Short report

Spastic paresis: impaired spinal reflexes and intact motor programs

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SUMMARY Leg muscle EMG responses evoked by short treadmill acceleration impulses applied during stance were analysed in patients with spastic hemiparesis. The compensatory reactions on the unaffected side consisted of a diphasic pattern of leg muscle activation. The first response could best be described as a polysynaptic spinal stretch reflex response. This response was absent on the spastic side, except for its later, declining component. This remainder of the first response and the following activation of the antagonistic muscle was identical on both the unaffected and the spastic side. This part of the pattern is assumed to be centrally programmed (at the spinal level) and triggered by the termination of the acceleration impulse.

In patients with spastic paresis, preserved central programming explains their ability to execute stepping movements, while the impaired function of spinal reflexes explains their difficulty in modulating their steps and in quickly adapting them to the actual ground conditions.

Patients with spastic paresis have difficulty in modulating their steps and in performing quick movements. In healthy subjects such a compensation for irregularities of the ground during stance and gait was shown to be mainly achieved by the activation of polysynaptic spinal reflexes which are part of a centrally programmed pattern.¹ Earlier studies have shown a defective function of the spinal reflex system in patients with spastic paresis during perturbations of gait² and balancing³; the inhibition of monosynaptic stretch reflexes seen in healthy subjects was lacking in the patients and the function of the essential polysynaptic reflexes was impaired.

The aim of this study was to evaluate the extent to which reflexes and the programmed pattern are impaired in spastic paresis and how far the two mechanisms can be separated. For this purpose forwards

and backwards directed perturbations with different treadmill accelerations were applied during stance. By this approach the tibialis anterior or the triceps surae muscles were stretched and compensatory reactions of foot flexor and extensor muscles could be studied. Patients with spastic hemiparesis were examined in order to use the unaffected leg as the control for the spastic one.

Patients and methods

During stance perturbations in 11 patients with spastic hemiparesis EMG activity was recorded, using surface electrodes, from the medial gastrocnemius and tibialis anterior muscles on both sides. The patients (aged 16.5 to 74.4, mean 52.1, SD 17.8 years) had a hemispheric lesion of vascular (8), inflammatory (1), neoplastic (1) or perinatal (1) origin. The study was carried out 1 month to 16 years (mean: 6 years, 8 months) after onset of symptoms. All patients had unilateral pyramidal signs with exaggerated tendon reflexes and extensor plantar responses. No very slightly affected or severely disabled patients were included. All patients were able to walk without support. Experiments were performed with the permission of the local ethical committee and with the informed consent of the subjects.

The ankle joint angles were monitored by goniometers fixed at the lateral aspect of the foot and leg. Care was taken to adjust the ankle joints of both legs to the same angle (90°). The knee joint movements were restricted by a bilateral splint which was adapted for the normal stance position of the respective subject, in order to prevent knee flexion as a

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Received 6 July 1987 and in revised form 11 November 1987.
Accepted 17 November 1987

part of the compensatory reaction. The general technique for recording and analysing the EMG responses of leg muscle have been fully described in earlier papers.^{1,4}

Forwards and backwards directed acceleration impulses were induced during standing on a treadmill (Woodway: MTR 250-40). Ramp impulses with four different accelerations were used, for which the following displacements were achieved: (a) 5.5 ms/s²: 4 cm; (b) 11.1 ms/s²: 7 cm; (c) 16.6 ms/s²: 10.5 cm; (d) 22 ms/s²: 15 cm. Impulse duration was held constant at 90 ms. All eight (forwards or backwards directed) impulses were released in a random order. The onset of the impulse delivered by an impulse generator was used as a trigger signal for averaging the biomechanical and EMG responses.

The EMG of the leg muscles, the joint movements and the treadmill velocity were monitored on an ink recorder (Siemens, Type EM 34) and on the computer screen and, after rectification of the EMG, were transferred on-line and sampled (rate 500 Hz) by a computer system (Sirius). The parameters were averaged ($n = 10$) over a period of 200 ms before and 600 ms after the impulse. Further processing and statistical analysis of the data were carried out by the computer system.

Fig 1 (a) Mean values of the rectified and averaged ($n = 10$) leg muscle EMG responses together with the ankle joint movements (starting with an angle of about 90° on both sides). Following backwards directed acceleration impulses during stance. Unaffected (a) and spastic (b) leg of 11 patients with spastic hemiparesis. a-d: different accelerations (see Methods). Impulse duration was constant (90 ms). Arrows indicate onset of displacement.

Results

Figure 1 shows the pattern of leg muscle activation induced by the different backwards directed accelerations (mean values of the averaged EMG responses of all 11 patients). The background EMG activity was the same on both legs from -10 to +35 ms. The EMG responses in both legs started with a latency of about 35 ms (for impulses c and d). On the unaffected side, the EMG responses increased continuously over 90 ms (corresponding to the duration of the displacement). The steepness of the increase of activity depended on the displacement velocity. On the spastic side the EMG amplitude remained constant at a low level over 80 ms (the difference in EMG activity between the two sides was significant ($p < 0.001$) for all impulses). At a latency of 115 ms (35 ms after termination of the backwards displacement) a steep increase in gastrocnemius activity occurred on the spastic side (b-d). This latter part of the gastrocnemius response coincided in amplitude and timing with the decreasing part of the gastrocnemius response on the unaffected side (starting with the peak (c and d)). Therefore, the responses on both legs ended with the same latency (about 175 ms). The gastrocnemius responses were followed by a tibialis anterior activation (b-d), the strength of which depended on the displacement velocity. This tibialis anterior activation did not differ in timing (100-220 ms, b-d) or amplitude on the spastic and unaffected side.

Fig 2 Same parameters as in fig 1 for forwards directed accelerations.

The displacement velocities were slower on the spastic side for the two higher accelerations despite the same or lower level of basic activity from 0 to 80 ms (unaffected leg mean velocity: 86°/s (c) and 109°/s (d) (initial velocity for d 145°/s); spastic leg: mean velocity 70°/s (c) and 82°/s (d) (initial velocity for d: 114°/s)). These differences cannot, however, explain the difference in the EMG responses.

Figure 2 shows the pattern of leg muscle activation induced by the different rates of *forwards* directed accelerations. On the unaffected side the tibialis anterior responses started with a latency of 60 ms. Once again, the steepness of the increase in activity depended on the displacement velocity. Qualitatively, the differences between the two sides were similar to those seen in the gastrocnemius responses in fig 1: the first part of the tibialis anterior responses was reduced on the spastic side. However, this latter reduction was less pronounced and the late part of the tibialis anterior response did not coincide to the same extent as the gastrocnemius responses did on the spastic and unaffected sides (the difference in EMG activity was significant only for c and d at the 5% level). The tibialis anterior responses ended on both sides at the same time (about 200 ms following displacement onset). They were followed by a gastrocnemius activation which again was of similar timing and amplitude on both sides. The strength of this activity depended on the displacement velocity, as did the tibialis activity in fig 1.

The displacement velocities at the ankle joint for forwards displacements were higher than those seen during backwards displacements and they did not differ between unaffected and spastic leg (unaffected leg, mean velocity: 110°/s (c) and 145°/s (d); spastic leg: 114°/s (c) and 159°/s (d)).

Discussion

The observations made in this study indicate that the compensatory reactions to stance perturbations consist of two parts: a functionally directed early EMG response, mediated by stretch reflexes which is *impaired* in spastic paresis, followed by a triggered component of complex form which is *unchanged* in the patients.

The generation of the gastrocnemius response following backwards directed gait perturbations was shown to be under continuous control by muscle proprioceptive information and can be best described in terms of a stretch reflex response.¹ In healthy subjects only a small early part of this response (35 to about 65 ms) is due to a monosynaptic stretch reflex activity mediated by group I afferents and the main part can be assumed to be mediated by muscle proprioceptive input from group II afferents on a polysynaptic spinal

level.⁴ The latter reflex system is needed to adapt the activity of leg extensor muscles to the actual ground conditions. This separation between these two reflex systems is hardly possible on the basis of the EMG traces shown here (mean values of averaged responses), but can clearly be seen in individual EMG responses.^{4,5} In addition, higher acceleration rates as used earlier,² could elicit more regularly monosynaptic reflex potentials.

The functionally essential polysynaptic reflex system is obviously defective in spastic paresis. As a result, no velocity sensitive response was seen on the spastic side. A similar reduction of the polysynaptic reflex responses, connected with exaggerated monosynaptic reflexes, was seen in small children at an early stage in the development of gait⁶ or perturbations of stance and gait.⁷ One may therefore conclude that the function of the polysynaptic spinal reflexes is dependent on intact supraspinal control: when this control is either immature (small children) or impaired (spastic paresis), inhibition of monosynaptic stretch reflexes is absent, associated with a reduced facilitation of polysynaptic spinal reflexes.

Despite the reduced activity in the gastrocnemius, higher backwards directed accelerations induced slower displacements on the spastic side. This was not the case for the forwards directed accelerations, stretching the tibialis anterior muscles. This observation could best be explained by changes of intrinsic muscle stiffness of the gastrocnemius muscle in spasticity, a finding described already elsewhere.^{2,8,9}

The later part of the compensatory reaction (the declining part of the gastrocnemius response and the late tibialis anterior activation for backwards directed perturbations) was, in contrast to the first part, identical in timing and amplitude on the unaffected and the spastic sides. This suggests that another mechanism is responsible for the generation of this part of the compensatory reaction. Although the EMG amplitude of this later part of the pattern did correlate with displacement velocity, its behaviour is different from a stretch reflex response. The observation that this part of the pattern is reduced or absent when the increasing and decreasing phases of the treadmill acceleration were similar (cf ref 2) indicates that the abrupt termination of the ramp impulses represents the trigger signal to induce this part of the response. The diphasic form of this pattern is reminiscent of the diphasic pattern associated with the voluntary ballistic arm and finger movements, which is supposed, to a large extent, to be centrally programmed (for review see refs 10, 11).

The short latency between the end of displacement and appearance of the gastrocnemius response in the spastic leg (about 35 ms) indicates firstly, that signals from fast conducting group I fibres trigger this

response and, secondly, that this response is generated by spinal interneuronal circuits. Such interneuronal circuits are known from cat experiments and are closely connected to the stepping generators.¹²⁻¹⁴ As discussed earlier⁴ the vestibular system is suggested to play no significant role in the generation of these responses.

This programmed pattern is obviously still functioning after a supraspinal motor lesion leading to a hemiparesis. This result is in accordance with the observation that the reciprocal activity pattern of the leg muscles during undisturbed gait of patients with severe spastic paraparesis is qualitatively preserved,⁸ as well as with experiments on spinalised cats which are able to perform stepping movements (for review see ref 15).

We thank Dr S Fellows for correcting the English text. This work was supported by the Deutsche Forschungsgemeinschaft (SFB 325).

We thank Dr D Kernell for helpful discussions on this topic.

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