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Short report

Quantitative analysis of stance in ataxic myxoedema

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SUMMARY Postural abnormalities in a 66-year-old woman with ataxic myxoedema were recorded by stabilography during treatment. After replacement therapy, 3 Hz power in the sagittal plane was greater than before treatment; this peak shifted to a higher frequency and finally its ratio became normal. This is the first report of recovery of a 3 Hz body oscillation in ataxic myxoedema.

Recently a regular fore-and-aft body oscillation, with an average frequency of 3 Hz, has been reported in patients with superior vermal atrophy.[1 2] The superior vermis is prominently affected in ataxic myxoedema.[3–5] This paper reports a 3 Hz body oscillation in a case with ataxic myxoedema and its change during therapy.

Case report

A woman aged 66 years was admitted on 19 February 1981 because of anorexia and falling. Over the last 20 years she had become increasingly slow in movements and intolerant to cold. Five years earlier she had noticed bilateral deafness, thinning of hair and unsteadiness of gait. On examination she was slightly somnolent and spoke and moved slowly. Cranial nerves were intact except for bilateral deafness. Muscle strength was normal. There was moderate ataxia in the trunk and lower limbs. Gait was wide-based and she fell easily. Romberg's sign was negative. The deep tendon reflexes were absent in all limbs. No pathological reflexes were observed. Sensation was normal except for diminished appreciation of vibration in the distal lower limbs.

Investigation showed: serum sodium 104, potassium 4·3, chloride 75 mmol/l; CK 4,360 IU (normal: 41–129), total cholesterol 314 mg/dl. Plasma osmolarity was 216 mosm/l (normal: 285–295) while urine volume was normal. BMR −33·5%, triiodothyronine 0·2 μg/ml (normal: 0·8–1·8), thyroxine 1·0 μg/dl (normal: 4·5–13·0), TSH 34·0 μU/ml (normal: less than 10), ^{131}I uptake 4·1% (normal: 10–40). Electroencephalogram showed diffuse slow wave activity at 5 to 7 Hz. Electromyogram showed myopathic change. Nerve conduction velocities were normal in upper limbs and decreased or not elicited in lower limbs. Otoneurological tests showed bilateral perceptive deafness and hypoactive caloric responses. Eye tracking test showed normal pursuit movement; optokinetic nystagmus was normally induced in the horizontal plane but suppressed in the vertical. Computed tomography showed slight atrophic changes in the superior vermis and pons.

After therapy for hyponatraemia, her conscious level improved but she was still unsteady and slow in movement. Thyroid replacement therapy was started on the 13th day in hospital with l-thyroxine 75 μg/day. This caused immediate improvement of ataxic gait. Seven days after replacement therapy she could remain standing even in the heel-toe posture. She was discharged on 25 March 1981 with normal neurological findings except for absent deep tendon reflexes.

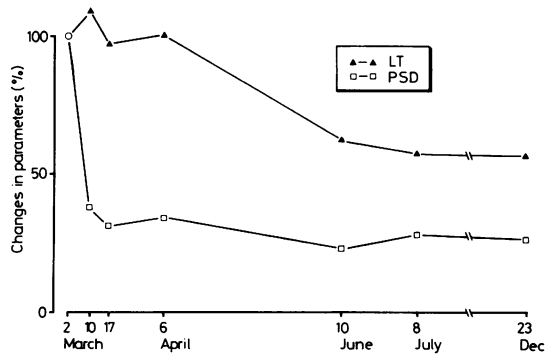

Fig 1 *Changes in sway parameters. The values 2 days before therapy are taken as 100%. LT shows no decrement while PSD shows remarkable decrements at the first to third recordings after therapy. LT: length of the trajectory for 30·72 seconds, PSD: product of the standard deviation of force at lateral direction by that at antero-posterior direction.*

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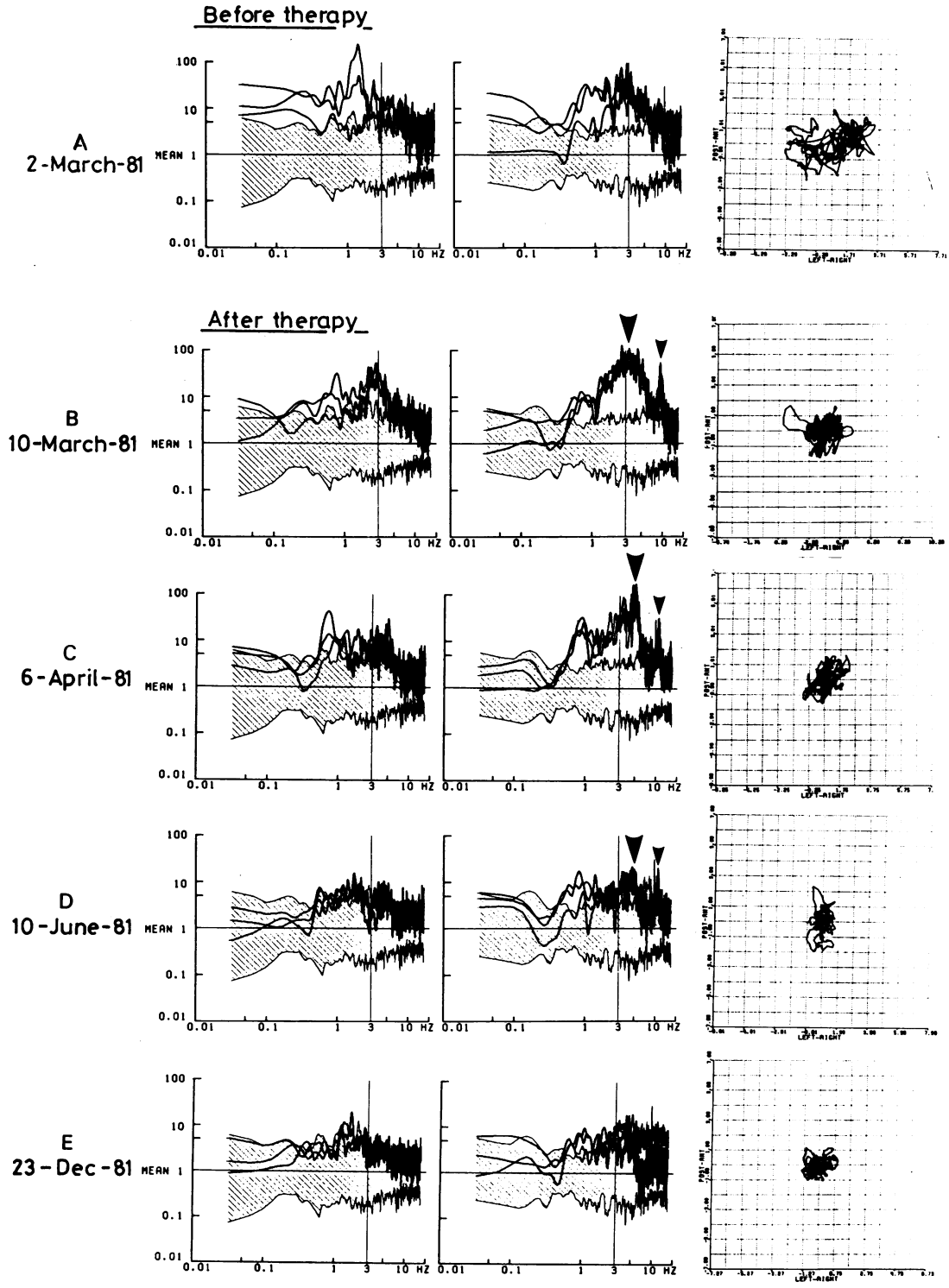

Fig 2 *Changes in the ratios of power (X: lateral, Y: antero-posterior direction) and trajectories of the centre of foot-pressure (Z). After therapy power ratios at lower frequencies diminished and widely spread high power ratios at around 3 Hz (large arrow) and at 9 Hz (small arrow) became evident (B). Follow-up study showed (C–E) that the 3 Hz peak shifted to a higher frequency as the high frequency peak, from 9 to 11 Hz, and both diminished in amplitude. Shaded areas show the range of power ratios of 30 normal subjects. Three records are superimposed in X and Y.*

Method

Instability in the upright posture was measured with a force-moment measuring platform (Sanei Sokuki IG02). The patient was instructed to stand as still as possible on the platform with both feet close together. The reaction moments about lateral (X) and antero-posterior (Y) axes were recorded on FM magnetic tape. These data were sampled at the rate of 33·3 points/second through an analog-to-digital converter and 30·72 seconds segments were subjected to Fourier analysis. Before computing the power spectrum, the digitalised data were standardised by the following formula: $Xi = Xi \times 160/H \times 60/W$, $i = 1, 2, \ldots 1024$, where Xi is the data, and H and W are respectively the height and weight of the subject. The power spectra of 30 normal subjects (mean age: 50) were computed. To demonstrate the changes in very small powers at higher frequencies clearly, the ratio of the power of the patient to the mean power of normal subjects was calculated. Some other sway parameters, the length of the trajectory of the centre of force for 30·72 seconds (LT) and the product of the standard deviations of forces in the X and Y axes (PSD) also were computed. Body sway at lower frequencies is more strongly represented in PSD, while that at higher frequencies is evident in LT. The recording was performed three times in succession before and in the course of therapy.

Results and discussion

The changes in sway parameters are shown in fig 1. PSD decreased remarkably after replacement therapy while LT did not show any decrement at the second to fourth recordings. This corresponds to the fact that body sway at lower frequencies diminished first and that at higher ones later. The power spectrum analysis shows this point more clearly. Before replacement therapy, when she was unsteady, power ratios at lower frequencies were prominent (fig 2A). This slow body sway may be partially due to reduced vestibular function suggested by hypoactive caloric response. After therapy two peaks at 3 Hz (fig 2B large arrow) and 9 Hz (small arrow) in the Y axis became more evident than before therapy. One month after therapy, when she was normal except for diminished Achilles tendon reflex, the 3 Hz peak shifted to 5 Hz and the 9 Hz peak to 11 Hz (fig 2C). The same frequency change was observed in the arm movement in the recovery course of a monkey with ablation of the cerebellum.[6] The changes in high frequency peak from 9 to 11 Hz may be caused by improved stretch reflexes.[7] The changes in muscle dynamics could not be responsible.[8] Three months after therapy, all the frequency peaks became normal (fig 2D, E).

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