

Benign recurrent vertigo

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SUMMARY Patients with recurrent vertigo in the absence of cochlear signs remain a diagnostic problem. The absence of a standard system of nomenclature further hampers the understanding of these disorders. The term benign recurrent vertigo is suggested as a useful term to characterise many of these patients. The disorder shares some of the features of migraine, and seems likely to have a similar vasospastic aetiology.

Diagnoses in patients with dizziness are often imprecise. A careful history and physical examination and the use of ancillary tests are helpful. Patients with psychogenic disease, head trauma, transient ischaemic attacks, and Ménière's syndrome, as well as patients with the entity of "benign positional vertigo" can at least be categorised even if the exact aetiology is obscure. However, there remain many patients for whom there appears to be no satisfactory diagnosis, even after restricting consideration to patients with recurrent attacks of true vertigo and objective vestibular disturbance.

Drachman and Hart (1972) in a comprehensive approach to patients with dizziness classified six patients out of a total of 125 as "acute and recurrent peripheral vestibulopathy." They point out the problem with the use of alternative terms such as "vestibular neuritis, vestibular neuronitis, acute labyrinthitis, epidemic vertigo, and viral labyrinthitis." All of these terms suggest knowledge of aetiology and location of pathology that is not justified clinically. Drachman and Hart do not, however, discuss "recurrent vestibulopathy" such as to define the term nor do they suggest any aetiology.

I have seen patients with recurrent vestibular disturbance quite frequently in my practice and report seven of these patients in the hope of suggesting a descriptive diagnosis that will lead to further study and characterisation of these disorders. Clinical data suggestive of a vasospastic aetiology will be discussed.

Patients

Patients presenting with acute attacks of vertigo between June 1975 and April 1978 were selected for review. One patient, case 6, was contributed by Dr Robert Lisak, Department of Neurology, Hospital University of Pennsylvania. Patients who had prominent aural symptoms of tinnitus or hearing loss were excluded as were those with a history of related head trauma or suspected cerebrovascular disease. All patients had normal otological and neurological examinations except for abnormalities confined to the vestibular system in the form of nystagmus, usually detected on electronystagmographic (ENG) tracing.

Electronystagmography with quantitative caloric tests, audiograms, and other audiometric studies when performed (unless otherwise indicated) were done at the Speech and Hearing Center at Delaware County Memorial Hospital. All ENG studies included testing in the primary and lateral positions with and without the head turned.

The Table contains an outline of the complaints and findings of the seven patients. Their ages ranged from 25 to 55 years at the time of evaluation, and the age of onset of the disorder from 7 to 55 years. There were five women and two men. Symptoms were described in almost identical fashion by these patients. The onset of vertigo was always acute without warning. The initial symptom was severe, usually requiring the patient to lie down, but not accompanied by vomiting. After a variable period of time, one minute to 24 hours (usually half to four hours), the acute constant vertigo would subside but was followed by a period of positional vertigo. Positional vertigo would occur only with head motion or placing the

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Table Summary of complaints and findings in seven patients

Patient	Age (yr)	Sex	Age at onset (yr)	Frequency	Duration	Tinnitus loss	Clinical examination		Audiograms	Tone decay	Impedance audiometry	ENG	Calories	Family history	Headaches	Precipitating factors
							Spontaneous nystagmus	Positional nystagmus								
1	30	F	7	1/week	1 1/2-24 hr	Mild AU	—	—	Normal	—	SN Right	Normal	—	*Mother *Brother *Child	Frontal 1/2 months to 2 hours unrelated to vertigo	Awakens from sleep with attacks
2	23	M	22	1/month	4 hr	Mild AU	—	—	Normal	—	PN DC	Normal	—	*Father	Unilateral severe paroxysmal unrelated to vertigo	—
3	56	F	55	1/2 weeks	20 min	Mild AD	—	—	Normal	—	SN Right PN DC	Normal	—	—	Severe unilateral scotoma when young	(Father suffered attack of total blindness lasting 24 hr at age 49 yr)
4	33	M	23	3/year	24 hr	—	—	—	Normal	—	PN Left	Normal	—	—	Severe age 11-12 yr	Lack of sleep Alcohol Attacks always upon awakening
5	25	F	50	1/day for 3 wk followed by a 3 month remission and then a similar 3 week episode	1 min	Mild AD	—	—	Normal	—	SN Left PN DF Right	Normal	—	—	ENG and audiograms performed at Temple University Hospital	Loss of vision for a split second with attacks of vertigo
6	39	F	35	1/2 years	2-3 days	Mild AU	—	—	Normal	—	SN Left	Normal	—	*Migraine only mother and two siblings	Pulsatile with nausea at times with vertigo	—
7	30	F	8	4/year	—48 hr	—	—	—	Normal	—	SN Right	Normal	—	*Son age 10 *Migraine, mother, sister, brother	ENG and audiograms performed by Dr S. T. Westermun, Shrewsbury, New Jersey	Emotional stress

AU = both ears.
 AD = right ear.
 SN = spontaneous nystagmus.
 PN = positional nystagmus.
 DF = direction fixed nystagmus.
 DC = direction changing nystagmus.
 * Family history refers to attacks of recurrent vertigo unless otherwise indicated.

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head in a given position, and was brief. The latter phase slowly improved over periods of hours or days: however, permanent positional vertigo occurred in patient 2, and positional vertigo lasting one month in patient 7. The frequency of attacks varied widely from one per day to two per year. Any patient with either tinnitus or pressure in the ear stated that these symptoms were unrelated to the attacks. The description was invariably quite vague, non-specific, and similar to the response given by many patients when asked if they ever had ringing or pressure in the ears. Audiograms were normal.

Electronystagmographic testing, in contrast to the audiograms, showed some type of pathological nystagmus, either spontaneous or positional, in all seven patients. Caloric responses were normal in these patients. A family history of an identical illness was present in three patients and was quite striking in patient 2, whose mother, brother, and child were all involved, thus suggesting an autosomal dominant inheritance pattern. Precipitating factors included lack of sleep, emotional stress, and alcohol. Four patients had a history of recurrent paroxysmal headache unrelated in time to vertiginous attacks. One patient developed headaches during periods of vertigo. One patient had the association of scotomata with headache, and patient 5 complained of momentary loss of vision with the vertiginous attacks.

An additional five patients were seen with identical symptoms but who either failed to have studies completed or showed bilateral symmetrical hearing loss.

Discussion

Attempting to classify patients with vertigo presents the clinician with a rather unsatisfying and frustrating task. Although the entity of Ménière's syndrome in its classical form is well described as recurrent attacks of vertigo with onset between ages 30 to 40 years, accompanied by hearing symptoms and cochlear signs, the classification of recurrent vertigo without cochlear signs is poorly defined. None of the patients presented here had asymmetric hearing loss. Although some authorities state that the hearing loss can follow attacks of dizziness by many years in Ménière's syndrome, this point is controversial. This consideration certainly would not apply to patients suffering from the disorder over 10 years and those with onset before the age of 30 years.

Textbooks of neurology and textbooks devoted to Ménière's syndrome would be expected to

classify or discuss this group of patients in detail. However, one finds instead only the entities of positional vertigo and vestibular neuronitis that might apply to these seven patients (Spector, 1967; Alpers and Mancall, 1971; Merritt, 1973; Baker and Baker, 1976). Neither of these disorders represents a satisfactory diagnosis for this group of patients. Positional vertigo or nystagmus refers to a single symptom of transient vertigo caused by postural change (Dix and Hallpike, 1952), and does not properly apply to spontaneous recurrent, prolonged attacks of vertigo such as these patients suffered. Even patient 5, whose attacks were brief, had them occur without positional change. Transient ischaemic attacks would be a possible consideration in an older age group but unlikely in this group of patients with onset of symptoms usually before the age of 40 years and no other signs of vascular disease. We are thus left with the diagnosis of vestibular neuronitis, a very unsatisfactory term. Although the description of vestibular neuronitis is appropriate in terms of age of onset and the absence of cochlear symptoms, the periodic recurrence of vertigo, the frequently normal caloric responses and brief duration of attacks are all atypical for this disorder (Dix and Hallpike, 1952; Harrison, 1962; Lumio and Aho, 1965).

Syndromes similar to those suffered by the patients presented here occur in childhood and have been labelled benign paroxysmal vertigo of childhood (Koenigsberger *et al.*, 1970) and paroxysmal dysequilibrium of childhood (Watson and Steele, 1974). The major differences between these previous reports and this group of patients are the age of onset and the shorter duration of attacks in the childhood variety. Otherwise the similarity is striking, including the possible association with migraine.

Other syndromes that might superficially resemble this group of patients include acute intermittent familial cerebellar ataxia (Hill and Sherman, 1968), paroxysmal ataxia associated with pyruvate decarboxylase deficiency (Blass *et al.*, 1971) and other metabolic diseases, and vestibulo-cerebellar ataxia (Farmer and Mustian, 1963). The distinction between ataxia and vertigo is not difficult in the supine patient, and associated neurological signs will readily distinguish these other patients. The disorder reported as familial paroxysmal nystagmus, vertigo, and ataxia (White, 1969), on the other hand, may represent a similar syndrome. The positive family history and association with alcohol and sleep lack as precipitating factors are compared with the patients presented here.

I suggest the term benign recurrent vertigo to describe the illness suffered by the patients presented in the Table. This label does not define a pathological entity, but it provides the patient with a name for his condition, which immediately resolves much of the anxiety suffered by patients with recurrent vertigo without cochlear symptoms who have not been offered a diagnosis, often after seeing numerous consultants. The term benign recurrent vertigo also avoids the ambiguity and incongruity of a term such as vestibular neuronitis. We would be unlikely to call recurrent attacks of transient visual scotoma optic neuronitis, and there is no parallel of any other nerve in the body subject to recurrent dysfunction of this sort, with the possible exception of Melkerson's syndrome with recurrent facial paralysis, a very rare syndrome.

The term "benign" is justified even if patients are left with vestibular dysfunction, just as a benign tumour can cause symptoms. It does, however, reinforce that the disease is limited to one set of symptoms and is not the harbinger of more serious neurological or systemic disease.

There is no justification for trying to subdivide this group on the basis of the type of nystagmus observed. Recent studies have shown that the type of positional or spontaneous nystagmus, with the exception of pure vertical nystagmus, does not help differentiate the various causes of vertigo (Stahle and Terins, 1965; Dayal *et al.*, 1974; Harrison and Ozsahinoglu, 1975). Indeed, it has even been shown that a patient with one type of nystagmus may show conversion to another type over time (Dayal *et al.*, 1974).

I suggest at the present that this syndrome of benign recurrent vertigo can be recognised on the basis of its clinical description. Attacks of spontaneous vertigo not precipitated by movement and not accompanied by other neurological symptoms recur in young or middle-aged adults. Attacks usually last hours to days and are followed by variable periods of "positional vertigo." No aural symptoms accompany the attacks, and audiograms are normal. Objective vestibular dysfunction can be demonstrated by electronystagmography and neurological examinations are normal, with the exception of positional nystagmus.

The disorder shows some features in common with migraine which include precipitation by alcohol, lack of sleep, emotional stress, female preponderance, and positive family histories. A similar vasospastic aetiology is thus suspected. Therapy with antimigrainous medication such as ergotamine or beta-adrenergic blockers may be worthy of clinical trials. Previous studies have also commented on the association of migraine and

vertigo, but have not emphasised the occurrence of vertigo as an independent symptom (Eadie, 1968; Loh and Chawla, 1972; Dursteler, 1975).

I do not suggest that this syndrome can never be mimicked by other diseases. Paroxysmal symptoms occur in multiple sclerosis and in some patients with intracranial tumours. Long-term follow-up of larger groups of patients with benign recurrent vertigo will be needed to assess how often non-benign illness may present in a similar way. Recognition of the clinical syndrome should speed this assessment.

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