Balancing as a clinical test in the differential diagnosis of sensory-motor disorders

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Summary During balancing on a seesaw normal individuals have a mean sway oscillation of 4.3 Hz, which is significantly lower (3.3 Hz) in patients with peroneal muscular atrophy. It is assumed that the oscillations in both cases are generated by spinal stretch reflexes and that the lower frequency in patients with peroneal muscular atrophy is due to their slower nerve conduction velocity. The balancing movements are altered when spinal stretch reflex activity is reduced by ischaemia but are normal in patients with a dorsal column lesion despite a similar sensory loss. The analysis of balancing movements can be a diagnostic tool in differentiating several sensory-motor disturbances.

As already described by Romberg in 18511 somesthetic sensations originating from the legs play an essential rôle in human postural regulation. A quantitative analysis of body sway in the absence of proprioceptive afferents has shown a typical 1 Hz anterior posterior instability.2 These studies were performed on stable ground. Even more important is the proprioceptive feedback during balancing on unstable ground, because quicker control is required for the maintenance of equilibrium. Normal people show in this condition a predominant 4 to 5 Hz body oscillation which is supposed to be generated by spinal stretch reflexes of the leg muscles.3 This assumption is based on the balancing latencies which are compatible with the segmental stretch reflex delay and the fact that these balancing oscillations could not be recorded in conditions with abolished or reduced spinal stretch reflexes (as in patients with tabes dorsalis or in normal people after block of group I afferents by ischaemia.4 A decrease therefore, in the main balancing frequency connected with an increase of instability would be expected in a condition with slower nerve conduction velocity.

The characteristic balancing movements were investigated in patients with peroneal muscular atrophy with nerve conduction velocities reduced to about 50% of normal but without clinically evident paresis. The results obtained were compared with the balancing characteristics of patients' with other disturbances of the afferent pathways and with cerebellar disorders. It is concluded that typical balancing sway parameters can be distinguished in different sensory-motor disturbances.

Patients and methods
To investigate the influence of reduced nerve conduction velocity on the balancing mechanism, six patients with peroneal muscular atrophy were examined and compared to 10 normal individuals of the same age group. Peroneal muscular atrophy is a hereditary neuropathy usually passed on as a dominant trait which becomes apparent in childhood or adolescence, and some patients have a dramatic slowing of motor conduction velocity.5 Relevant clinical data of the patient group are shown in the table. None of them had subjective complaints of muscle weakness and only in two was slight muscular atrophy of the foot muscles found. Four patients were examined electromyographically only because they had elder relatives with clinically manifest peroneal muscular atrophy.

Subjects stood with eyes closed on a seesaw which was placed on a force-moment measuring platform. The anterior–posterior sway of the seesaw was measured as displacement of the centre
of force by four piezo-transducers in the corners of the measuring platform. The ankle angle, the hip and head tilt were recorded by goniometers in the anterior–posterior direction. Simultaneously the surface EMG of the tibialis anterior and gastrocnemius muscles was registered.

The power spectra of the centre of force displacements were calculated by a fast Fourier analyser (Nicolet, Minibiquitous 444 A). The sampling period lasted 160 seconds. The dominant frequencies of head, hip and ankle angle tilt are equal to those obtained from the centre of force displacements (unpublished observations).

In two normal subjects, balancing was also recorded during a partial ischaemic block of the muscle spindle afferents of both legs when the efferent motor nerve fibres were still intact. Thereby the contribution of group I afferents to the balancing movements was demonstrated. The detailed method by which the partial ischaemic block was achieved and its physiological background is described elsewhere.2 3 6–8 For comparison the sway characteristics of two patients with a loss of vibration and position sense in both legs due to a dorsal column lesion by demyelination were investigated (table).

**Results**

When standing on a seesaw normal people compensate for the yielding of the unstable ground by
Balancing as a clinical test in the differential diagnosis of sensory-motor disorders

short reciprocal activity bursts of the antagonistic leg muscles acting at the ankle (fig 1 upper part). Normally the main frequency of the resulting anterior–posterior oscillation was 3.9 to 5 Hz as evaluated by the frequency peaks in the Fourier spectra for the displacement of the centre of force and the movements of angle at ankle, hip and head (fig 2A). The amplitudes of the rapid angle fluctuations during balancing were up to 5° and the stretch velocity reached 150° to 170°/s.

From physiological studies\(^9\)\(^ {10}\) it can be assumed that in a balancing motor task as described here, spinal stretch reflexes in the leg muscles are present due to an increased reflex gain, although tendon tap reflexes are reduced or abolished. It is therefore suggested that these fast fluctuations are the consequence of segmental stretch reflex activity of the leg muscles.

Considering the segmental stretch reflex latency and the electromechanical coupling time, the threshold in ankle angle displacement which is needed to evoke a significant reflex contribution must be below 5° which is the mean ankle angle change measured in our normal subjects. From studies in the cat\(^ {11}\) it can be deduced also for humans that ±1% change in the muscle length is sufficiently suprathreshold for the activation of primary muscle spindle afferents. In other studies on human subjects\(^ {12}\)\(^ {13}\)\(^ {14}\) it has been demonstrated that ±1° change in angle joint corresponds to approximately ±1mm of length change in the triceps surae muscle corresponding to ±0.5% of its total length. Therefore in humans an ankle change of about 2° should already be well above threshold for the activation of muscle spindles.

It was shown by the original recordings for one patient with peroneal muscular atrophy in fig 1 (lower half) that the amplitude of the ankle angle oscillation was much higher (above 10° in the average). Consequently the displacement of the centre of force and head and hip movements was larger, which is an expression of an enhanced instability on the seesaw. As can be seen from the Fourier analysis (fig 1, right half) for this patient, the sway frequency peak is considerably lower than the normal.

These typically altered sway parameters were found in all patients with peroneal muscular atrophy whose nerve conduction velocities were about 50% of normal values. Fig 2B gives the in-

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**Fig 2** Individual Fourier spectra of 6 normal subjects (A) compared to those of 6 patients with peroneal muscular atrophy (B). The dotted area gives the range of the frequency peaks in normals (average frequency 4.3 Hz, n=10) and in patients (average frequency 3.3 Hz, n=6).

For graphical reasons the spectra are shifted as indicated by the scale on the ordinate.
dividual spectra of six patients compared to six normals (fig 2A). The dotted area represents the range of peak frequencies for both groups. It may be observed that both groups can easily be separated because none of the frequency peaks of patients overlaps with those of normals and the frequency difference is significant. The additional frequency peaks at 8 to 10 Hz seen in the power spectra of some normals and some patients, coincide with the physiological tremor rate.

As can be seen from the power spectra of fig 2 the sway frequency is lowered on average from 4.3 Hz for normals to 3.3 Hz for peroneal muscular atrophy patients. This means, that one sway cycle in patients is about 70 ms longer than in normal subjects. This additional time needed for one balancing cycle, which itself presumably involves the spinal stretch reflex of tibial anterior and of triceps surae muscles, must be expected if the nerve conduction velocities are reduced to about 50% of normal. The larger sway amplitudes in patients with peroneal muscular atrophy are presumably the consequence of the slower compensation mechanism during balancing movements.

It was proved that the postural oscillations were not directly related to the body inertia and the natural frequency of the body, by imposing 30 kg on normals and patients which did not alter the predominant sway frequency.

By the close correlations between the stretch reflex delay and the sway frequency and amplitude it is concluded that the fast conducting segmental stretch reflex is essential for quick balancing movements which are necessary to hold equilibrium. Patients with slower nerve conduction velocity are more unstable compared to normal subjects despite the absence of overt sensory or motor symptoms. From these results the question arises whether a subject is capable of balancing without or with a defective spinal stretch reflex mechanism. It was shown that the former condition is present in patients with tabes dorsalis and in normal subjects after ischaemic blocking of group I afferents of the leg muscles, when no H-reflex could be elicited in the gastrocnemius muscles. Standing on a seesaw these subjects are extremely unstable and the typical balancing frequency of 4 to 5 Hz is abolished (fig 3). Their balancing movements often have a main frequency around 1 Hz when the eyes are closed. This sway characteristic probably is due mainly to vestibularly-induced trunc movements. It is also present during almost exclusively vestibularly controlled standing on stable ground. Therefore balancing is severely hampered when no spinal stretch reflex is active.

Fig 3  Balancing reactions before and during ischaemic blockage (22 min) of group I afferents from both legs in a normal subject. Original recordings of leg muscle emg, head and hip movements, ankle angle and centre of force displacement (left half). Corresponding Fourier spectra of the centre of force (right half). During ischaemic blockage of the afferents the predominant rapid balancing movements are abolished and slower frequencies appear.
Balancing as a clinical test in the differential diagnosis of sensory-motor disorders

Fig 4  Fourier spectra of the centre of force displacement during balancing in a typical normal subject compared to a patient with dorsal column lesion with deep sensory loss on both legs and a patient with peroneal muscular atrophy. Whereas both the normal subject and the patient with dorsal column lesion have their balancing frequency peak well above 4 Hz (4.35 Hz and 4.8 Hz respectively as indicated by arrows) the frequency peak in the peroneal muscular atrophy patient is well below at 3.5 Hz (dotted line).

Patients with peroneal muscular atrophy often present signs of dorsal column atrophy as with a slow peripheral nerve conduction velocity. Therefore we studied patients with isolated dorsal column lesions who showed neurological defects similar to those of patients with tabes dorsalis and to those in normal subjects after ischaemic blocking of group I afferents of the leg muscles. Clinically they presented with loss of deep sensation and reduced position sense in ankle and knee joints. However, these patients with a dorsal column lesion in our study had an intact spinal stretch reflex pathway as shown by preservation of the electrically induced H-reflex. In agreement with the segmental stretch reflex hypothesis for rapid balancing movements, these patients with dorsal column lesions showed the typical frequency peak of normals when standing on a seesaw. Fig 4 shows the Fourier spectrum of the centre of force displacement for one of these patients in comparison to a normal subject and a patient with peroneal muscular atrophy who exhibits a significant frequency reduction to 3.5 Hz.

Discussion

Balancing on a seesaw is a very simple provocative test which reveals specific sway characteristics even in patients with minor neurological defects. In normal subject it was shown that the predominant sway frequency around 4.3 Hz is caused by segmental stretch reflexes of the leg muscles. We suggest that the slower balancing oscillations around 3.3 Hz in patients with peroneal muscular atrophy are due to the same mechanism as in normal subjects. The lower sway frequency and larger sway amplitude in these patients can be explained by the considerably slower nerve conduction velocity. However, if this stretch reflex mechanism is defective as in ischaemic blocking of group I afferents the sway characteristic is basically altered and typical 4 to 5 Hz balancing oscillations are abolished. In patients with dorsal column lesions who had intact spinal reflex mechanisms the quick balancing movements are preserved despite the loss of deep sensation of both legs.

A predominant sway frequency around 3 Hz, comparable to that of peroneal muscular atrophy patients, was also found in patients with late cortical cerebellar atrophy. In contrast to the findings in the patients with peroneal muscular atrophy, the postural oscillations in this disease also were present while standing on a stable floor. These oscillations in the cerebellar patients, who had normal nerve conduction velocities, are suggested to be the result of delayed long latency reflexes (unpublished observations).

The experimental condition described here is quite different from the posture control during standing on a stable ground. For the latter some authors stress the importance of suprasegmental reflex loops for the maintenance of upright standing. In our balancing experiments, however, the muscle stretch velocities for the anterior tibial and triceps surae muscles were much higher and were in the range in which significant spinal reflex activity can be expected. Therefore it is concluded that spinal reflex delays are responsible for the altered balancing frequency. The possibility cannot be excluded, however, that additional long loop reflexes are acting in balancing tasks which stabilise slower body shifts.

The method described here is to our know-
ledge the first attempt to use balancing reactions as a simple and convenient diagnostic test in the differentiation of sensory-motor disorders. Applied in addition to the Romberg test it has the advantage that it is more sensitive and gives characteristic deviations from normal even in the first subclinical stages of sensory-motor disorders.

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References
