J. Physiol. (1984), **357**, *pp.* 109–125
With 10 *text-figures*
Printed in Great Britain

CORRECTIVE REACTIONS TO STUMBLING IN MAN: NEURONAL CO-ORDINATION OF BILATERAL LEG MUSCLE ACTIVITY DURING GAIT

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(*Received 1 March 1984*)

SUMMARY

1. Electromyogram (e.m.g.) responses of lower leg muscles, and corresponding movements were studied following a perturbation of the limb during walking, produced by either (a) a randomly timed, short acceleration or decelerating impulse applied to the treadmill, or (b) a unilateral triceps surae contraction induced by tibial nerve stimulation.

2. Bilateral e.m.g. responses following the perturbation were specific for the mode of perturbation and depended on the phase of the gait cycle in which the perturbation occurred. Treadmill deceleration evoked a bilateral tibialis anterior activation; acceleration evoked an ipsilateral gastrocnemius and contralateral tibialis anterior activation (latency in either condition and on both sides was 65–75 ms, duration about 150 ms).

3. Tibial nerve stimulation at the beginning of a stance phase, was followed by an ipsilateral tibialis anterior activation; during the swing phase it was followed by an ipsilateral tibialis anterior and contralateral gastrocnemius activation (latency about 90 ms, duration about 100 ms). These patterns differed from the response seen after a unilateral displacement during static standing, which evoked a bilateral tibialis anterior activation.

4. These early responses were in most cases followed by late ipsilateral responses, but the e.m.g. pattern of the next step cycle was usually unchanged, or affected only at its onset.

5. The e.m.g. responses were unaltered by ischaemic nerve blockade of group I afferents, by training effects or by pre-warning of the onset of perturbation (randomly or self-induced).

6. Despite the different e.m.g. responses following a perturbation during gait, the same basic functional mechanism was obviously at work: the early ipsilateral response achieved a repositioning of the displaced foot and leg, while the early contralateral and late ipsilateral responses provided compensation for body displacement.

7. It is suggested that the e.m.g. responses may be mediated predominantly by peripheral information from group II and group III afferents, which modulate the basic motor pattern of spinal interneuronal circuits underlying the respective motor task.

INTRODUCTION

Regulation of bipedal gait shows fundamental differences to that of quadrupedal locomotion, requiring specific neuronal mechanisms to maintain the body in an upright position during locomotion. The spinal stretch reflex has been suggested as functioning so as to adapt the pre-programmed leg muscle motor patterns to the terrain encountered (Dietz, Schmidtbleicher & Noth, 1979) and to compensate for unexpected changes in ground level (Noth & Dietz, 1979). While this neuronal mechanism explains quick unilateral patterns of reflex activity in leg extensor muscles, a more complex, bilateral co-ordination of leg muscle activation is needed to maintain the body equilibrium when gait is disturbed by an obstacle.

When a subject stands on an unstable surface, a spinal reflex mechanism rapidly compensates for a unilateral foot displacement by producing bilateral activation of tibialis anterior muscles (Dietz & Berger, 1982). Such an activation of homologous muscle of both legs is not surprising, since during normal stance upright posture was found to be regulated by an activation of homologous leg muscles (Bonnet, Gurfinkel, Lipchits & Popov, 1976). A different mechanism might, however, be expected during gait, when homologous leg muscles are being activated reciprocally.

The aim of this study was to analyse the bilateral co-ordination of leg muscle activity after perturbations at different points of the step cycle. It will be shown that bilateral responses of antagonistic leg muscles are more complex during gait than in upright stance, and more complex than those seen in quadrupedal locomotion in the cat; they depend on the phase of the step cycle in which the leg is displaced.

METHODS

General procedures and recording methods

Electromyogram (e.m.g.) activity was recorded from medial gastrocnemius and tibialis anterior muscles in both legs using surface electrodes in fifteen normal volunteers aged between 20 and 39 years during different conditions of walking on a treadmill. The treadmill consisted of a 160 × 43 cm bearing surface and a toothed-belt power transmission.

1. The volunteers were subjected to random disturbances at various phases of the step cycle. The motive power of the treadmill consisted of a d.c. motor with a gear unit, a tachometer generator, and a transistor-servo-amplifier in a four-quadrant system. This allowed fast acceleration and deceleration of the treadmill; the impulse capacity of the amplifier (Woodway: MTR/70/25; Weil, F.R.G.) was 12750 W for 1 s, and when this was applied, the treadmill velocity for a walking adult, changed from 4 to 9·0 km/h in 70 ms or from 4 to −1·5 km/h in 60 ms. Electrical switches in the shoes provided trigger pulses at different phases of step cycle. The ankle joint movements were indicated by a goniometer fixed at the lateral aspect of the foot and leg.

2. The volunteers were examined while walking on the treadmill at speeds equivalent to 2 and 4 km/h. At random intervals, the tibial nerve of one leg was stimulated at supramaximal intensity in one of the following phases of a step cycle: contact by the heel; contact by heel and ball, and by ball alone during stance; at the beginning of swing, with contact by neither by heel nor ball. The phases were indicated by signals from the shoe switches. For 1 and 2 the experimenter determined, in which step cycle and at which moment in the step cycle the stimulus was delivered. Each trial consisted of ten stimuli delivered at a distinct phase of gait. The moment of stimulation was used as a trigger for averaging the biomechanical and e.m.g. responses.

3. As a control, the walking experiments were repeated at low speed, but in the middle of the stance or swing phase of randomly chosen paces, a supramaximal electrical stimulus was delivered to the dorsum of one foot. Again, each trial consisted of ten stimuli. For two subjects the tibial nerve was stimulated at defined phases of gait, but with submaximal pulses which failed to evoke a visible muscle contraction.

4. In other experiments the subjects were examined while standing with each leg on a separate see-saw. The see-saws consisted of platforms with a curved base (see Fig. 8) which were placed on two separate force-measuring platforms (Kistler; Winterthur, Switzerland). During balancing, the tibial nerve of one leg was stimulated by single pulses at supramaximal intensity (stimulus duration 2 ms). The resulting tibialis anterior and gastrocnemius e.m.g. responses, and the body and see-saw displacements were analysed by methods recently described (Dietz & Berger, 1982).

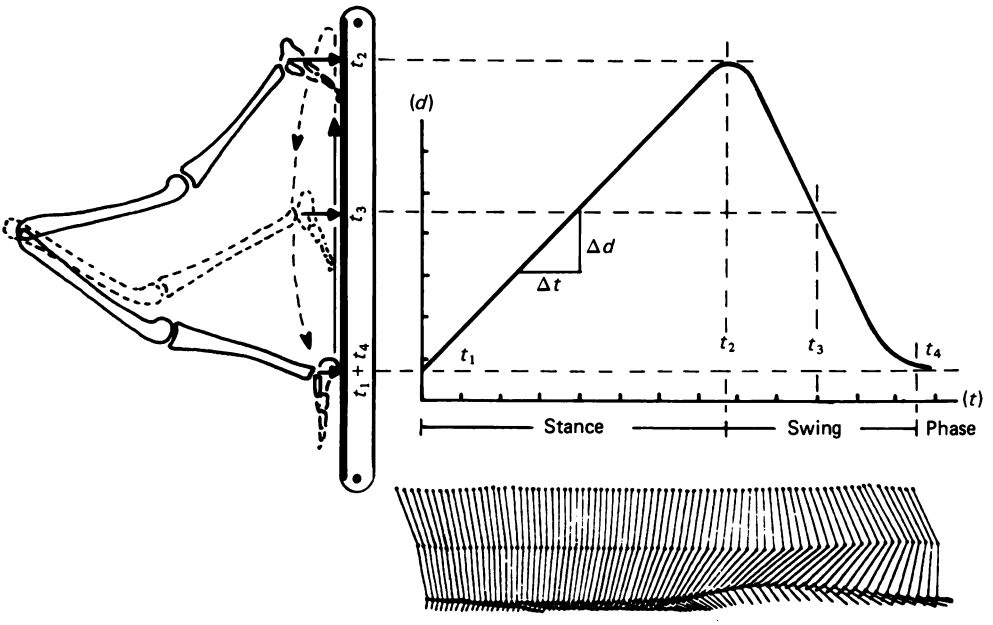

Fig. 1. Stick diagrams of leg movements during a step cycle reconstructed from filmed bony markers (below). A schematic illustration of the time course (t) of ankle joint movements in relation to a fixed point at the treadmill (d) during a normal step cycle is shown above.

Data analysis

The e.m.g. was recorded by a telemetric device (Schwarzer: Model 4024, FS; Munich, F.R.G.) and filtered (time constant 3 ms). Mechanical artifacts were shown to be minimal by passively shaking the legs and monitoring the e.m.g. on an ink recorder. The e.m.g. signals, together with signals from a goniometer and electrical switches were stored on a four-channel tape recorder (Tandberg) for off-line analysis. The e.m.g. was rectified and averaged (Nicolet model 1072) by a procedure described in previous papers (Dietz *et al.* 1979; Dietz, Quintern & Berger, 1981). The rectified e.m.g.s following ten unilateral displacements evoked by a stimulus at a defined phase of the step cycle were averaged, using the electrical switch in the shoes as the trigger which indicated the timing of the displacement. Goniometer signals were averaged in the same way. The resolution of the averaged histogram amounted to 256 points for each step cycle. All averaged recordings were transferred to an Apple micro-computer system and stored on disk for further processing and statistical analysis (e.g. mean values and standard deviations).

Later experiments (largely those involving treadmill acceleration) were analysed on-line and the recordings were averaged by a Sirius computer system (hence the different calibrations used in the Figures).

Three volunteers were filmed during perturbation of gait (Locam-camera: 220 frames/s). Stick diagrams and ankle angles were reconstructed and analysed from films which showed the positions of markers fixed over bony points.

RESULTS

Treadmill acceleration

Biomechanical events. The leg and body movements during gait were reconstructed from films of the bony markers. The schematic drawing in Fig. 1 represents the anterior and posterior movement of the ankle joint in relation to a fixed point during

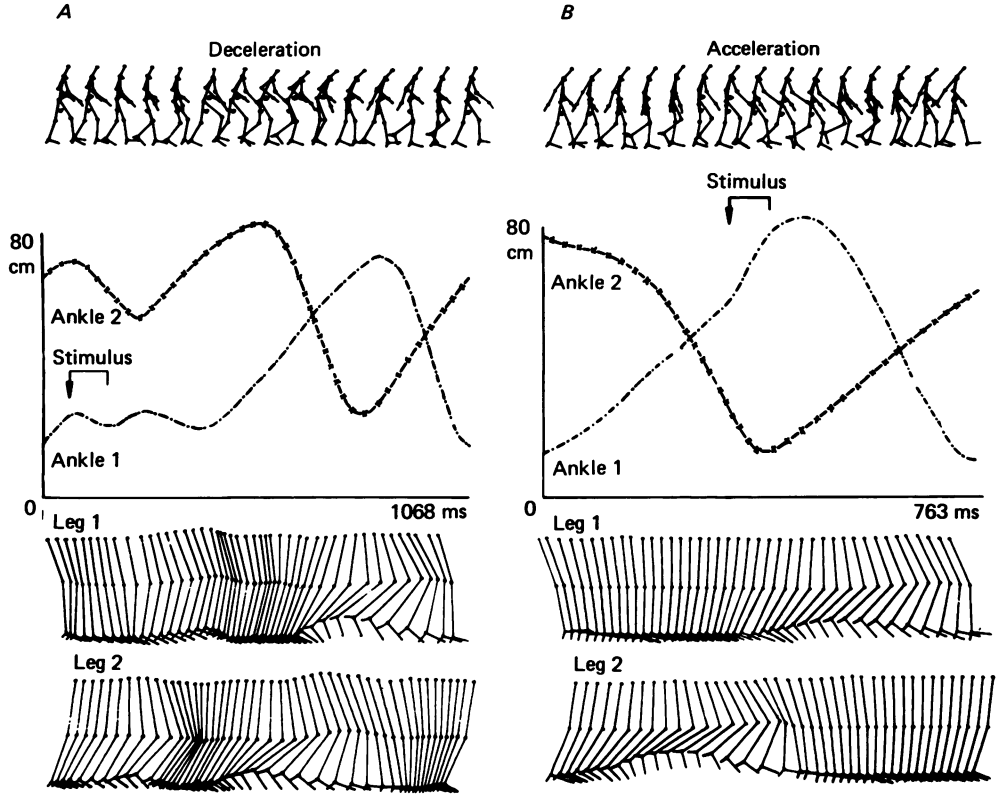

Fig. 2. Stick diagrams of leg movements (below) and the time course of ankle joint movements of both legs (above) during a step which was perturbed. *A*, deceleration of leg 1 in the middle of stance phase; *B*, acceleration of leg 1 at the beginning of stance phase. The arrow (↓⌐) indicates the onset and duration of the stimulus. The diagrams are normalized to one step cycle; compare with the undisturbed step cycle in Fig. 1.

walking on the treadmill (speed 3 km/h). Stick diagrams of the whole leg during the respective phases of the step cycle are shown below.

Fig. 2 shows the compensatory movements following a decelerating (*A*) and an accelerating (*B*) impulse to the treadmill. The trajectory of the ankle joint movements and the stick diagrams both demonstrate early bilateral reactions to the disturbance. Treadmill *deceleration* at the beginning of the stance phase prevented the usual backward movement of the supporting foot and forward tilt of the body. During this time the contralateral leg, which had started to swing in an anterior direction, swung backwards.

Treadmill *acceleration* resulted in accelerated backward movement of the weight-bearing leg. During this time, the forward swing of the contralateral leg was shortened. Deceleration thus displaced the supporting leg forward in relation to the centre of gravity, while acceleration displaced it backward (see stick diagrams). In both conditions the centre of gravity remained in the same relation to the treadmill. The step cycle was prolonged in the first condition while it was shortened in the second one.

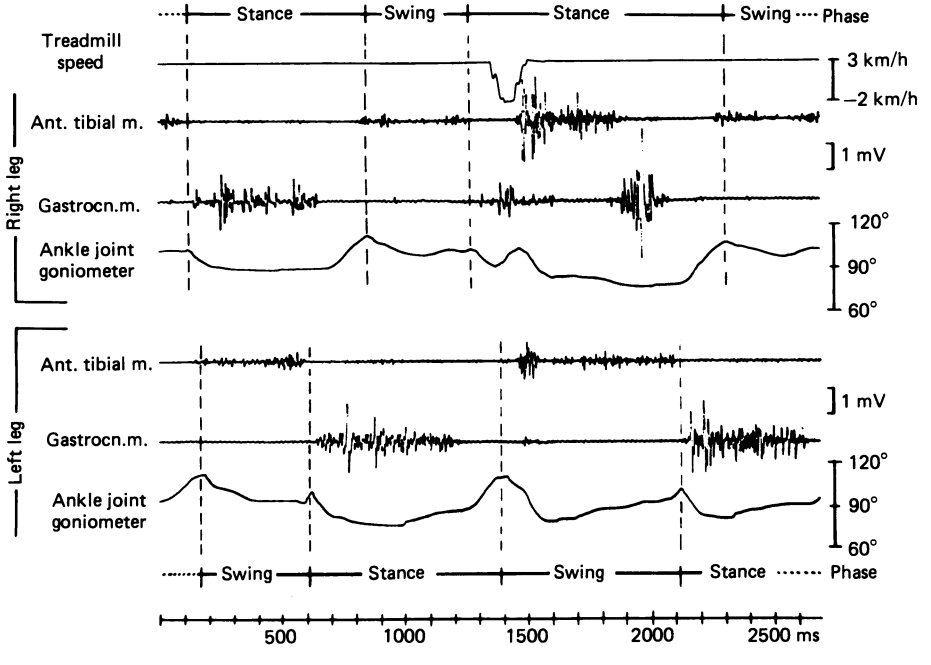

Fig. 3. Raw records of e.m.g. responses and ankle joint movements following deceleration of a treadmill moving at a speed of 3 km/h. Deceleration was unexpectedly imposed 80 ms after right heel contact. Vertical lines indicate touch-down and lift-up of the respective foot.

E.m.g. responses in the leg. Reproducible records of raw and averaged e.m.g.s were obtained from both legs after decelerating (Figs. 3 and 4) and accelerating (Figs. 5 and 6) impulses. Either direction of impulse was initiated at random on contact of the right heel. In Figs. 3 and 4 (treadmill deceleration) the ipsilateral tibialis anterior was activated at a latency of about 65 ms, and the burst of activity lasted 100 ms, while the gastrocnemius, which is normally active during stance (see Fig. 3, 300–400 ms, right leg), was silenced (see Fig. 4, 300–400 ms, right leg). Following the tibialis anterior activation, the angle of extension at the ankle was reduced beginning at time 300 ms, in Fig. 4, and returning to its normal trajectory (400–600 ms, right leg). At the end of stance phase, the exaggerated tibialis anterior activity was followed by gastrocnemius activity which was also greater than normal. The enhanced activity of tibialis anterior on the stance side was accompanied by a similar enhancement of activity in the swinging leg with the same latency of about 65 ms.

When the treadmill was accelerated (Figs. 5 and 6), the usual passive dorsiflexion

of the foot was faster (250–300 deg/s compared with 60–80 deg/s in undisturbed walking) and larger. This perturbation strongly activated the gastrocnemius (latency about 65 ms, duration 150 ms), its e.m.g. amplitude being about 8 times larger than normal for this phase of the gait cycle. Following this gastrocnemius activation the

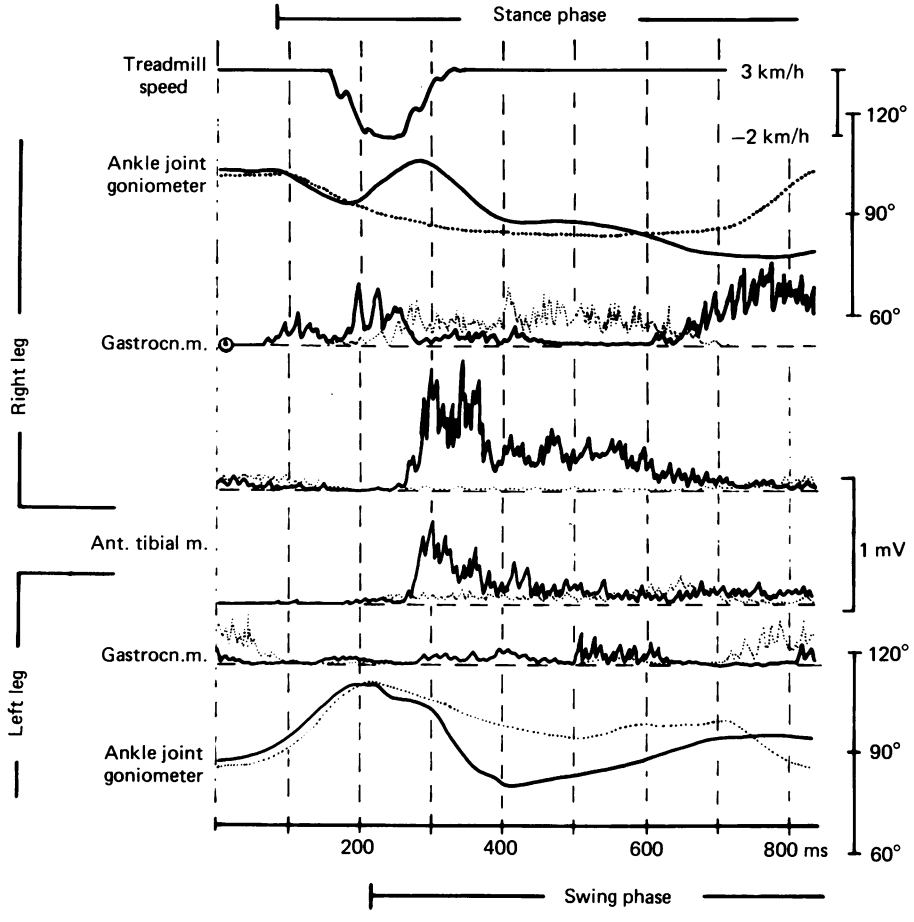

Fig. 4. Bilateral leg muscle e.m.g. responses following deceleration of the treadmill moving at a speed of 3 km/h. The e.m.g. was rectified and averaged ($n = 16$) together with the ankle joint movements. Dotted lines represent the normal walking pattern at 3 km/h. The impulse was released as in Fig. 3 and was used for triggering recording of the different parameters.

passive dorsiflexion of the ankle joint produced by the treadmill acceleration was halted and then reversed, so that the joint angle trajectory was returned to its normal position (see Fig. 6, 350–450 ms). This was then followed by another burst of gastrocnemius e.m.g. activity which prematurely ended the stance phase. This increase in gastrocnemius activity at about 65 ms was accompanied by activity in the contralateral tibialis anterior which was enhanced beyond its normal activity during the swing phase. In some of the records the gastrocnemius e.m.g. of the contralateral leg was also enhanced at the beginning of the next stance phase.

The ipsilateral gastrocnemius e.m.g. responses evoked by an accelerating impulse were tested under several conditions: (a) the impulses were introduced after 20 min of ischaemia induced by compression of the thigh; (b) the impulses were initiated by the subject himself; (c) the impulses were randomly introduced at the beginning and

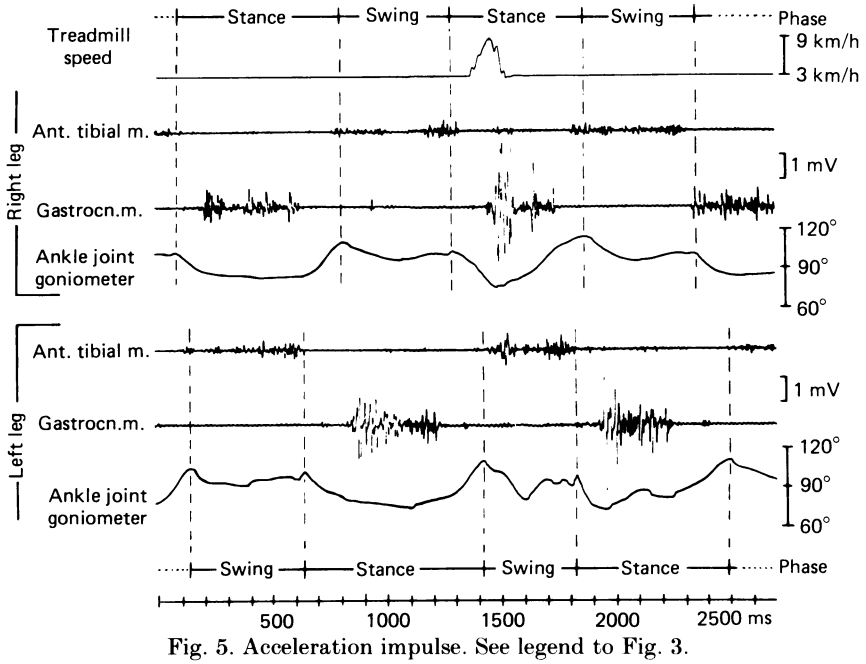

Fig. 5. Acceleration impulse. See legend to Fig. 3.

the end of a 2 h experiment. In the first condition the foot was numb, the Achilles tendon jerk was absent and vibration sense at the ankle joint (lateral malleolus) was much reduced. It would be expected that at this time a large proportion of the group I afferent fibres was blocked (Dietz, 1978; Dietz *et al.* 1979). The different e.m.g. responses can be seen in Fig. 7. Although the ankle joint movements induced by the perturbation were quite different, the latency and timing of the e.m.g. responses were similar in all conditions, and their amplitude changed by only a small amount, being only slightly diminished when acceleration was self-induced and during ischaemia. The e.m.g. responses in the control trials at the end of the experiment did not differ significantly from the responses obtained at the beginning.

In Fig. 7 the strength of gastrocnemius activation and the ankle joint movements during the stance phase of undisturbed gait can be compared with the changes of these parameters induced by the perturbation: although in (c) the treadmill acceleration caused only a moderate additional dorsiflexion of the foot, the gastrocnemius e.m.g. exceeded the normal e.m.g. activity by about 8 times.

The accelerating impulses used for the experiments of Fig. 7 were rather strong, but all subjects were able to compensate for them without support. When the perturbations were smaller (for example a change from 3 to 5 km/h), no distinct separate e.m.g. response could be detected above the basic activity, but as the amplitude of the perturbation was progressively increased, the additional e.m.g.

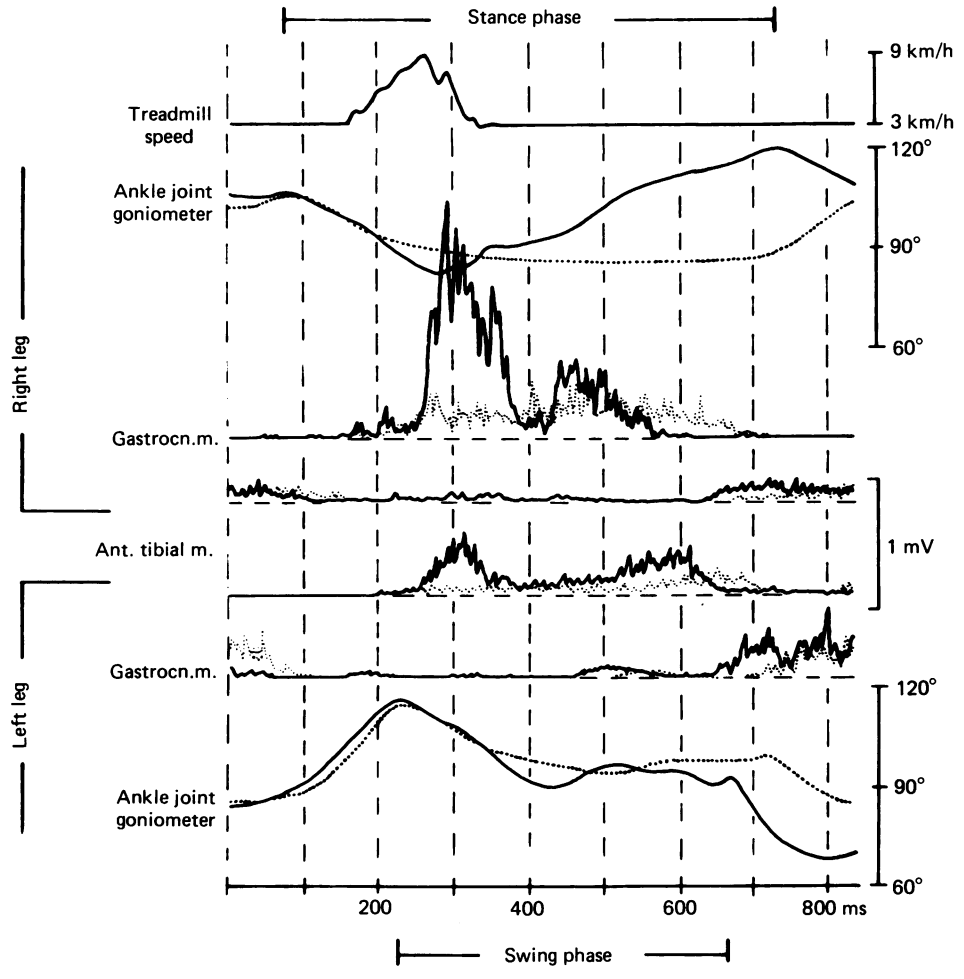

Fig. 6. Acceleration impulse. See legend to Fig. 4.

activity appeared rather abruptly, and this response did not increase continuously with increasing acceleration.

Unilateral tibial nerve stimulation

Transition from posture to gait. Whereas during normal stance or balancing there is co-activation of homologous leg muscles to maintain body equilibrium (Dietz & Berger, 1982), the same leg muscles are reciprocally activated during locomotion. The regulation of balance, however, remains an important requirement for locomotion. We therefore investigated those mechanisms which regulate posture during gait. Fig. 8 shows the e.m.g. responses following a unilateral displacement in three different conditions which represent the transitional stages from unstable posture, standing on two see-saws, to the initiation of gait. When the subject balanced in an upright posture on the two separate see-saws, one of which was placed one pace in front of

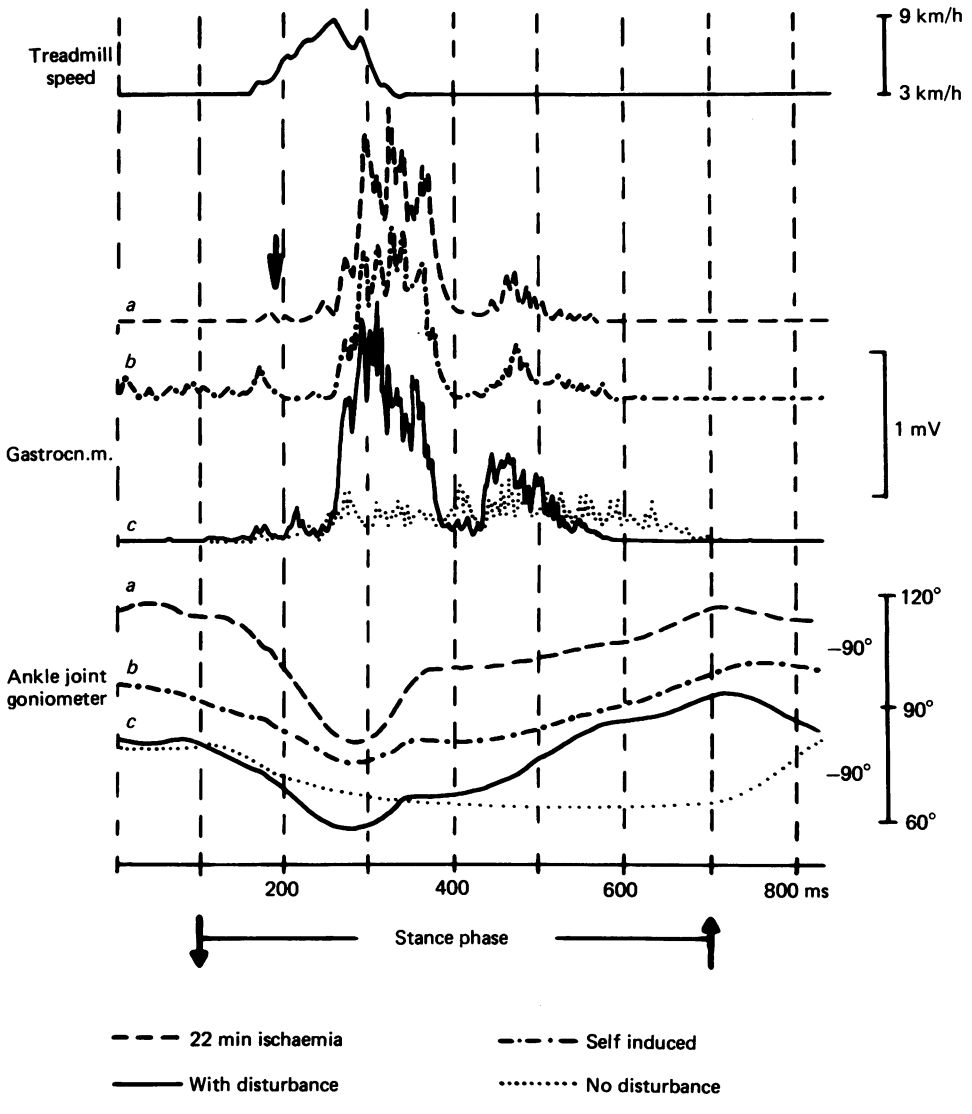

Fig. 7. Rectified and averaged (n = 16) gastrocnemius e.m.g. and ankle joint movements following acceleration of the treadmill 80 ms after heel contact in three different conditions: *a*, after 22 min of ischaemia; *b*, acceleration initiated by the volunteer; *c*, acceleration unexpectedly induced. The dotted line represents the undisturbed walking pattern. The arrow above indicates the time of onset of effective change in belt speed. The arrows below, indicate touch-down (↓) and lift-up (↑) of the respective foot.

the other (Fig. 8*B*), and the front leg was displaced by a single stimulus to the tibial nerve, then, after a delay of about 70 ms, both tibialis anterior muscles were activated simultaneously, followed by gastrocnemius activation. This pattern was slightly different from the condition with parallel see-saws (*A*); the delays were longer, and the tibialis anterior e.m.g. was less vigorous. When the subject mimicked the initiation of a step by tilting the body forwards and slightly plantarflexing the foot

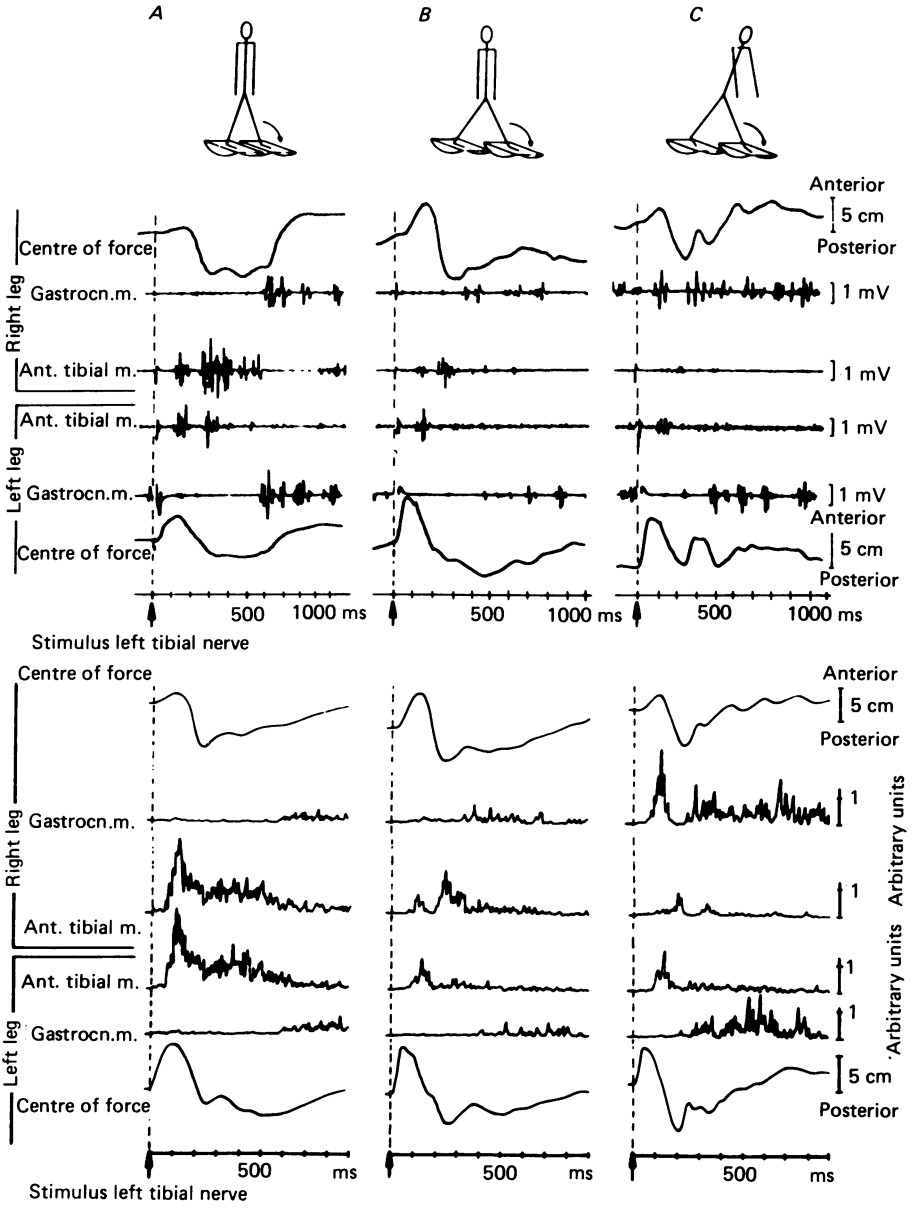

Fig. 8. Bilateral tibialis anterior and gastrocnemius e.m.g. responses following stimulation of the left tibial nerve in different balancing conditions representing the transition from stance to gait. *A*, normal balancing task; *B*, a distance of one pace between the two see-saws; *C*, initiation of a step. Raw e.m.g. (above) and averaged ($n = 10$) (below). The arrow indicates the moment of tibial nerve stimulation.

of the rear (right) leg (*C*), the response to a similar left tibial nerve stimulus changed; there was then activation of the left tibialis anterior muscle at about 60 ms after the displacement but with silence of the contralateral tibialis anterior. The e.m.g. response to the disturbance then resembled the one seen during walking, with

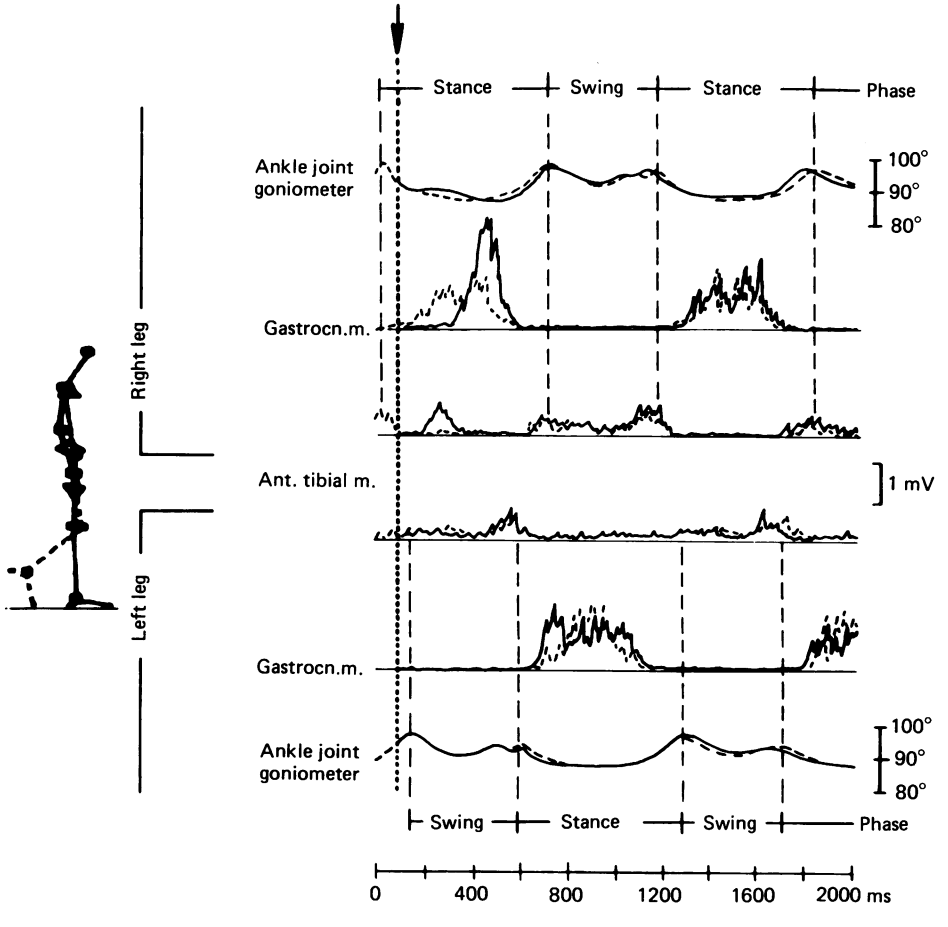

Fig. 9. Rectified and averaged ($n = 10$) tibialis anterior and gastrocnemius e.m.g. responses with records of ankle angle following stimulation of right tibial nerve when both heel and ball of foot were in contact (speed 4 km/h). For comparison, the normal walking pattern is drawn as a dashed line (– – –). ↓, indicates time of stimulation. The diagram on the left was drawn from filmed coordinates of the position of bony markers. The dashed line indicates the unstimulated leg. For vertical lines see Fig. 3.

powerful gastrocnemius activation of the rear-most leg, and tibialis anterior activation of the leading leg. This pattern was obtained regardless of whether the displacement was induced by an electrical stimulation of tibial nerve or by an anterior tilt of a see-saw: each of these stimuli caused a sudden plantarflexion of the foot.

Displacement during gait. During walking, the bilateral e.m.g. responses observed

after a displacement were much more complex than during standing, and depended on the phase of the step cycle in which the stimulus was delivered.

The averaged recordings for one subject displaced at the beginning of the stance and the swing phase are shown in Figs. 9 and 10. For more ready comparison, the

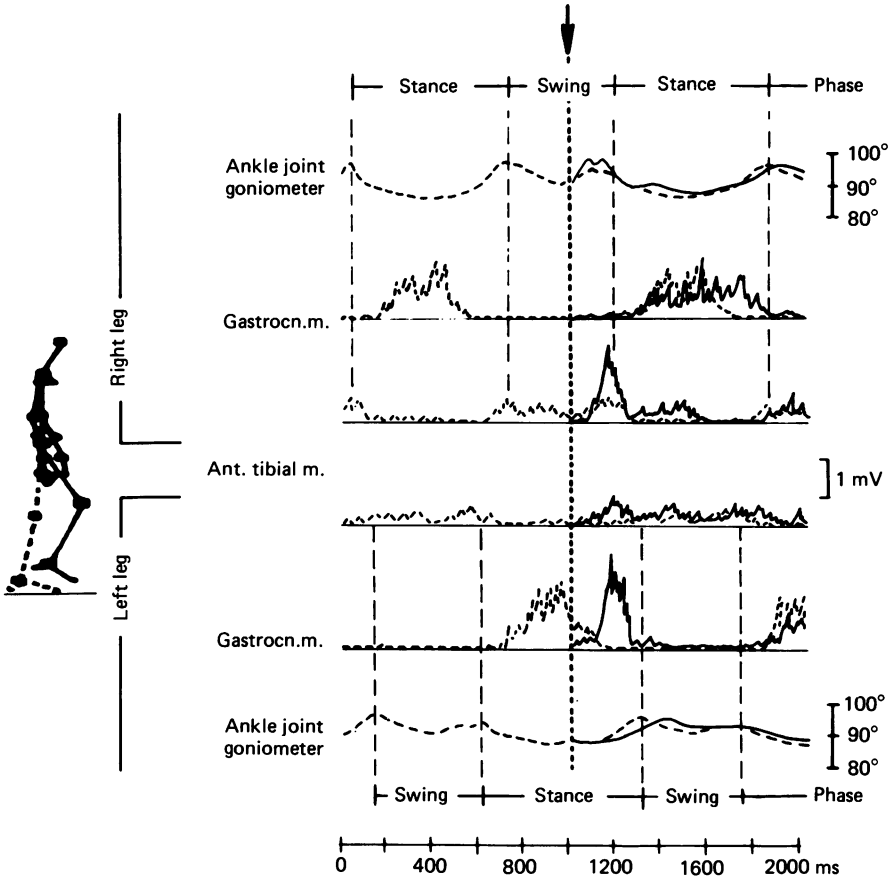

Fig. 10. Stimulation of right tibial nerve during contact of the contralateral foot. See legend to Fig. 9.

averaged recordings during undisturbed walking at the same speed have been added to each trace. As can be seen from the goniometer traces of the right ankle joint, the electrical stimulus applied to the tibial nerve caused a plantarflexion of the right foot. This deviation was rapidly followed by activity in the ipsilateral tibialis anterior, which was significantly stronger than the normal tibialis anterior e.m.g. at a similar phase of the undisturbed gait cycle.

The strength of this ipsilateral tibialis anterior response was strongly dependent on the phase of step cycle in which the right foot was displaced. The strongest responses were observed when the plantarflexion induced by the stimulus was inappropriate for the movement occurring in this phase of undisturbed gait (the

beginning of the stance and swing phase). As seen in the goniometer recordings, a rapid correction of the ipsilateral foot position was achieved by the tibialis anterior activation. In contrast, the weakest responses were obtained when the foot was displaced during the normal plantarflexion of the foot at the end of stance phase.

When the stimulus was delivered at the beginning of stance phase (Fig. 9), the usual gastrocnemius activation was inhibited during the tibialis anterior response. After this response, the gastrocnemius activation was more or less enhanced and ended later in the stance phase than normal. The goniometer traces show that in this condition the right foot was plantarflexed more rapidly at the end of stance but despite this the whole step cycle was prolonged. These late changes of e.m.g. activity on either side were confined to the one step cycle. The next step cycle did not show any significant alterations in timing or strength of e.m.g. activity, irrespective of whether the stimulus was delivered at the beginning or the end of the step cycle. In addition, the speed of locomotion did not influence this finding. Both early and late responses were present at locomotion speeds of 2 and 4 km/h, but the amplitudes of the responses and the background activity were both larger at higher locomotory rates.

When the stimulus was delivered in the middle of swing phase, i.e. stance phase of the contralateral leg (Fig. 10), the ipsilateral tibialis anterior and the contralateral gastrocnemius were activated. The latter appeared with the same latency of 90 ms. In this condition, the stance phase of the contralateral leg was prolonged (about 100 ms, see goniometer trace).

In order to exclude other indirect effects of the electrical stimulation on the generation of e.m.g. responses, the dorsum of the right foot was stimulated supramaximally, while on other occasions the tibial nerve was stimulated submaximally at a strength that caused no visible muscle contraction. There were no visible e.m.g. responses to these electrical stimuli, and there were no obvious changes in the gait pattern. It may therefore be supposed that it was the triceps surae contraction and the subsequent mechanical effects of foot plantarflexion which provided the stimulus for the bilateral leg muscle e.m.g. responses.

DISCUSSION

Perturbations of gait evoked adequate and useful compensatory responses in both legs, their form being dependent on the type and direction of the perturbation, as well as on the phase of the step cycle in which they were induced. As shown by the film analysis the centre of body gravity remained unaffected by the perturbations. To hold this factor constant may therefore be the 'variable' controlled by the compensatory responses with the purpose being to prevent falling.

Four questions arise from these investigations: (1) how far are the different modes of perturbation reliable in regard to the e.m.g. responses analysed in this study? (2) by which pathway are the e.m.g. responses mediated? (3) in which structure and in what way are they generated, and (4), what is their relation to corresponding animal experiments?

1. Significance of e.m.g. reactions evoked by different stimuli

Although the nature of the two stimuli used to induce perturbations of gait were different, their effects were similar in so far as the e.m.g. responses evoked by treadmill acceleration or tibial nerve stimulation were specific and purposeful.

While treadmill acceleration mimics a natural, mechanical mode of perturbation, the assumption that supramaximal tibial nerve stimulation is likewise 'natural' seems to be questionable because of the different orthodromic and antidromic effects which could influence the response. However, there are some observations which support the view that the e.m.g. responses following a tibial nerve stimulation are due to its mechanical effect, i.e. the foot plantarflexion: (i) The bilateral tibialis anterior activation following a unilateral displacement during balancing was the same whether the foot plantarflexion was induced by an anterior tilt of a see-saw or by tibial nerve stimulation (Dietz & Berger, 1982); (ii) The e.m.g. responses did not appear when a noxious electrical stimulus was applied to the dorsum of the foot or when the tibial nerve was submaximally activated at a level not producing a visible mechanical effect.

Unilateral tibial nerve stimulation during stance and gait respectively produced different types of e.m.g. responses: while during stance, the bilateral tibialis anterior activity was sufficient to counteract a unilateral perturbation; during gait more differentiated reactions were necessary, dependent on the phase of step cycle.

The appearance of different responses during stance and gait is not in agreement with previous observations of e.m.g. adjustments elicited by imposed platform movements in standing (Nashner, 1977; Nashner, Woollacott & Terma, 1979) and in walking subjects (Nashner, 1980), which showed a similar organization of responses in both conditions. This discrepancy may be related to the different modes of perturbation used. In these earlier studies the platform displacements were smaller (5 deg compared with 10–15 deg) and slower (40 deg/s compared with 200–250 deg/s).

The reactions following perturbations induced by changes in treadmill speed consisted of an ipsilateral leg muscle contraction which opposed changes of the angle between foot and leg. An enforced foot dorsiflexion was also evoked in the contralateral swinging leg. In combination with the stick diagrams, this contralateral response can be interpreted as being a quick preparation for touch down of this foot behind (deceleration) or in front of the body (acceleration), to support the body and to compensate for imbalance.

2. Reflex pathway

The fact that the e.m.g. responses to the perturbations arise bilaterally with a latency of 65–75 ms (following treadmill impulses), which is about 25 ms longer than the H-reflex latency, makes it rather unlikely that they represent monosynaptic stretch reflexes. (The longer latencies measured after tibial nerve stimulation can be explained by the longer mechanical delay needed to produce the displacement (Berger & Dietz, 1983).) Although the triceps surae was stretched with a velocity of 250–300 deg/s by treadmill acceleration, no e.m.g. responses appeared within the range of monosynaptic latencies. According to the results of those experiments in which muscle stretch was applied by a torque motor (Gottlieb & Agarwal, 1979), such

early responses should be expected. This discrepancy supports the notion of a suppression of the monosynaptic stretch reflexes during gait (Morin, Katz, Mazieres & Pierrot-Deseilligny, 1982; Dietz, Quintern & Berger, 1984).

Another observation, that of the preservation of these e.m.g. responses in the ischaemic walking condition, indicates that group I afferents from leg and foot muscles are not likely to play a dominant role in mediating these responses. As has been previously shown (Dietz, 1978; Dietz *et al.* 1979), at this stage of ischaemia a large percentage of group I afferents could be expected to be blocked.

The latency of 65–75 ms seems, however, to be too short for a 'long-loop' pathway. This at once becomes evident when these latencies are compared with those observed in muscles with a shorter reflex pathway. The so-called 'M 2'-response following stretch of the hand extensor muscles, which was suggested to be mediated by a transcortical reflex pathway, appears only with a latency of about 60 ms (Lee & Tatton, 1978). It is therefore suggested that at least the early part of the stumbling e.m.g. responses in the leg muscles is mediated mainly by group II and III afferent fibres in a spinal pathway. Alternatively, the responses may remain after ischaemia because a sufficient afferent input is generated from the thigh muscles.

3. Generation of e.m.g. responses

The above-mentioned factors suggest that the e.m.g. responses are not simply mediated along classical monosynaptic stretch reflex pathways, but that they represent complex reactions generated at a spinal level. They are specific for a given mode of perturbation and are not much affected by additional external factors (such as ischaemic nerve blocking, learning effects during repeated trials or self-induction of the perturbations). The appearance of the e.m.g. responses was not much modified by a further increase of the stimulus. This supports the idea that a complex reaction becomes released or triggered at a distinct threshold.

The e.m.g. responses generated at a spinal level are dependent on a supraspinal control. This becomes obvious when the reactions to perturbations are studied in patients with spastic paresis due to a supraspinal lesion of the motor system (Berger, Horstmann & Dietz, 1984; Dietz & Berger, 1984). While the monosynaptic stretch reflexes are exaggerated in these patients, the polysynaptic spinal reactions to perturbations are reduced or absent. These observations are in accordance with results of animal experiments demonstrating that spinal neuronal circuits are controlled by supraspinal centres (Baldissera, Hultborn & Illert, 1981, p. 512 *et seq.*) and that a variety of spinal polysynaptic reflex actions are depressed in the decerebrate preparation (Baldissera *et al.* p. 564 *et seq.*).

4. Animal models

In cats, tactile, electrocutaneous and direct nerve stimuli during different phases of the step cycle have been applied to the paw, to study the interaction of peripheral inputs with the spinal stepping generators (Forssberg, Grillner & Rossignol, 1975; Duysens & Pearson, 1976; Rossignol, Julien & Gauthier, 1981). The reactions seen in spinalized or in intact cats (Forssberg, 1979) are phase dependent, but they are quite stereotyped; there is an increase in flexion if the stimulus is applied during the swing phase of the step cycle, but the extensor thrust is enhanced when the same

stimulus is imposed during the stance phase. In cats, as well as in man, unpredictable perturbations are rapidly compensated for, the responses being adapted to the underlying locomotor activity. In the cat the responses are simpler and more stereotyped than in man, a difference in behaviour that may reflect the simpler problems of maintaining a balanced equilibrium in quadrupedal rather than bipedal gait.

We thank Professor Dr J. Stephens for critical review and correction of the last version of the manuscript and Mr S. Fellows for correcting the English text. Technical assistance was provided by Mrs U. Römmelt and Mr G. Brenneisen.

The work was supported by the Deutsche Forschungsgemeinschaft (SFB 70 – Hirnforschung und Sinnesphysiologie).

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