Stepping before standing: hip muscle function in stepping and standing balance after stroke

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Abstract

Objective—To compare the pattern of pelvic girdle muscle activation in normal subjects and hemiparetic patients while stepping and maintaining standing balance.

Design—Group comparison.

Method—Seventeen patients who had regained the ability to walk after a single hemiparetic stroke were studied together with 16 normal controls. Median interval between stroke and testing was 17 months. Amplitude and onset latency of surface EMG activity in hip abductors and adductors were recorded in response to sideways pushes in either direction while standing. Similar recordings were made in the same subjects during gait initiation and a single stride.

Results—In the standing balance task, normal subjects resisted a sideways push to the left with the left gluteus medius (74 ms) and with the right adductor (111 ms), and vice versa. In hemiparetic patients, the amplitude of activity was reduced in the hemiparetic muscles, the onset latencies of which were delayed (gluteus medius 96 ms, adductor 144 ms). Contralateral, non-paretic, adductor activity was increased after a push towards the hemiparetic side of patients with stroke and the latency was normal (110 ms). During self initiated sideways weight shifts at gait initiation, hemiplegic muscle activation was impaired. By contrast, the pattern and peak amplitude of hip muscle activation in stepping was normal in both hemiparetic and non-hemiparetic muscles of the subjects with stroke.

Conclusions—In ambulant patients with stroke, a normal pattern of activation of hemiparetic muscles is seen in stepping whereas the response of these muscles to a perturbation while standing remains grossly impaired and is compensated by increased activity of the contralateral muscles. This suggests that hemiparetic patients should be able to step before regaining standing balance.

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Stroke commonly disrupts the automatic postural responses which contribute to standing balance. This can lead to difficulty walking and increased risk of falling. Induced forward sway in normal subjects elicits a pattern of distal to proximal muscle activation of the

gastrocnemius muscle, hamstrings, and paraspinal muscles with latencies ranging from 80-120 ms.¹ Patients with hemiparetic stroke show either a delay in onset of the normal ordered pattern of distal to proximal activation of agonist muscles, or the sequence of activation is replaced by cocontraction of agonist and antagonists.^{2,3}

Sideways sway has been little studied.^{4,5} By applying identical sideways forces in each direction, the responses of the hemiparetic and unaffected side of the body may be compared. We have used this technique to compare the postural responses of hemiparetic muscles and compensatory activity in contralateral muscles.

Absolute amplitude of EMG activity in different normal muscles may vary widely, making direct comparison of results from one subject to another less reliable. In normal subjects, EMG activity in the task of interest may be expressed as a percentage of activity in a maximum voluntary contraction, but this is impractical in hemiparetic patients. We therefore used gait initiation as our standard task. This involves a self initiated lateral weight shift followed by a forward step. Both are very consistent movements⁶ which required little explanation to patients and allowed us to compare activity in the same muscles in postural responses, self initiated movements, and stepping.

Subjects and methods

METHODS

Surface EMGs of bilateral hip abductors (gluteus medius) and adductors (adductor longus), lateral torque component of the ground reaction forces, and pelvic displacement were recorded when balance was disturbed by a sideways push and when taking a step forwards. The EMG amplifiers were constructed by John Barton, 27 Izaak Walton Way, Cambridge CB4 1TY, from the design of Johnson *et al.*⁷ Electrodes were placed 5 and 8 cm below the iliac crest, directly above the greater trochanter, and 7 and 10 cm below the pubic tubercle.^{8,9} There is no EMG interference from the adjacent tensor fascia lata or quadriceps at these sites.¹⁰ The perturbing forces were generated, and pelvic movement measured, by computer controlled linear motors (Linear Technology Ltd, Rayleigh, Essex SS6 7XF, UK) attached to a semirigid belt around the subject's pelvis. Subjects stood on a six axis force plate (type 4060H, Bertec Corporation, 819 Loch Lomond Lane, Worthington, Ohio 43085, USA): the lateral torque component of the ground reaction force data was used to align the gait initiation trials before averaging.

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Table 1 Subjects' age and functional scores

	Age (mean (y))	10 m walk time (median (s))	Functional reach (median (cm))	Rivermead mobility index (median /15)	Leg power motricity (median /100)
Controls	46	7	36	15	100
Strokes	54	13	27	13	70
p (t test or Mann-Whitney)	NS	<0.0001	<0.05	<0.001	<0.0001

Sideways push trials were aligned using the applied force data.

Individual trials were filtered, rectified, and averaged before analysis using LabView (National Instruments, 6504 Bridge Point Parkway, Austin, Texas 78730–5039, USA) software. Electromyographic data was band pass filtered between 25 Hz and 250 Hz (gait) or 500 Hz (sideways push); 50 Hz interference was removed by a 48–52 Hz Butterworth

fourth order notch filter before rectification. Applied force and ground reaction force data were low pass filtered at 30 Hz and pelvic displacement at 12 Hz. To calculate peak muscle activity in stepping, averaged EMG data was low pass filtered to remove single outlying points before each muscle's highest value was identified: a 175 ms window for integration was then centred at this point. Increase in EMG amplitude during the initial sideways weight

Figure 1 Averaged EMG of (A) a control subject and (B) right hemiparetic patient after a sideways push, indicated by the vertical line. After a push to the right, the control subject shows early suppression of left gluteus medius, then activation of right gluteus medius followed by left adductor. The same pattern is seen in contralateral muscles after a push to the left. The right hemiparetic patient shows loss of the normal response in the right gluteus medius after a push to the right, and in the right adductor after a push to the left. * Loss of normal response in hemiparetic muscles.

shift could not be measured reliably due to recording insufficient stable baseline activity in the earlier control and stroke subjects.

In the sideways push task, amplitude of muscle activity (area under the curve) was measured by integrating the EMG data between 60 and 235 ms after force onset (IEMG). Baseline activity was estimated in the same interval before the perturbation. To partially normalise the range of EMG data across subjects, the response to the perturbation was expressed as (IEMG after perturbation – IEMG before perturbation) / IEMG before perturbation. Onset of applied force and muscle activity was identified by visual examination of the averaged data.

In the standing balance task, subjects were told to maintain their starting posture. Pushes were applied in blocks of five to the left and five to the right, up to a total of 20 pushes in each direction. Stance width was standardised at 27–30 cm. In the gait initiation task, subjects stood at one end of the force plate and took one step forward to finish with feet side by side. Forty trials were recorded, in alternate blocks of five trials leading with the left foot and five trials leading with the right foot.

SUBJECTS

Patients with stroke had had a single hemiparetic stroke, which initially prevented them walking, but they had all recovered the ability to walk short distances. Patients with gross language, or visuospatial, or visual field defects were excluded. There were 12 male and five female patients with stroke, with 13 right and four left hemiplegias. Seven patients also had partial hemianaesthesia and one had sensory inattention. The age and sex distributions of the 16 controls and 17 patients with stroke did not differ significantly. The median interval since their stroke was 74 weeks. Patients with stroke walked more slowly (median 13 s *v* 7 s, $p < 0.0001$) and had lower Rivermead mobility index scores than control subjects (table 1).

All controls and 16 patients with stroke resisted sideways forces of 3% body weight and one patient resisted 2% body weight.

Results

SIDEWAYS PUSHES

Figure 1 shows the surface recorded EMG after a sideways push of a control subject and a hemiparetic patient after a push to their hemiparetic and unaffected side. Table 2 shows the average latencies, and figure 2 shows the median amplitudes for both groups, expressed as percentage increase in IEMG activity over baseline. In the control subjects, a push to the

Figure 2 Median (interquartile range) increase in integrated EMG activity over baseline after a sideways push. Activation of muscles in the hemiparetic limb is reduced and activity in the unaffected adductor is greater than normal. * $p < 0.05$; ** $p < 0.001$ *v* controls.

right was resisted initially by an increase in activity in the right gluteus medius (330%, 74 ms), and this was followed by a smaller increase in the left adductor activity (70%, 111 ms), and a push to the left elicits the same response in corresponding muscles on the other side.

In the patients with stroke, after a push to the hemiparetic side, there is a smaller, later, increase in activity in the hemiparetic gluteus medius (75%, 96 ms), but activity in the contralateral adductor is greater than usual at latency similar to the control group (170%, 110 ms).

After a push to the unaffected side, the unaffected gluteus medius shows a clear response at a latency slightly longer than controls (200%, 83 ms), and there is either a small and delayed or no response in the hemiparetic adductor (10%, 144 ms).

STEPPING

Figure 3 shows the EMG and ground reaction of a control subject taking a step from rest, leading with the right leg (above) and left leg (below). The initial sideways weight shift is accompanied by increases in activity of the gluteus medius of the stepping leg and contralateral adductor, indicated by the box. Lifting the weight off the swing leg is accompanied by large increases in activity of the stance leg gluteus medius and swing leg adductor. At heel strike, weight is transferred to the other leg and the corresponding gluteus medius and adductor become active. The pattern of contralateral activation of gluteus medius and adductor during the self initiated sideways weight shift is similar to that seen after a sideways push, indicated by the box in the right column of figure 3.

Figure 4 shows a typical response in a right hemiparetic patient at gait initiation (left column) and after a sideways push (right

Table 2 Muscle onset latencies after a sideways push (ms)

	Gluteus medius		Adductor	
	Mean	SD	Mean	SD
Controls	74	8	111	16
Strokes: strong muscles	83	10	110	30
Strokes: hemiparetic muscles	96	20	144	39
p Control <i>v</i> strong	<0.01		NS	
p Control <i>v</i> hemiparetic	<0.001		<0.05	
p Strong <i>v</i> hemiparetic	<0.05		NS	

column). The pattern of muscle activation during the self initiated sideways weight shift again mirrors that seen after a sideways push—that is, absent or very impaired activation of the hemiparetic muscles, with more prominent

activity in the unaffected adductor. The absent hemiparetic muscle responses are marked with asterisks. By contrast, the pattern of activation during stepping with either leg is relatively normal, with marked phasic recruitment of the

Figure 3 Control subject's averaged EMG at gait initiation (left) and in response to a sideways push (right). The different phases of the gait cycle can be identified from the ground reaction force data. Before stepping with the left leg, the weight is moved towards the right by the left gluteus and right adductor (bottom). As the entire body weight is transferred to the right leg, right gluteus medius activity peaks and the left adductor is active during left leg swing phase. Activity of the left gluteus medius increases as weight is transferred to the left leg, and right adductor activity increases during right leg swing phase. Postural activity during the initial sideways weight shift and after a sideways push is enclosed in boxes. The pattern of muscle activation is similar in these two tasks.

Figure 4 Right hemiparetic subject's averaged EMG at gait initiation (left) and in response to a sideways push (right). Postural activity during the initial sideways weight shift and after a sideways push is enclosed in boxes. The pattern of muscle activation is similar in these two tasks, with absent or very diminished activity in the hemiparetic muscles. By contrast, the pattern of hemiparetic muscle activation during stepping is relatively normal. * Loss of normal response in hemiparetic muscles

hemiparetic gluteus medius when standing on the right leg, and of the hemiparetic adductor when stepping with the right leg.

To compare muscle activation in standing balance and stepping, each subject's IEMG after a sideways push was expressed as a percentage of the (greater amplitude) IEMG in the stepping phase of gait (fig 5). This shows that, compared with controls, recruitment of the hemiparetic gluteus (Mann-Whitney $p < 0.05$) and adductor ($p < 0.001$) was relatively smaller in standing balance than stepping. The unaffected adductor was recruited more than in controls ($p < 0.05$).

Discussion

When a hemiparetic patient is pushed while standing, the normal pattern of muscle activity is altered to compensate for the weak side. The unaffected adductor is recruited as early as possible and with greater amplitude than normal. This increased response compensates for the weak and delayed response of the hemiparetic muscle. This modified motor programme presumably has to be learnt as part of the rehabilitation process. Theoretically, the unaffected leg should be able to generate a greater sideways force when more of the body weight is on this leg, which may partially

Figure 5 Activity of muscles in standing balance compared with gait, expressed as the median increase (interquartile range) in individual muscles' IEMG after a sideways push as a percentage of peak activity in the same muscle in gait initiation. Recruitment of the hemiparetic gluteus and adductor in standing balance is reduced relative to gait. * $p < 0.05$; ** $p < 0.001$.

explain why most patients with stroke tend to stand in this position naturally.¹¹ If this is the case, patients who have not recovered normal postural responses in the hemiparetic limb should not be discouraged from standing with more weight on the unaffected leg.

By contrast with the reduced activation in standing balance, the pattern of hemiparetic muscle activation during the stepping phase of gait was essentially normal. It is only during the self initiated sideways movement before stepping that the pattern clearly differs from controls. This resembles the abnormal pattern seen in response to a sideways push. This finding suggests different central pathways for the postural changes at initiation of gait and regular stepping. The only previous report comparing EMG activity in gait with voluntary or other tasks¹² reported a similar contrast between normal activation of the hemiparetic tibialis in the stepping phase of gait, and loss of activity at gait initiation.

Other workers have also shown the same abnormal patterns of activation in self initiated movements and after external perturbation.¹³ Similar abnormalities are seen in hemiparetic muscles making associated postural adjustments.^{14,15} By contrast, coordinated stepping movements and rhythmical EMG activity during treadmill gait are preserved in decerebrate or paraplegic cats,^{16,17} dogs,¹⁸ and humans after spinal cord injury.^{19–21} Patients with stroke are able to step on a treadmill even though voluntary leg motion is clinically impaired.^{22–27}

The greater disruption of muscle recruitment during the generation of sideways force suggests that postural responses when maintaining standing balance, or making self initiated movements are dependent on descending input from the brain.^{21,27} Functional stretch reflexes, musculocutaneous reflexes and postural responses are abolished after lesions of the dorsal columns, cervical cord, brainstem, internal capsule and sensorimotor cortex.^{28,29} This suggests that they are controlled by the brain although it is not clear if the afferent pathway ascends in the cord, in a "long loop" to synapse in the brain stem, basal ganglia, cerebellum, or cortex. An alternative method allowing the brain or higher centres to control the multisegmental response is by selective facilitation of transmission ("gating") or depolarisation among interneurons in adjacent segments of the cord.^{26,30} By contrast, stepping is thought to be orchestrated by a signal generator in the lumbar spinal cord,^{19,31–35} which continues to function when it is no longer under supraspinal control after functionally complete spinal cord injuries.

The pattern of hemiparetic muscle activity in postural tasks (resisting external and generating internal sideways forces) and in stepping supports the view that these two tasks are neurophysiologically distinct. At present patients with stroke are taught to stand before they are taught to walk, because standing balance is a prerequisite for independent transfers and unsupported walking. However, these results support the earlier start of gait training, without waiting for standing balance to be achieved. This can be done by supporting the patient in a harness over a treadmill, which has the added advantage of allowing partial weight support if necessary.^{36,37}

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