

Abnormal head movements

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SUMMARY Abnormal head movements have been studied in a variety of diseases using objective recording techniques and the data analysed with respect to the frequency content of the movement. Flopping, nodding, tic, chorea, myoclonic jerks, and most head tremors involve frequencies of approximately 2 and 4 Hz which correspond to the natural fundamental and second harmonic resonances of the head as determined by the mechanical properties of the head/neck system. These findings provide a basis for classification of abnormal head movements as well as an explanation of the characteristics of those arising from hypotonia of the neck muscles. The similarities between tremor frequencies and natural resonances suggest that in the case of the head, tremor arises from disorders of neural mechanisms normally responsible for the fine control of voluntary head movement and for stabilisation of the head during disturbance of posture. Head movements in cases of congenital nystagmus were found to be of two types. Some were of bizarre waveform, in no way assisted vision, and were taken to be of primarily pathological origin and classified as tremors. Others were learned adaptive responses which assisted vision either by interrupting the nystagmus, as in the case of spasmus nutans, or by compensating for the nystagmus with an inverse waveform and were called nodding. A prerequisite for true compensatory nodding is modified vestibulo-ocular reflex.

Head movements differ from movements of other body parts in two important ways. Firstly the head is responsible for the directional orientation of the special senses and its movements are influenced by the information these provide. It is, therefore, not unexpected that certain disorders of the special senses may lead to unusual head movements and that disorders of head movement may force unusual conditions upon the special senses. Secondly, the mechanical properties of the head/neck system, which normally influence head movements, also determine many of the characteristics of abnormal head movements. In this study we examine abnormal head movements and analyse them in terms of these mechanical properties and in terms of their interaction with the special senses.

Fundamental dynamics of head movement

Head movements have complicated trajectories be-

cause the cervical spine, which is primarily responsible for head movement, is a jointed-rod system with alternative ways of achieving a given displacement (Fielding, 1957; Barnes and Rance, 1974; Viviani and Berthoz, 1975). This is illustrated in Fig. 1 which shows the trajectories of a point of light on the occiput of a normal human subject during head shaking and nodding at different frequencies. The path the light makes during the down stroke of the head is not necessarily the same as during the up stroke and during severe tremors these differences may become exaggerated. For most of our purposes, however, it may be assumed that the head makes fairly simple rotations about one universal joint.

Secondly, as a mechanical system with viscoelastic properties, the head/neck combination is associated with certain natural resonant frequencies of movement.

Barnes and Rance (1974) have investigated these resonances in normal subjects by oscillating the whole body and observing the resulting uncontrolled oscillations of the head. Their results showed that there was a fundamental resonant frequency at approximately 2 Hz and a second

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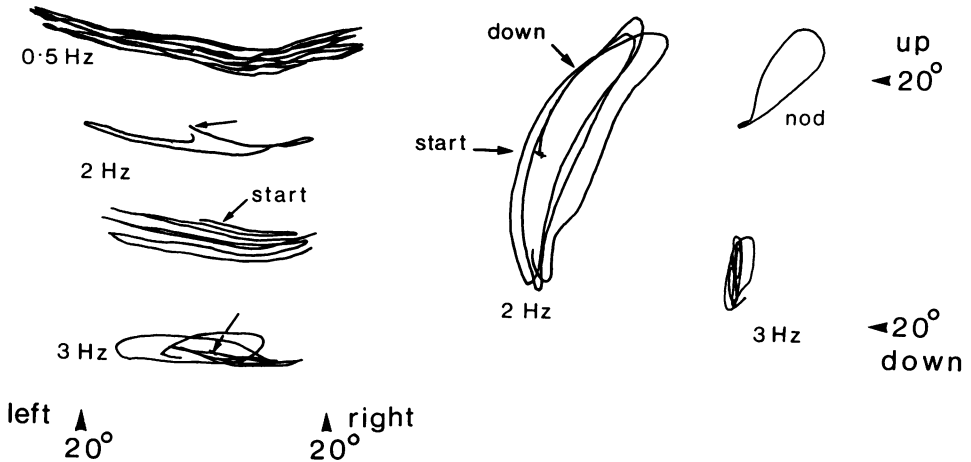


Fig. 1 Loci of a point of light mounted on the occiput of a normal adult subject nodding and shaking his head at various frequencies while seated. During vertical movements the head is viewed from the side. During horizontal movements the head is viewed from above. Arrows indicate the starting point of the head movement. The movement of the light sources were transduced by means of a Schottky barrier photodetector mounted in the focal plane of a 35 mm camera placed to view the head.

harmonic component at approximately 4 Hz which results from the geometry of head/neck articulation.

The same two frequencies can be identified during voluntary head movements and are illustrated in Fig. 2. In A the recordings present a normal head movement made to fixate a target in lateral gaze. The head moves in an S-shaped trajectory, the movements being a reasonable approximation to the response of a second order system with visco-elastic properties which are slightly over-damped. Co-ordinated with the head movement there is a stereotyped eye movement consisting of a saccade towards the target (sac) followed by compensatory, primarily vestibulo-ocular reflex movements (VOR). The VOR maintains stable fixation during the head movement. The duration of the head movement is about 500 milliseconds which suggests that the system should have a natural frequency of just above 2 Hz. Such a pattern of co-ordination occurs in man (Crawford, 1960; Bartz, 1966; Gresty, 1974) and animals (Collewijn, 1977).

In Fig. 2, trace B, the subject is asked to make a violent head movement sideways which he would not normally do; as a result the head moves faster and at the termination of the movement produces one cycle of oscillation at a frequency of about 4 Hz. This, for convenience, we have referred to as the second harmonic resonance, but in truth it will have active myogenic components mixed with

the passive response.

During everyday activities the tendency of the head/neck system to resonate is damped out. When one considers the high inertia of the head it is evident that the forces responsible for this must be quite powerful.

Subjects and methods

Patients with head movement disorders were surveyed prospectively over a 12 month period at the National Hospital, Queen Square and the Hospital for Sick Children, Great Ormond Street, London. Head movements and movements of other body parts were examined by a photoelectric method previously described (Gresty *et al.*, 1976). In some patients, eye movements were also recorded using direct coupled electro-oculography. In most cases supplementary use was made of closed circuit video recording.

Classification of abnormal head movements

The head may be affected by any of the five basic types of dyskinesia in Marsden and Parkes' (1973) classification which lists tremor, tic, chorea, myoclonus, and dystonia. However, in addition, the head is subject to two dyskinesias which we call "flopping" and "nodding."

We have previously distinguished two types of

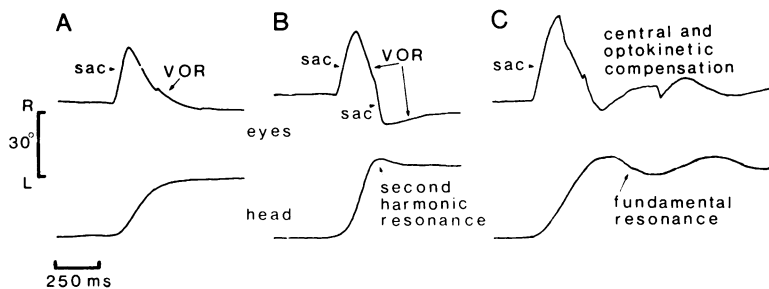


Fig. 2 Patterns of co-ordination of the head and eyes in the horizontal plane during voluntary movements to fixate targets in lateral gaze. (A) Normal stereotyped response consisting of an overdamped head movement, the onset of which is synchronised with a voluntary saccade to the target (sac). During the latter part of the movement the direction of the eyes in space is stabilised by predominately vestibular, "doll's head" reflexes (VOR). (B) Head and eye movements of a normal subject trying to move his head very quickly. The head overshoots the final displacement and oscillates for one cycle which reflects the natural second harmonic resonance of head movement although in reality the movement also contains active myogenic components. The VOR cannot generate fast enough eye movements to compensate for such a rapid head displacement and when the head overshoots the target a second saccade is made to acquire the target position. (C) Head and eye movements of a patient who had lost labyrinthine function one week before the recording as a result of gentamicin toxicity. Removal of labyrinthine control of the neck musculature reduced its tone with the result that the head became underdamped, thus during active or passive movement the head oscillated for several cycles at the fundamental resonant frequency of 2 Hz.

head dyskinesia associated with congenital nystagmus. One is essentially a learned movement which helps the child overcome his nystagmus and improve vision (Gresty *et al.*, 1976), and we classify this as nodding. The other resembles the nystagmus in waveform, and is a true disordered movement probably generated by the same pathological mechanism (Gresty *et al.*, 1978), and here it is classified as a tremor.

FLOPPING

Flopping is a passive, involuntary movement characterised by transient, exponentially decaying, pendular oscillations, occurring at the end of active head movement or when head posture is disturbed by body movement.

In the normal subject the tendency for the head to resonate is well controlled by damping due to neck muscle tone and by control signals which correct for external disturbances. However, when muscle tone is reduced or the braking signal which arrests movement is inadequate, the head/neck system becomes underdamped and tends to oscillate both at the termination of voluntary movement and when head posture is passively disturbed by other body movements. This produces

the appearance of a "floppy head." The head may oscillate in the horizontal or vertical planes; however, gravity contributes to the disturbance in the vertical plane. Sudden loss of labyrinthine function produces a transient reduction of neck muscle tone. Figure 2, C shows the effect of such hypotonia on the voluntary head movements of a patient who had lost all labyrinthine function as a result of gentamicin toxicity one week before the recording. At the end of the voluntary displacement the head tends to swing from side to side at the fundamental resonant frequency of about 2 Hz. Several cycles of oscillation occur before the motion comes under control. Such passive oscillations of the head are particularly caused by unexpected movements which "jar" the whole body. Flopping occurs in any disorder which causes neck hypotonia: neurogenic or myogenic muscle atrophy, cerebellar syndromes, cervical deafferentation, and absent labyrinthine function.

Cerebellar syndromes, especially in multiple sclerosis, may be associated with both head flopping and head tremor. In such cases frequency analysis will clearly distinguish the 4 Hz tremor components of the dyskinesia from the 2 Hz head flopping (see below).

TIC AND NODDING

These are acquired behavioural patterns, substantially under voluntary control and not the direct result of some basic pathology but rather, an adaptive response to a pathological condition. A tic is a single, rapid, stereotyped movement, occurring intermittently and on appearances difficult to distinguish from chorea or myoclonus. Nodding is an active, regular, sustained, usually pendular oscillation consisting of 2 and 4 Hz frequency components. Several tics occurring in sequence take on the appearance of nodding. Nodding is a sustained rhythmic movement which on waveform alone is indistinguishable from a tremor. Nodding and tic are in some sense voluntary, as each may be suppressed or imitated by the patient, and each tends to occur when the patient's attention is drawn to it. Two conditions in which these occur are:

1. *Tic and nodding of neurotic origin* These movements appear to have no direct biological value and to have no organic pathological cause. Nodding usually takes a 4 Hz sinusoidal waveform and may be accompanied by synchronised gentle rocking of the torso. The reader may prove to himself how easy it is to produce continual nodding at this frequency by shaking his head from side to side as fast as comfortably possible. It becomes evident that there is a natural rhythm (of 4 Hz) which is comfortable and can be sustained.

2. *Congenital nystagmus with head nodding* Certain patients with congenital nystagmus report an improvement in vision when they shake their heads. These head movements are presumably an adaptive behavioural strategy and classified here as head nodding.

During normal visual fixation on a target, the direction of visual fixation is the simple sum of the position of the eyes in the head and the direction in which the head is pointing. The compensatory "doll's head" reflex, which consist of a combination of vestibular and optokinetic reflexes, normally works to preserve the direction of fixation by producing movements of the eyes which are equal in magnitude yet opposite in direction to head movements. The two thus cancel leaving the direction of fixation undisturbed.

The problem of a patient with congenital nystagmus is that his eyes are moving relative to the object of fixation, producing image movement across the retina, thus degrading vision. Firstly, one may consider the effect of head movement on someone with nystagmus who has normal doll's head, and in particular vestibulo-ocular reflexes. As before, the direction of visual fixation is determined by the sum of the position of the eyes in

the head and the direction of the head itself. In this case the sum consists of the head movement less the compensatory eye movement plus the ongoing nystagmus. The normal compensatory eye movements, of course, cancel with the head movement leaving the actual direction of fixation still determined by the nystagmus. Therefore, a patient who possesses normal compensatory eye movement reflexes cannot ordinarily use his head to overcome the visual deficiency produced by his nystagmus.

From this analysis it follows that for head nodding to improve vision, as patients testify, the nodding must either (a) diminish the nystagmus by some means, or (b) during the nodding the compensatory reflexes must be radically altered, so that the combination of nystagmus and nodding provides periods of relatively stable visual fixation.

The cases below illustrate these two ways in which head nodding can improve visual fixation. The traces in Fig. 3 are from a child thought to have spasmodic nutans. With the head still there was a high frequency, convergent, pendular nystagmus in the horizontal plane. When looking attentively the patient would shake his head from side to side, and during this time there would be normal, compensatory eye movements without any nystagmus. This child could apparently "switch off" his nystagmus by some unknown mechanism associated with head movement and perhaps related to vestibular function.

A second child demonstrates the effect of modified vestibulo-ocular reflexes on nystagmus. He presented with a gross nystagmus with high and low amplitude components. When concentrating on a visual task he would shake his head in an

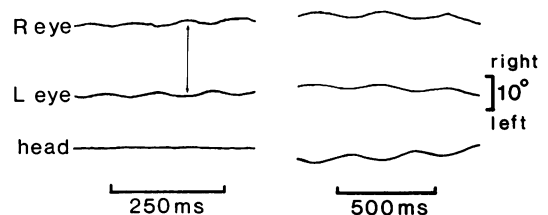


Fig. 3 Nystagmus of a child with spasmodic nutans. The nystagmus consists of high frequency, convergent oscillations of the eyes with the phase relationship indicated by the vertical arrow (right hand traces). Whenever the head moved the nystagmus ceased; thus, in the left hand traces the head is shaken from side to side and normal compensatory doll's head eye movements are evoked. The implication in this case is that the head shaking is not pathological but a learned response used by the child to "switch off" the nystagmus.

irregular fashion as illustrated in Fig. 4. During this manoeuvre, however, there was little change in the pattern of his nystagmus which clearly indicated that the head shaking was not eliciting a vestibulo-ocular reflex. If a vestibulo-ocular reflex had been evoked it would have added to the nystagmus and modified the eye movement wave form. In fact the head shaking took the same pattern as the nystagmus but was executed in the opposite direction, thus cancelling the eye movements and providing a period of relatively stable visual fixation. Although the vestibulo-ocular reflex was absent during the nodding, the presence of responses to impulsive rotational testing in darkness demonstrated that under other circumstances vestibulo-ocular reflexes were present.

These findings are consistent with those of Forssman (1964) who demonstrated the apparent absence of vestibulo-ocular reflexes in nearly half his patients with congenital nystagmus. He attributed his findings to central adaptive processes rather than to a pathological state. Similar voluntary nonvisual modification of vestibulo-ocular

reflexes has been demonstrated previously (Barr *et al.*, 1976) in normal subjects. These two cases show that the types of nodding which are frequently referred to as congenital in origin, when examined objectively, can be shown to be learned behaviour patterns of adaptive value.

CHOREA

Chorea is an active, wholly involuntary, single movement which resembles a fragment of normal voluntary movement but is random and inappropriate in timing and often exaggerated in character. Its basic frequency of 2 Hz is the same as the basic frequency of normal voluntary movement and for these reasons it is attributed to the abnormal triggering of a single movement routine from the normal repertoire of head movements (Fig. 5).

Although Marsden and Parkes (1973) classify athetosis with the dystonias, we have chosen not to include dystonia or torticollis in the present discussion of head dyskinesias but will consider athetosis alone.

ATHETOSIS

Athetotic movements of the head resemble certain voluntary movements which have low frequency content, and a typical athetotic writhing of the head contains frequencies below 1.0 Hz. It also may be an elementary part of normal behaviour which is released inappropriately. Normal voluntary movement contains slow components and "optimally timed" faster ones. Thus, for example, the head can be moved either in a slow, smooth fashion or if a refixation is made, quickly, in a time period determined by the resonant frequency, with optimal speed and accuracy. Chorea and

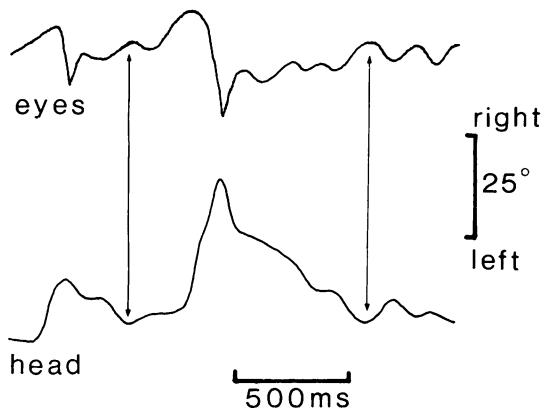


Fig. 4 Raw data records of head and eye movements during readings of a visual display by a 10 year old child with congenital nystagmus and nodding. In the absence of nodding the nystagmus was irregular. When nodding occurred the nystagmus took the form indicated in the Fig.—an overall slow drift from left to right with occasional saccadic movements to the left upon which were superimposed oscillatory movements at a frequency of 4 Hz. The pattern of head movement was more or less a mirror image of the eye movement and thus was able to compensate successfully for the nystagmus, maintaining relatively stable direction of vision. The arrows indicate the successful phase locking between head and eye movement. As discussed in the text the ability to produce compensatory head movement such as this is possible only in the absence of labyrinthine function. The scaling of the traces is accurate to within 25%.

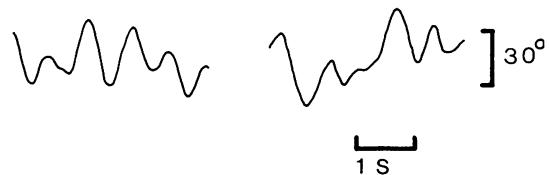


Fig. 5 Normal voluntary and choreic head movements in the horizontal plane recorded from a patient with Parkinson's disease and drug-induced chorea. In the left hand traces the patient was required to turn his head from side to side while looking at the examiner as if to test doll's head reflexes. Being a naive subject it is presumed that he made a fairly normal unselfconscious movement. The right hand traces were recorded during an episode of chorea. The traces are indistinguishable in terms of velocity or frequency content of the movement.

athetosis are similarly related in that the trajectories of the movements in each resemble those of voluntary movements. They are dissimilar in that the chorea is time optimised.

MYOCLONUS

Myoclonus occurs in two forms (Halliday, 1975). "Jerk" myoclonus consists of rapid, shock-like, at times violent contractions involving one body part at a time and responsive, sometimes excessively so, to sensory input. Figure 6B shows recordings from such a case, a 7 year old girl with a cerebellar and myoclonic syndrome from a degenerative disorder, perhaps a lipidosis. Brief, 4 Hz crescendo-decrescendo sinusoidal oscillations occur in response to a loud noise. Jerk myoclonus may be distinguished from flopping of the head in terms of frequency components and response to arousing stimuli (compare traces A and B of Fig. 6).

"Rhythmical" myoclonus when affecting the head, closely resembles tremor and is discussed below.

TREMOR

Head tremor is an active, wholly involuntary, sustained pendular oscillation that is related to rest, posture, action, and intention in the same way as limb tremor and occurs in the same diseases.

The Table gives details of 18 patients with head tremor prospectively surveyed and examined in 12 months. The most common causes of head tremor were cerebellar syndromes, essential tremor, and Parkinson's disease. Figure 7 shows

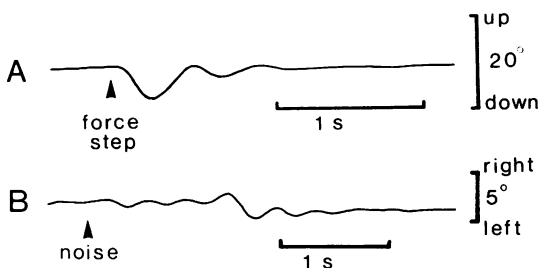


Fig. 6 (A) Head displacement in the vertical plane of a patient with bilateral loss of labyrinthine function as a result of gentamicin toxicity. The patient presses down on a lever loaded with 2 kg, the lever is suddenly released, and the patient attempts to maintain his head posture. The head oscillates at the fundamental resonant frequency of 2 Hz after the force step input. (B) Myoclonic jerk response consisting of horizontal oscillations of the head at a frequency of 4 Hz in a 7 year old boy. The myoclonus was induced by a loud noise.

the distribution of tremor frequencies and from this it is evident that there is a modal frequency of 4 Hz with lesser peaks at 2.5 and 6 Hz. The tremors occurring at 6 Hz are within the range of physiological tremor and may originate in a different neural mechanism. In some cases there is synchronisation between head tremor and tremor of other parts, in other cases there is none. The two lower frequencies of head tremor coincide with the fundamental and second harmonic resonances of the head/neck system, and therefore it is possible that some head tremors at least result from derangements of the neural mechanisms responsible for damping and fine control of head movement and for maintenance of head posture during body movement.

Deterioration of head tremor

The postural head tremor of case 3 was observed to deteriorate over a year, and the findings are presented in Fig. 8. Trace B shows that the tremor was particularly evident with head posture to the left and as in trace A, was sinusoidal in nature when the patient was first examined. The tremor was absent when the patient was lying down. One year later the tremor was no longer a simple sinusoidal movement (trace C) but appeared to be a mixture of frequencies over a wider band, its amplitude had increased slightly and it was present with the head straight. With the head turned to the left the tremor decreased in amplitude considerably and became of very high frequency, with an irregular waveform. The high frequency of the tremor was within the range of the "physiological" variety (Halliday and Redfearn, 1956). It is likely that the tremor represented in Fig. 8 C is the result of progressive deterioration and that the frequencies contained within reflect the variability in tremor frequencies found in our patients. It was felt that the high frequency tremor of trace D was a newly appearing phenomenon unrelated to the lower frequency tremor and perhaps involving the mechanisms of physiological tremor.

Head tremor associated with congenital nystagmus

Figure 9 shows the head and eye movement records of a woman with congenital nystagmus which was of the sawtooth variety when passive, but changed to the complex waveform shown during reading. Head shaking occurred only when her attention was engrossed in a visual task or when she was tired. It was of relatively small amplitude compared to the eye movements but consisted of a complex waveform which resembled the eye movement waveform in its periodicity. The

Table Patients with head tremors

Case	Sex	Age (yr)	Head tremors			Other tremors			Diagnosis
			Plane	Frequency (Hz)	Other features	Body part	Frequency (Hz)	Other features	
1	F	21	H and V	5–5.6	—	—	—	—	Multiple sclerosis with pyramidal and cerebellar deficits Kearns-Sayre syndrome with cerebellar signs Spinocerebellar degeneration Brainstem stroke
2	M	25	H	4.2	2.2 Hz flopping	Upper limb	3.7	Postural tremor	
3	M	51	H	3–4	2.0 Hz flopping	—	—	—	
4	M	56	O	2.5–5.4	? two frequencies, one just above and one just below 4 Hz	—	—	—	
5	M	81	H and V	4.0–4.6	—	Upper limbs	—	—	Essential tremor
6	F	21	H	4.0	—	—	—	—	Multiple sclerosis with pyramidal and cerebellar deficits
7	F	32	H	4.3	—	Limbs	?	Intention tremor	Multiple sclerosis
8	F	28	H	2.4	—	Upper limbs	3.5	Postural tremor	Cerebellar degeneration
9	F	38	H	2.4	Flopping at 2 Hz	—	—	—	Multiple sclerosis
10	M	68	H	6.7	—	Limbs/trunk	6.7/8.0	Postural tremor	Progressive muscular atrophy and tremor ? cause
11	F	51	H	4.0	Head tremor only when turned to the right	—	—	—	Idiopathic head tremor and dystonia
12	F	59	H	5.5–6.5	Maximal with head turned to the left	—	—	—	Idiopathic head tremor and dystonia
13	M	40	H	3.4	Head to the left	Upper limbs	3.3	Postural tremor	Cerebellar degeneration
14	F	54	H	3.4	—	Palate and larynx, finger and eyes (in vertical plane)	3.4 2.6	Synchrony of head, palate and larynx	Syndrome of palatal myoclonus
15	M	24	H	2.7–3.5	—	Palate and larynx, upper lip, eyes (vertical plane)	—	Variable usually in phase with head	Palatal myoclonus
16	F	62	H	3.4	—	Upper limbs	6.0	Resting tremor	Parkinson's disease: on Levodopa
17	M	58	H and V	2.4	Irregular tremor or chorea	Foot and finger	4.0	Resting tremor	Parkinson's disease: on Levodopa
18	F	56	H	3.8	—	Upper limbs	4 and 18	Postural tremor	? Essential tremor

Abbreviations: H = horizontal, V = vertical, O = oblique.

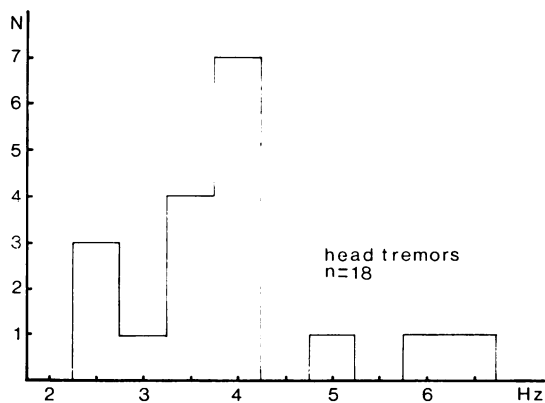


Fig. 7 Histogram of the frequencies of head tremor in the 18 patients documented in the Table. The histogram frequency resolution is 0.5 Hz.

head movement appeared to modify the nystagmus. In this patient it is quite clear that the head movement did not switch off the nystagmus but did change its waveform. We interpret these findings as indicating that the head movement elicited a normal compensatory doll's head reflex which combined with the nystagmus and did not assist in stabilising vision. The similarity between the head and eye waveforms and the preservation of the VOR suggest that both the head and eye movement abnormalities were the result of a common basic pathology. For these reasons we have called this abnormal head movement a tremor.

Mechanisms of head tremor

Because of the similarity between the frequencies of head tremor and the natural resonant fre-

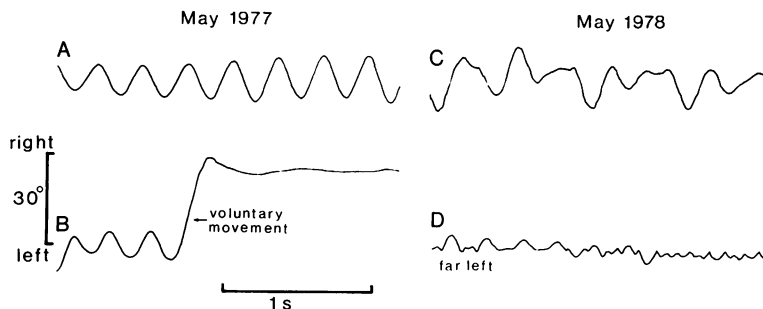


Fig. 8 Tremor of the head in the horizontal plane of a patient with spinocerebellar degeneration (case 3). The patient was observed for a year. Trace A shows the sinusoidal tremor present in the head with eccentric positions to the left. B shows how the tremor varied with head position. One year later the tremor was present in the primary head position (C) and had assumed an irregular waveform. When the head was turned to the far left a high frequency irregular tremor appeared (D).

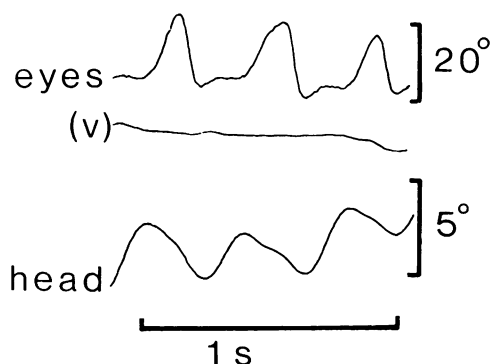


Fig. 9 Example of a type of congenital nystagmus with nodding. The recording is taken from a woman with congenital nystagmus and nodding whose head movement waveform reflects certain characteristics of the eye movements. The pattern of head movement is not compensatory but is pathological in its own right. (v)=vertical eye movement.

quencies of the head and neck, it is tempting to speculate that the two are connected in some way, and our approach to understanding the link is to begin by considering the basic physical requirements for movement. Most limb movements (with the possible exception of ocular movements) require a distinct sequence of muscle contractions to initiate, continue, and finally stop the movement. An optimally efficient movement is made with respect to the natural resonant frequency of the limb. Movement of a limb at its resonant frequency, however, risks the limb becoming unstable and oscillating, which means that at the termination of the movement the muscles have to stop the limb and counter any tendency to

resonate. Should the stopping action be weak or timed incorrectly the limb will overshoot, producing dysmetria. Should the activity responsible for countering resonance be insufficient or wrongly timed, the limb will oscillate.

In addition, there are important passive mechanical influences on the head created indirectly by body movement which have frequencies close to 2 Hz. Thus Cavagna *et al.* (1976) have shown that during walking and running the frequency of oscillation of the centre of gravity of the entire body tends towards 2 Hz as the pace increases. Without effective measures to counter resonance, activities such as running would set off uncontrolled oscillations of the head at the resonant frequencies.

During body movements it is obvious that the head is not held rigidly on the trunk to prevent it from swaying out of control. Quite the opposite. The head is suspended "fluidly," stabilised in space, and moving with respect to the body to counter mechanical disturbances. Therefore, the mechanism responsible for countering unwanted movement and resonance must have access to precise timing signals and because of the high inertia of the head, be quite powerful. It is quite conceivable that when such a mechanism breaks down in some way it will produce involuntary oscillations of the head. Furthermore, the oscillation would tend to be at the natural resonant frequencies which the mechanism was designed to overcome. There is then the possibility that tremor in some central nervous diseases is the result of the breakdown of mechanisms normally responsible for countering unwanted movements.

Head tremor may be phase locked to tremor of

other parts of the body such as the palate and the larynx (case 15). There are two possible interpretations of this finding. The first is that since these body parts do not have the same natural frequencies of movement, the tremor is related to some function of the nervous system other than control of unwanted movement. The second view is that since body movement is organised harmoniously with use of a few basic rhythms (try walking while swinging the arms twice as quickly as the legs), most limbs do have similar natural frequencies of movement and some body parts may simply use certain dominant rhythms to time their functions.

A common explanation of the mechanism of some cerebellar tremors (Marshall, 1968) is that they arise from the hypotonia which is characteristic of cerebellar disease. During posture, through loss of tone the limb is insufficiently supported and tends to fall away. A corrective adjustment is made which leads into a further cycle of oscillation and so forth. This view of the origin of the tremor is incomplete, for spectral analysis of the movement wave form (Gresty and Halmagyi, in preparation) reveals two principal frequencies of movement, one due to hypotonic postural swaying at about 3 Hz, and a second at 4–5 Hz which is apparently an active component. The two combine to give the “ragged” appearance of many cerebellar tremors. The origin of cerebellar tremor lies in hypotonia compounded with deficits in the mechanism responsible for the scaling and timing of neuromuscular signals which brake movement and convert movement to posture. By implication of the clinical context the cerebellum is concerned with these functions.

Conclusions

We have shown that in a variety of diseases the frequency characteristics of head dyskinesias are closely related to the mechanical properties of the head/neck system. A classification of head dyskinesias is elaborated, emphasising this relationship. In particular, distinctions are made between passive dyskinesias caused by neck hypotonia, adaptive dyskinesias sometimes the result of abnormal eye movements, and dyskinesias such as tremor and chorea.

Movement of the head/neck system is of importance to the organisation of whole body movement. We know, for example, that if the neck is deafferented orientation in space is lost although the special senses are intact (Cohen, 1961). Furthermore, there is the consideration that the

head is the platform which carries the special senses and orients them directionally, and to this end much of body movement is subservient. These considerations suggest that a clue to understanding body movement is via examination of their relationship to the static and dynamic requirements of the head.

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