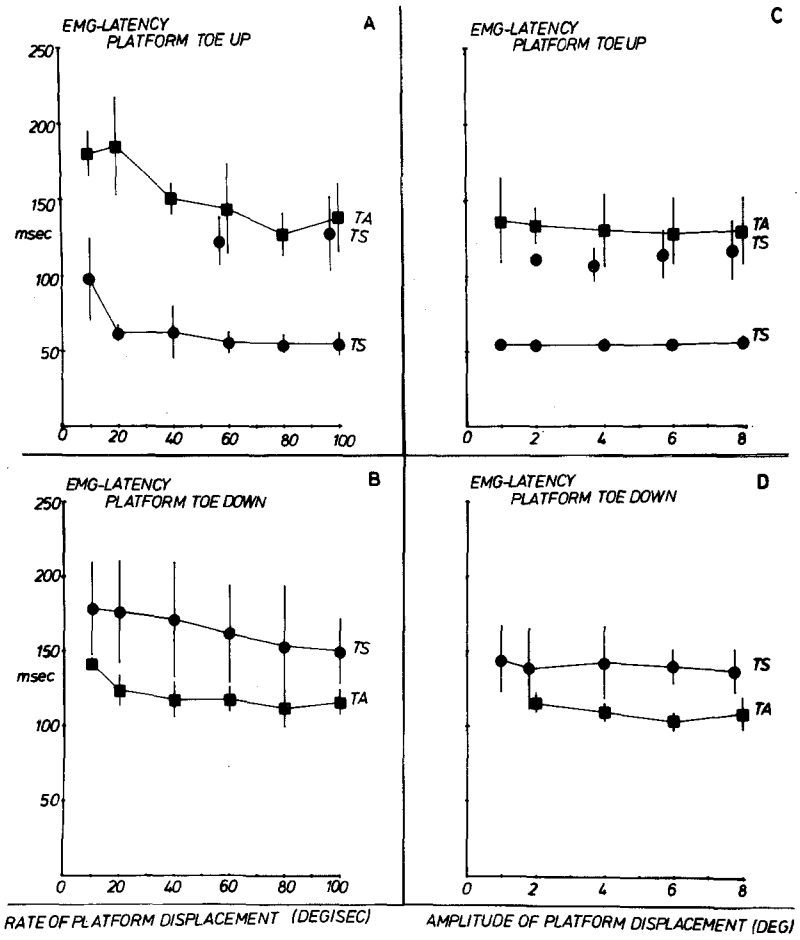

Fig. 2A–D. Mean latencies and standard deviations of EMG responses after sudden displacements of the platform up **A, C,** and down **B, D.** After a platform rotation toe-up a stretch reflex (dark circles) and a medium latency response (dark circles) can be observed in the triceps surae muscle. The stabilizing long-latency response in the anterior tibial muscle is indicated by a black square. **A, B** amplitude = 4°, **C, D** velocity of displacement = 80°/s. After platform rotation downward the medium latency response in the anterior tibial muscle (TA) is indicated by squares, the long latency response in the triceps surae muscle (TS) by black circles

function of platform velocity, however, an analysis of variance showed that only the latencies for 10 and 20°/s rates were significantly larger than the others. In contrast to medium-latency responses, the antagonistic late responses were evoked in 90% of the subjects from 40°/s and 4° displacement on.

c) Postural response versus voluntary action

The mean latency of the long-latency response in the anterior tibial muscle (= 121.9 ms) after a platform tilt toe-up was significantly shorter than the fastest voluntary action of the same muscle (159.5 ms, t-test). The mean standard deviation across the eight trials was also much smaller in the first than in the second condition (8.7 versus 40.9 ms). Both results were consistent in all single subjects. While the voluntary dorsiflection of the foot was practised before the eight runs, subjects were not exposed to platform tilts 'toe-up' prior to the actual tests.

d) Integrated EMG in relation to rate and amplitude of platform displacement (Table 1)

The integrated EMG activity was linearly related to the amplitude of the platform displacement. In agreement with Gottlieb and Agarwal (1979, 1980a) we also observed a linear relationship between the integral of EMG activity on the one hand and the rate of platform motion on the other. The slope of the function relating the two variables, EMG activity and rate, designated "gain" by Gottlieb and Agarwal (1979), characterizes the input-output relation of the short-, medium- and long-latency EMG responses and helps to interpret their possible functional significance. The relationship was as follows: the steeper the slope, the higher the gain, the stronger the torque with increasing rate and amplitude of platform movement.

We calculated the regression lines separately for each of the three EMG responses across our 20 subjects. They are depicted in Figs. 3 and 4 for the relation between integrated EMG and amplitude of

Table 1. Regression analysis for the relation between rate of platform displacement (20°/s to 100°/s, amplitude 4°) and integrated EMG activity (N = 200)

Muscle	Platform	y = mx + b	r	p(r)
TS (SL)	up	y = 0.379x + 23.17	0.379	$p<0.001$
TS (ML)	up	y = 0.480x + 12.05	0.497	$p<0.001$
TA (LL)	up	y = 0.468x + 56.64	0.485	$p<0.001$
TA (ML)	down	y = 0.127x + 7.46	0.238	$p<0.001$
TS (LL)	down	y = 0.082x + 45.87	0.096	n. s.

Regression analysis for the relation between amplitude of platform displacement (2–6°, velocity 80°/s) and integrated EMG activity (N = 200)

Muscle	Platform	y = mx + b	r	p(r)
TS (SL)	up	y = 0.01x + 57.20	0.010	n. s.
TS (ML)	up	y = 9.79x + 9.26	0.467	$p<0.001$
TA (LL)	up	y = 20.15x + 1.09	0.771	$p<0.001$
TA (ML)	down	y = 2.59x + 8.04	0.203	$p<0.001$
TS (LL)	down	y = 4.64x + 38.00	0.255	$p<0.001$

SL = Short-latency response; ML = Medium-latency response; LL = Long-latency response

platform displacement and give an impression of the main trend and the interindividual variance.

Because of our normalization procedure the slope of the functions (gain) may be compared between the two muscles. The average functional characterization for each EMG response (short-, medium- and long-latency response) under each of the four conditions can be seen from Table 1. The table displays the mean regression coefficients, the coefficients of correlation from the individual values (N = 200) and the significance of the coefficient of correlation.

Relating rate of platform displacement and EMG resulted in a positive slope and significant coefficients of correlation in the destabilizing EMG responses as well as in the stabilizing long-latency response when the platform was tilted toe-up. The slope of the function was nearly identical for all three responses, thus, disproving our hypothesis. The same notion holds true for the comparison of the two functions when the platform is moved 'toe-down'. The posture-stabilizing response in the triceps surae muscle showed no relationship to the stimulus and had an even lower slope than the destabilizing medium-latency response.

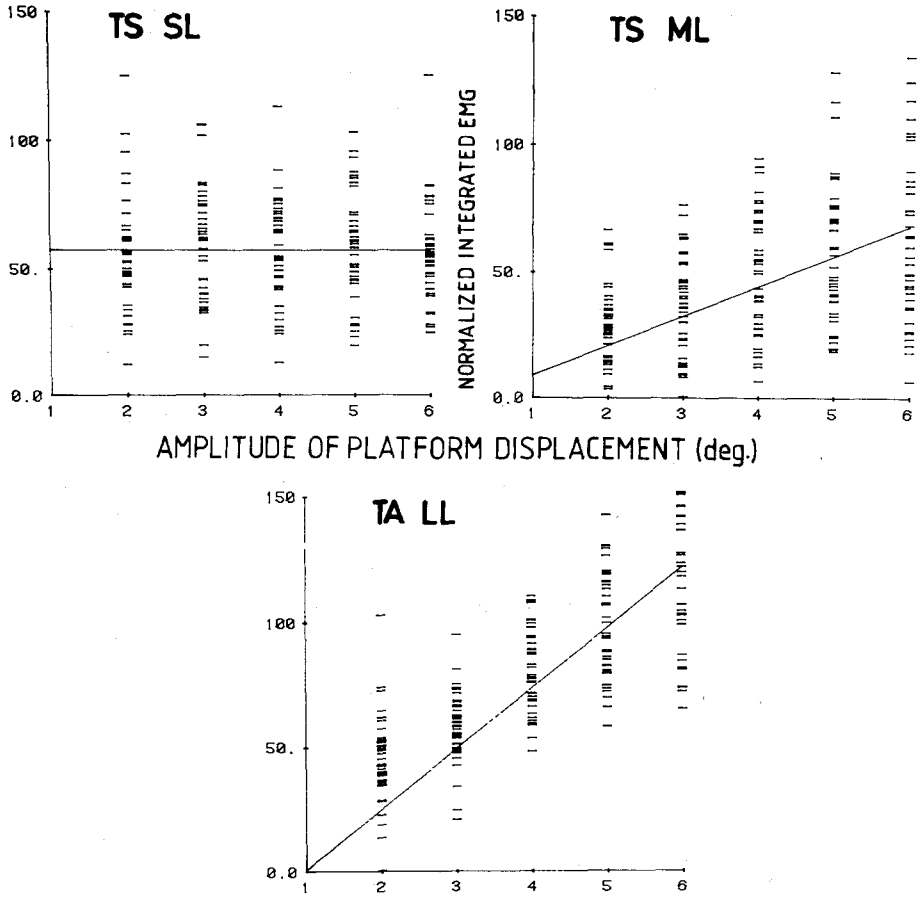

Fig. 3. Normalized integrated EMG responses (ordinate) of the stretch reflex (SL) and the medium latency response (ML) from the triceps surae muscle (top) and of the long-latency response (LL) in the anterior tibial muscle (bottom) after a platform tilt toe-up with varying amplitudes of platform tilt (abscissa). The solid lines are linear regression curves. The slope of the function relating integrated EMG and stimulus amplitude is steeper for the posture stabilizing response (LL) than for the destabilizing SL and ML responses

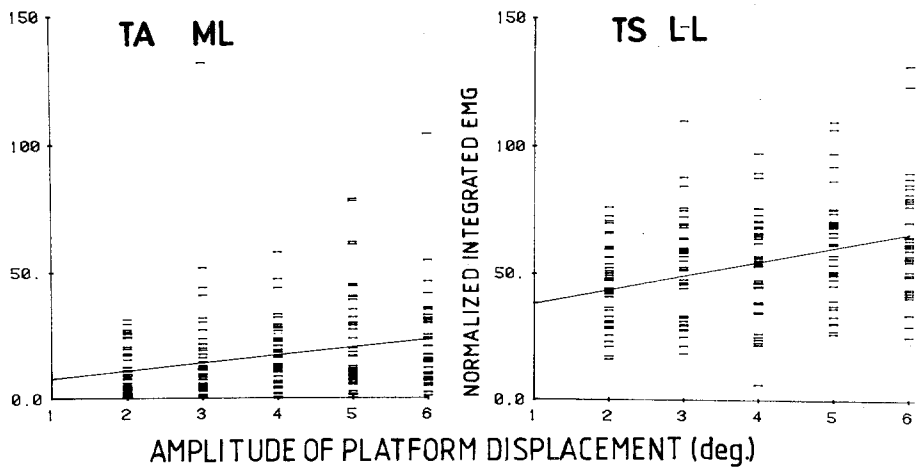

Fig. 4. Normalized integrated EMG responses of the medium-latency response (ML) from the anterior tibial muscle and the long-latency response (LL) from the triceps surae muscle after platform tilts toe-down. The solid lines are linear regression curves

On the other hand we were able to confirm our hypothesis when we related IEMG and amplitude of platform displacement (Figs. 3 and 4). The stretch reflex was unrelated to the amplitude of stretch. The gain of destabilizing EMG responses was invariably lower than that of the stabilizing long-latency response irrespective of the direction of platform displacement.

Discussion

Sudden angular displacements of the support caused by the tilting of the platform cannot entirely be counterbalanced by the viscoelastic properties of muscles and inertia of the body, but require muscle activity. Short-, medium-, and long-latency responses in cooperation with intersegmental corrective responses constitute the early phase of muscular activity.

Segmental stretch reflex

Tilting the platform 'toe-up' evokes a segmental – short latency – stretch reflex in the triceps surae muscle with a mean latency of 55 ms. Reflex latencies between 35 and 56 ms were measured by others using the same method or by pushing a standing subject (Elner et al. 1972; Litvinsev 1973; Allum and Büdingen 1979; Chan et al. 1979; Gottlieb and Agarwal 1979; Bussel et al. 1980; Kearney and Chan 1981). The large range of latencies is for the most part due to the differences in measurement technique. We measured with respect to the trigger stimulus, others with respect to the displacement of the foot. The increase due to mechanical delay in our experiment was 10 ms. When the stimulus amplitude was small, the stretch reflex was monophasic and had a duration of 20 ms. We found no correlation between amplitude of displacement and latency. This is probably due to the rather high velocity of displacement used (80°/s).

The latency of the stretch response in the triceps surae muscle reaches its minimum of 55 ms at a 20°/s rate of stretch. Lower stretch velocities lead to an increased delay and lower probability of reflex occurrence. At 10°/s the average latency increases to 98 ms and the probability falls to 70%. In the sitting man the threshold for the stretch reflex is higher (between 40 and 80°/s, Elner et al. 1972; Gottlieb and Agarwal 1979). Upright stance lowers the threshold of the stretch reflex, probably by descending tonic activity to the alpha-motoneuron. This modification of the excitability of the spinal reflex arc is apparently inaccessible to voluntary control (Burke and Eklund 1977; Burke et al. 1980).

As have others, we too did not observe a short-latency reflex in the anterior tibial muscle (Allum and Büdingen 1979; Gottlieb and Agarwal 1979). In the case of standing subject, however, a velocity threshold for the reflex exceeding 100°/s cannot be excluded due to the limited range of action of our equipment. When sitting, stretch velocities of up to 400°/s were not able to evoke a short-latency reflex in the anterior tibial muscle (Gottlieb and Agarwal 1979); a barely detectable response was obtained with a stretch velocity of up to 800°/s (Chan and Kearney 1982).

During voluntary contraction, however, passive displacement elicits a stretch reflex in the anterior tibial muscle (Iles 1977). Similarly a stretch response is obtained in this muscle without preactivity if subjects are warned beforehand (Burke et al. 1980). This change in the excitability of the stretch reflex cannot be explained by changes in fusimotor activity

since the dynamic response of spindle endings is not altered (Burke et al. 1980). In our experiments the time intervals between successive trials and tilting directions varied randomly. Our subjects, therefore, were not able to anticipate the next stimulus.

Medium-latency response

The responses after 110–120 ms correspond to the "functional stretch reflex" of Melvill Jones and Watt (1971) or the "postmyotatic reaction" of Gottlieb and Agarwal (1980a). This EMG response could be evoked in only 50% of the subjects. Since the contraction of the stretched muscle functionally destabilizes posture under our experimental condition, this response may have been suppressed in the remaining 50%. This assumption is supported by the finding of Nashner (1976), who observed a rapid adaptation of this response when he shifted from a linear displacement backwards, in which case TS stabilizes, to platform tilt 'toe-up'. Subjects who exhibited this medium-latency response, however, did not show adaptation in our experiment. The difference to the results of Nashner (1976) may be due to the higher velocity of displacement employed in our experiments.

The origin of the medium-latency response is unclear. In agreement with the findings of authors who studied stretch responses in arm and finger muscles (Philipps 1969; Marsden et al. 1973, 1976; Lee and Tatton 1975) we feel that a transcortical loop may be considered, but thus far cannot be proven.

Preloading is necessary to evoke an analogous resonse in arm muscles and muscles of the lower leg in sitting subjects, but is not a prerequisite for such a response when the subjects are standing. The triceps surae is usually slightly loaded and, therefore, active since the body's center of gravity normally projects anterior to the ankle angle axis when subjects are standing (Thomas and Whitney 1959). The response is, however, also seen in the unloaded anterior tibial muscle with an unexpected toe-down displacement. Influences such as expectation or a special instruction (Gottlieb and Agarwal 1980a) were excluded by the experimental paradigm. It may be concluded that the task of standing per se comes about with the appropriate setting of the spinal cord connectivity and its supraspinal control centers.

Long-latency response

This response was exclusively and almost invariably obtained in the antagonist of the stretched muscle. When the supporting platform is tilted, this EMG response is the only one which functionally stabilizes posture, since it counteracts the body displacement forward or backward. The late stabilizing response of the antagonist muscle is specific to upright posture since it is not found when the triceps surae is stretched in a supine position (Chan and Kearney 1982) or when sitting (Gottlieb and Agarwal 1980a; Allum et al. 1982a, b). We were unable to determine whether this response is present with linear displacement. Recordings of Nashner and Grimm (1978) seem to indicate that this is not the case.

The long latency response of the antagonist muscle in its stabilizing effect is supported by body inertia when the platform is tilted. With linear motion, however, body inertia has a destabilizing effect. In addition to the muscular effects of the long-latency response and inertia, the counterphase oscillations of the different body segments also help to stabilize posture.

With regard to the long latency it may be considered whether this response is voluntary. Although well-trained subjects may finally initiate a voluntary contraction of leg muscles within 90–130 ms after a kinesthetic or acoustic stimulus (Iles 1977; Chan et al. 1979) postural reactions, as in our situation, could clearly be differentiated from these voluntary responses. We observed that some members of our laboratory research team were able to shorten their voluntary reaction-time for dorsiflection of the foot from 150 ms to 90 ms within a training period of some days. The latencies of the long-latency responses were unchanged, even after several months of experiments.

The nature of these late responses remains unclear. Vestibulospinal reflexes (Nashner 1973) are unlikely, since one subject without labyrinthine function (due to bilateral 8th nerve section) showed these late responses with a normal latency. Thus far it remains unclear whether this response is due to spindle afferences from the stretched muscle, from the joint or from pressure receptor afferences. That spindle unloading is the triggering source of information from the shortened muscle is unlikely since, while standing quietly, the discharge of Ia-afferents is minimal (Burke and Eklund 1977).

Involuntary postural adjustments can also be observed in leg muscles before voluntary arm movements are executed (Bouisset and Zattava 1981; Cordo and Nashner 1982). This and the clinical observation of a disturbance of postural reactions in patients with diseases of the basal ganglia or cerebellar diseases, in whom voluntary movements of the arms may be for the greater part unaffected, although postural reflexes are absent, delayed or

even enhanced (Traub et al. 1980; Dichgans and Mauritz 1982), support the concept of a largely subconscious organization of postural motricity.

Functional significance of involuntary EMG responses after platform tilting

The functional role of short, medium, and late responses is poorly understood. The assumption that the stretch reflex is only a length regulating mechanism and compensates for changes in external mechanical load could not be confirmed in man and monkeys (Crago et al. 1976; Bizzi et al. 1978). Its mechanical significance under our experimental conditions is small. If prominent, it would add passive destabilization. Houk (1978, 1979) assumed that the stretch reflex in response to stimulation of spindles and tendon organs regulates muscle stiffness (the ratio of force change to length change) and therefore, linearizes the nonlinear mechanical response of a muscle to length changes. With platform tilt, increased muscle stiffness would aid passive destabilization, a fact which, if true, would illustrate the occasional maladaptivity of segmental reflexes.

Medium- and long-latency responses are generally considered to be of greater significance for the compensation of a disturbance of an actively contracting muscle (Hammond 1960). The "gain" of this "servo reaction" is proportional to the initial load; it is nearly zero in a relaxed muscle (Marsden et al. 1977). In the *sitting man* a high gain (integrated EMG versus rate of stretch) for both segmental reflexes and "postmyotatic responses" was observed by Gottlieb and Agarwal (1979, 1980). In agreement with the above results we found in the *standing man* a positive gain of short-, medium-, and long-latency responses when we compared integrated EMG and rate of platform displacement. Although short- and medium-latency responses to the muscle stretching destabilize posture with platform tilt, whereas long-latency responses tend to stabilize it, the increase in IEMG activity with an accompanying increase in the velocity of the platform tilt is similar in both stabilizing and destabilizing responses. Evidently, the velocity of platform tilt per se is not a critical variable for the passive displacement of the body. The equally increasing gain of the stabilizing long-latency response compensates for the increase of the short- and medium-latency responses.. This behavour could possibly account for the increasing stabilizing effect of inertia with increasing velocity.

In agreement with our hypothesis, we found a much higher gain in the long latency response as compared to the short- and medium-latency responses when the amplitude of the platform displacement was increased. In this situation the antagonistic response must not only compensate for the destabilizing effects of the earlier EMG responses, but it must also restore upright position after passive displacements in the forward or the backward direction. Admittedly the gain of the medium- and long-latency response is higher when the platform moves 'toe-up' as compared to moving 'toe-down'. This probably reflects th subjective experience that it is much easier to compensate a displacement 'toe-down' than 'toe-up'. The more difficult a task the higher is the amount of coactivation of TS and TA.

The relatively high gain of the response of the triceps surae to stretch at first glace seems to contradict the interpretation given so far. One of the reasons for the relatively high gain of the medium latency response in TS might be that the range of possible dorsiflection from the neutral position of the ankle joint is much smaller than that for plantarflection. In the case of an 8 degree stretch toe-up, as in our experiment, a reflex opposing this stretch might be necessary to protect muscle and tendons from being overstretched. The exclusive comparison of gains within one muscle overlooks that it is the proportion of gains in agonist and antagonist that determines the biomechanical consequences.

The fact that the late stabilizing EMG response in the antagonist of the stretched muscle shows a linear relationship to rate and amplitude of platform displacement with a positive slope indicates a "servo-like" reaction rather than a "triggered response" (Marsden 1976; Tatton and Bawa 1979; Chan and Kearney 1982). Triggering might be suspected for the onset, but responses require a certain rate and amplitude of the disturbance to reach a minimal latency.

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References

Allum JHJ, Büdingen HJ (1979) Coupled stretch reflexes in ankle muscles: An evaluation of the contribution of active muscle mechanism to human posture stability. Prog Brain Res 50: 185–195

Allum JHJ, Mauritz KH, Vögele H (1982a) Stiffness regulation provided by short-latency reflexes in human triceps surae muscles. Brain Res 234: 159–164

Allum JHJ, Mauritz KH, Vögele H (1982b) The mechanical effectiveness of short latency reflexes in human triceps surae muscles revealed by ischemia and vibration. Exp Brain Res 48: 153–156

Bizzi E, Dev P, Morasso P, Polit A (1978) Effect of load disturbances during centrally initiated movements. J Neurophysiol 41: 542–556

Bouisset S, Zattava M (1981) A sequence of postural movements precedes voluntary movement. Neurosci Lett 22: 263–270

Burke D, Eklund G (1977) Muslce spindle activity in man during standing. Acta Physiol Scand 100: 187–199

Burke D, McKeon B, Skuse MF, Westerman RA (1980) Anticipation and fusimotor activity in preparation for a voluntary contraction. J Physiol (Lond) 306: 337–348

Bussel B, Katz R, Pierrot-Deseilligny E, Bergego C, Hayat A (1980) Vestibular and proprioceptive influence on the postural reactions to a sudden body displacement in man. In: Desmedt JE (ed) Spinal and supraspinal mechanisms of voluntary motor control and locomotion. Karger, Basel, pp 310–322

Chan CWY, Melvill Jones G, Kearney RE, Watt DGD (1979) The "late" electromyographic response to limb displacement in man. I. Evidence for supraspinal contribution. Electroencephalogr Clin Neurophysiol 46: 173–181

Chan CWY, Kearney RE (1982) Is the functional stretch reflex servo-controlled or preprgrammed? Electroencephalogr Clin Neurophysiol 53: 310–324

Cordo PJ, Nashner LM (1982) Properties of postural adjustments associated with rapid arm movements. J Neurophysiol 47: 287–302

Crago F, Houk JC, Hasan Z (1976) Regulatory actions of human stretch reflex. J Neurophysiol 5: 925–935

Dichgans J, Mauritz KH (1982) On the pathophysiology of postural imbalance in cerebellar patients. In: Honrubia V, Brazier MAB (eds) Nystagmus and vertigo. Academic Press, New York, pp 179–189

Elner AM, Popov KE, Gurfinkel VS (1972) Changes in stretch reflex system concerned with the control of postural activity of human muscle. Agressologie 13D: 19–23

Gottlieb GL, Agarwal GC (1979) Response to sudden torques about ankle in man. I. Myotatic reflex. J Neurophysiol 42: 91–106

Gottlieb GL, Agarwil GC (1980a) Response to sudden torques about ankle in man. II. Postmyotatic reactions. J Neurophysiol 43: 86–101

Gottlieb GL, Agarwal GC (1980b) Response to sudden torques about ankle in man. III. Suppression of stretch-evokated responses during phasic contraction. J Neurophysiol 44: 233–246

Gottlieb GL, Agarwal GC, Jaeger RJ (1981) Response to sudden torques about ankle in man. IV. A functional role of alpha-gamma linkage. J Neurophysiol 46: 179–190

Gurfinkel VS, Lipshits MI, Popov KY (1974) Is the stretch reflex the main mechanism in the system of regulation of the vertical posture of man? Biofizika 19: 744–748

Hammond PH (1960) An experimental study of servo action in human muscular control. 3rd International Conference on Medical Electronics, pp 190–199

Hof AL, van den Berg J (1977) Linearity between the weighted sum of the EMGs of the human triceps surae and the total torque. J Biomech 10: 529–539

Houk JC (1978) Participation of reflex mechanisms and reaction-time processes in the compensatory adjustments to mechanical disturbances. In: Desmedt JE (ed) Cerebral motor control in man: Long loop mechanisms. Karger, Basel, pp 193–215

Houk JC (1979) Motor control processes: New data concerning motoservo mechanisms and a tentative model for stimulus-response processing. In: Talbott RE, Humphrey DR (eds) Posture and movement. Raven Press, New York, pp 231–242

Iles JF (1977) Response in human pretibial muscles to sudden stretch and nerve stimulation. Exp Brain Res 30: 451–470

Kearney RE, Chan CWY (1981) Interlimb reflexes evoked in human arm muscles by ankle displacement. Electroencephalogr Clin Neurophysiol 52: 65–71

Lee RG, Tatton WG (1975) Motor responses to sudden limb displacement in primates with specific CNS lesions and in human patients with motor system disorders. Can J Neurol Sci 2: 285–293

Litvintsev AI (1973) Mechanisms of man's vertical posture control. Agressologie 14: 17–22

Marsden CD, Merton PA, Morton HB (1973) Is the human stretch reflex cortical rather than spinal? Lancet i: 759–761

Marsden CD, Merton PA, Morton HB (1976) Stretch reflex and servo action in a variety of human muscles. J Physiol (Lond) 259: 531–560

Marsden CD, Merton PA, Morton HB, Hallett M, Adam J, Rushton DN (1977) Disorders of movement in cerebellar disease in man. In: Rose F (ed) Physiological aspects of clinical neurology. Blackwell Sci Publ, Oxford, pp 179–199

Melvill Jones G, Watt DGD (1971) Observations on the control of stepping and hopping movements in man. J Physiol (Lond) 219: 709–727

Nashner LM (1973) Vestibular and reflex control of normal standing. In: Stein RB, Pearson KG, Smith RS, Redford JB (eds) Control of posture and locomotion. Plenum Press, New York, pp 291–308

Nashner LM (1976) Adapting reflexes controlling the human posture. Exp Brain Res 26: 59–72

Nashner LM, Grimm RJ (1978) Analysis of multi loop dyscontroles in standing cerebellar patients. In: Desmedt JE (ed) Cerebral motor control in man: Long loop mechanisms. Karger, Basel, pp 300–319

Phillips CG (1969) Motor apparatus of the baboon's hand. Proc R Soc 173: 141–174

Tatton WG, Bawa P (1979) Input-output properties of motor unit responses in muscle stretched by imposed displacements of the monkey wrist. Exp Brain Res 37: 439–457

Thomas DP, Whitney RJ (1959) Postural movements during normal standing in man. J Anat 93: 524–529

Traub MM, Rothwell JC, Marsden CD (1980) Anticipatory postural reflexes in Parkinson's disease and other akinetic-rigid syndromes and in cerebellar ataxia. Brain 103: 393–412

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