

BENIGN PAROXYSMAL VERTIGO OF CHILDHOOD  
(A VARIETY OF VESTIBULAR NEURONITIS)

BY

L. S. BASSER<sup>1</sup>

INTRODUCTION

IN the past fifteen years and particularly in the last decade there has been a considerable and remarkable clarification of the varieties of vertigo occurring in adult life. This has been due in part to the establishment of consistently reliable techniques of estimating vestibular function and also to the increased attention given to these conditions.

The position in children is very different. Vertigo in childhood has received very little attention and in fact often goes unrecognized, as will be discussed. The general view is exemplified by Ford (1960), who refers to the purulent and rare serous forms of labyrinthitis complicating gross infections of the middle ear and mastoid process, to vertigo as a symptom of a number of conditions particularly brain-stem lesions and posterior fossa neoplasms, and to Ménière's disease which he states is "so rare in childhood that it does not require more than a very brief discussion." Confirming this view, the case quoted in illustration by him, as well as being atypical of Ménière's disease, also had a posterior fossa exploration during the course of her investigations. No other form of vertigo is mentioned. Harrison (1962*a*) has discussed other varieties of vertigo occurring in children.

The purpose of this paper is to draw attention to the problem and to describe a particular variety of paroxysmal vertigo occurring in childhood, not previously reported, not uncommon, with characteristic and readily recognizable features, and differing from other forms of paroxysmal vertigo found in adult life. It has both practical and theoretical importance.

<sup>1</sup>193 Macquarie Street, Sydney, N.S.W., Australia.

## FEATURES

Benign paroxysmal vertigo of childhood describes the essential features of the disorder. The cardinal symptom is vertigo occurring in isolation. The absence of cochlear symptoms is conspicuous and characteristic, there being no tinnitus or deafness at any time during the course of the condition. Hearing remains intact with normal audiograms. The age of onset is usually within the first three or four years of life but may be later, for example seven or eight years. Both girls and boys are affected. The attacks occur in a setting of good health, are of sudden onset and often of such severity as to be totally disabling but may be less severe. They are brief, lasting seconds only, usually less than a minute, and rarely more than a few minutes. Pallor is frequently associated and there may be sweating and occasionally vomiting. Nystagmus is seen during the attacks in some cases and may be prominent. There are no known precipitating factors. The attacks may occur standing, sitting or lying. No relation to head posture or movement has been observed. Consciousness is not impaired during the attacks. There is no pain or headache associated with the attacks. As a rule, in a severe attack the child remains perfectly still, unable to move, stand or even sit without support. Frequently the child is limp for the duration of the attack, incapable of using the limbs or controlling the position of the head. When the attack is less severe, the child typically clutches the mother or her clothing until the attack ceases. Either immediately following the attack or after a few minutes during which there may be some slight persisting instability, the child has completely recovered and resumes play or other normal activity. The attacks are recurrent. The intervals between attacks vary, four to six weeks being common, or they may be as short as one or two weeks or even days, and unusually, as long as six months or a year. The child is well between the attacks. The attacks cease spontaneously after a period which may be months but is usually years. There is no residual disability. No abnormalities are found at any time on neurological or general examination. The radiological appearances of the skull, including special views of the petrous temporal bones, are normal. The electroencephalogram is characteristically normal.

The one outstanding abnormal finding is demonstrated by the caloric tests which show disordered vestibular function in the form of moderate, severe, or complete canal paresis which may be bilateral or unilateral and which not infrequently persists after the attacks finally cease. Positional vertigo and nystagmus are not found.

The attacks are clearly and sometimes graphically described by the children and this is stressed. The striking uniformity of the features is emphasized and permits ready recognition of the disorder. The distinction from adult forms of paroxysmal vertigo will be dealt with later in the paper.

## CASE MATERIAL AND ANALYSIS

Seventeen cases were studied for the purpose of this report. The disorder is not uncommon, as will be discussed. The cases were seen over a period of seven years. All cases were examined personally. The investigations were done at the Northcott Neurological Diagnostic Centre. The audiograms were done by the Commonwealth Acoustic Laboratory.

There were 10 girls and 7 boys. The age of onset of the attacks was in the first four years of life except in 2 cases aged 7 and 8 years respectively. The frequency of the attacks was mostly about every four to six weeks but could be as often as weekly or even daily or as seldom as six monthly or yearly. The frequency varied from time to time for the individual. The duration of the attacks was usually seconds and less than a minute and rarely more than a few minutes. The attacks ceased spontaneously. The duration of the liability to attacks was usually years and up to six years but in 4 cases was several months only. The onset of the attacks was always sudden. Vertigo was the cardinal symptom and occurred in isolation. Nystagmus was observed in attacks in 3 cases. No definite precipitating factors were established. In 1 case the attacks were noted often to precede an attack of acute tonsillitis. There was no evidence of ear infection in any case at the time of onset or during the course of the disorder, nor was there evidence of upper respiratory tract infection except in the 1 case mentioned where attacks were often noted to precede acute tonsillitis. There was a history of earache at some time in the past remote from the onset of the attacks in 3 cases, and in 2 cases there was a history of otitis media in the first year of life; in none was there evidence of residual ill-effect.

The caloric tests typically demonstrated moderate, severe or complete canal paresis. The technique of Fitzgerald and Hallpike (1942) is readily applicable to children, giving consistent results and even in the young involved no particular difficulty once their confidence was gained. The children did, of course, experience very little discomfort as vertigo was often either absent or very mild during the tests. It should be mentioned that all these children were notably co-operative, alert and attractive. With complete canal paresis there were absent responses to stimulation at both 30°C. and 44°C. There was bilateral canal paresis in 8 cases, being more marked on one side than the other, and there was unilateral canal paresis in 6 cases. The responses were normal in 1 case when the tests were done for the first time two and a half years after the cessation of the attacks. It was not possible to arrange for the tests in 2 cases. Follow-up revealed canal paresis to be present in 6 cases after the attacks had ceased after intervals of from two to six years. There was improvement in responses to caloric testing in 1 case although the

attacks persisted. As mentioned, there were normal responses in 1 case two and a half years after the cessation of the attacks.

There were no abnormal neurological signs.

The electroencephalogram was typically normal. In 2 cases an early record showed a mild dysrhythmia and in 1 case paroxysmal changes; in 2 of these cases there was a family history of epilepsy; later records were normal in all 3 cases. In 1 case there was a normal record at the age of onset of the attacks but three years later right temporal spikes were found and these persisted although the attacks ceased. The electroencephalograms were normal in all other cases. There was nothing to suggest a personal history of epilepsy in any case except one in which there had been two infantile convulsions a year before the onset of the attacks of vertigo.

Hearing was clinically normal in all cases at all times during the course of the disorder and in 11 cases audiograms done by the Commonwealth Acoustic Laboratory were normal.

The period of follow-up from the age of onset of the attacks was from six to nine years in 8 cases, three to four years in 3 cases, one year in 3 cases and 3 cases were seen recently for the first time.

The children's comments on their attacks were clear, even in the young, and although simple were often eloquent. There was frequently a strong similarity in the remarks from case to case. The accounts of the older children could be graphic. Some of the children's words may be quoted. "The house is going round." "Everything around me is spinning." "Things go around and my legs seem to go in all directions." "Everything is going around." "I am falling. The walls are falling." A child described by the mother as "walking all over the place" said "I can't walk, Mummy, I can't walk." Another complained "The room has gone away." Another child suddenly screamed and said "The house is falling over," while another complained "The ceiling is falling in." A boy aged 13 years said "In an instant I am very giddy, and everything seems to be turning in a clockwise direction." At the onset of an attack while he was watching television he suddenly threw his hands in the air and said "I'm giddy," and shut his eyes.

#### DIFFERENTIAL DIAGNOSIS

Vertiginous crises in childhood frequently go unrecognized and often are regarded as functional disturbances, manifestations of a behaviour disorder. The possibility of minor epileptic seizures is usually considered and this diagnosis may become accepted. Quite often they are dismissed as rather mysterious "turns." When recognized as vertiginous, suspicion is directed to the existence of a brain-stem lesion or posterior fossa neoplasm particularly one in the neighbourhood of the fourth ventricle,

and extensive and unnecessary investigations may be done. Although there has been considerable clarification of the varieties of vertigo occurring in adult life, it has been found that these seldom, in fact rarely, occur in the young, especially in childhood, and this in itself has been an obstacle to the recognition of vertiginous attacks in children.

*Functional disorder.*—There has been nothing in the personalities or behaviour of these children to suggest a functional disorder. The children have been notably co-operative, alert and attractive. Characteristically, they resume play or other normal activity immediately after the attacks which are very brief. The features of the attacks are strikingly uniform from case to case and typical of a paroxysmal vestibular disorder.

*Epilepsy.*—It is well known that vertigo may occur as an epileptic phenomenon either as an aura or constituting the seizure (Gowers, 1901). Cortical stimulation may produce similar experiences (Penfield, 1954). The diagnosis rests on establishing a lapse of consciousness during the attack or amnesia for the attack (Alpers, 1960). If the attacks occur without there being other epileptic phenomena, it is impossible to establish their epileptic nature without one or other of these criteria. The absence of post-ictal phenomena is a feature of these attacks. The electroencephalogram may support the diagnosis. The subject has been discussed by Spillane (1950). Vestibulogenic epilepsy is a form of “reflex epilepsy” initiated by stimuli arising from a damaged labyrinth. These attacks are considered to arise from a brain-stem reticular system as a result of labyrinthine discharge (Behrman and Wyke, 1958). Loss of consciousness may rarely occur in association with aural vertigo and is then syncopal.

In benign paroxysmal vertigo of childhood, the attacks are not infrequently mistaken as epileptic. However, consciousness is not impaired and the attacks are vividly recalled. There are no other epileptic phenomena. The electroencephalogram is characteristically normal. The condition is self-limited. An adequate history establishes the purely vertiginous nature of the attacks and there is objective evidence of disordered vestibular function as revealed by the caloric tests. The mechanism of symptom production which will be suggested offers an explanation for the superficial resemblance of the attacks to minor epileptic seizures.

*Brain-stem lesions and posterior fossa neoplasms.*—Vertigo may be the leading symptom of such conditions and may for a time occur alone. The vertigo may be paroxysmal. Sooner or later, and generally from the onset, other features indicative of brain-stem or cerebellar disorder or raised intracranial pressure become apparent. The relative frequency of subtentorial neoplasms in children as compared with adults need not be stressed.

*Ménière's disease.*—There have been reports of the condition occurring in children. Crowe (1938) reported a child aged 6 years as having Ménière's disease; in a series of 138 cases 3 were under the age of 20 years. Ford (1944) reported a case aged 4 years and Sorensen (1958) a girl aged 7 years with the disorder. Harrison (1962*a*) in a series of 429 cases included 5 under the age of 10 years. These 5 cases were considered by the author to have "pseudo Ménière's disease" differing significantly from true Ménière's disease in the adult as there was a tendency for complete recovery to occur in the child with no residual loss of either hearing or vestibular response. The present writer has not had experience of cases of this kind although paroxysmal vertigo in children with hearing impairment has been met on a few occasions. In these cases the hearing defect has become permanent. However, the overall features have been atypical of Ménière's disease as it occurs in adult life. When strictly defined, it is probable that Ménière's disease is a disorder of adult life (Brain, 1938) and that a degenerative factor or some process requiring to act over a period of years is necessary to cause symptoms. Cawthorne's series (1954) bears this out; there were no cases under the age of 10. In children with paroxysmal vertigo associated with cochlear symptoms it is highly probable that the ætiology (Seymour, 1960) and pathology differ from those of Ménière's disease occurring in adult life.

*Vestibular neuronitis.*—Hallpike (1959) first described the disorder in a short communication to the Fourth International Congress of Otolaryngology in London. The condition began to be recognized as a distinct clinical entity about 1946. A more detailed account was given by Dix and Hallpike (1952*a* and *b*). Most of their cases were between the ages of 30 and 50 and only 5 of their series of 100 cases were under the age of 20 years. They found that "the vertigo may consist of sudden and transient seizures accompanied by sensations of blackout, but on the other hand there may be no severe paroxysms and disequilibrium may take the form of feeling 'top-heavy' or 'off balance' particularly when walking or standing. This disequilibrium is aggravated by head movements of all kinds." Cawthorne (1959) described intense vertigo at the onset made worse by head movement and remarked that the vertigo diminishes daily. The non-paroxysmal features described were not encountered in benign paroxysmal vertigo of childhood, the attacks in this condition being purely paroxysmal from the onset, nor was there aggravation by head movement. Dix and Hallpike emphasized that "in a fairly high proportion of the subjects the onset of symptoms is associated with some kind of febrile illness or evidence of infection of the ears, nose and throat." This was not so in the cases of benign paroxysmal vertigo of childhood, the onset being in a setting of good health and evidence of infection was significantly lacking. They stressed the absence of cochlear symptoms and

signs and the consistently reduced caloric responses, features also characteristic of benign paroxysmal vertigo of childhood. They argued that "the lesion is central to the labyrinth in accordance with the well-established principle of otoneurology, viz. that destructive labyrinthine lesions, whatever their pathology, tend on the whole to involve the cochlear apparatus." They considered the lesion likely to be central to Scarpa's ganglion. Harrison (1962*b*) also found that the disorder affected adults and only 3 of the 67 cases reported were under the age of 20 years. The findings accorded closely with those of Dix and Hallpike. In 21 cases the vertigo was limited to a single attack; it was also found that "the cessation of the vertiginous attacks was usually followed by a period of unsteadiness which lasted on an average for five and a half months." These features were also notably absent in the cases of benign paroxysmal vertigo of childhood. Harrison (1962*a*) reported 2 cases as examples of vestibular neuronitis in children. Case IV, a boy aged 10 years, had attacks of pain in the central occipital region for three years. The attacks lasted thirty minutes and were associated with clockwise rotational vertigo. There was no hearing loss. The caloric responses were within normal limits. It is doubtful whether this case is typical of vestibular neuronitis and the features rather suggest migraine which may not infrequently be associated with vertigo in children. The other, Case V, had features suggesting benign paroxysmal vertigo of childhood but no detailed comment on the case was made by the author.

Benign paroxysmal vertigo of childhood is a purely vestibular disorder with distinct clinical features. It is characterized by the absence of cochlear symptoms and signs and by reduced or absent caloric responses, and in these respects resembles vestibular neuronitis as described in adults. It may in fact be regarded as falling within this category of disorder. The term vestibular neuronitis is a comprehensive one and has a degree of deliberate ambiguity, having regard to the imperfect state of knowledge which exists concerning the ætiology, pathology and the precise site of the lesion within the vestibular neurone. It is possible and indeed probable that a number of different conditions are included by the term. The concept grew originally from the recognition of "a distinct clinical entity" (Hallpike, 1952*a* and *b*). The condition so defined differs significantly from the strikingly uniform pattern of benign paroxysmal vertigo of childhood. The differences indicated in the discussion of the differential diagnosis from vestibular neuronitis set benign paroxysmal vertigo of childhood apart as a particular variety of paroxysmal vestibular disorder. These differences may reflect differences in the ætiology and pathology of the vestibular neuronal disorder and probably do, but this is necessarily a matter for speculation. The distinction rests on the childhood incidence and also the uniformity and purely paroxysmal nature of the attacks, their brevity, their recurrence, the absence of any prolonged episodes of

disequilibrium and the absence of febrile illness at the onset or evidence of upper respiratory tract infection. It may be added that during the period of seven years over which the cases of benign paroxysmal vertigo of childhood have been seen, vestibular neuronitis in adults has also been seen with the characteristic clinical features described by Dix and Hallpike, but none has exhibited the same clinical features as the children and conversely, no children in the age group under consideration have been seen with the clinical features of vestibular neuronitis described in adults.

*Epidemic vertigo.*—Pedersen (1959) described a series of epidemics in Denmark. These occurred in epidemiological association with encephalitis. Asthenic symptoms were usually associated. There were two main types, one dominated by gastro-intestinal symptoms and the other by upper respiratory tract infection. It was considered that the localization of the disease was in the brain-stem. Tinnitus was rare and hearing was normal in all but one case. The caloric responses were not characteristically reduced. There have been a number of other somewhat similar series reported.

Benign paroxysmal vertigo of childhood is a sporadic disorder. There is no particular seasonal incidence. The features of epidemic vertigo described above do not occur in this condition. Furthermore, the caloric responses are characteristically reduced.

*Positional vertigo.*—Positional vertigo, either of the peripheral or central type, is not found in cases with benign paroxysmal vertigo of childhood.

#### DISCUSSION

It is apparent from experience and discussion that benign paroxysmal vertigo of childhood is not uncommon and that those in neurological or pædiatric practice may see several cases each year. There is little doubt that it is the most common form of paroxysmal vertigo occurring in children. The disorder is sporadic, there have been no epidemic features and as far as can be judged, the incidence remains much the same from year to year.

A purely vestibular disorder, it is submitted that the lesion is central to the labyrinth which is in accord with the absence of cochlear symptoms and signs. The alteration in the caloric responses is in the form of a simple reduction or paresis and this suggests that the lesion is peripheral to the brain-stem. There is no evidence to indicate involvement of the brain-stem or central vestibular connexions. The postulated site of the lesion is therefore in the vestibular pathway between the labyrinth and the brain-stem.

The pathology is unknown but the reduced caloric responses indicate a destructive lesion. The persistence of the reduced caloric responses after the cessation of the attacks indicates that the lesion is permanent

in these cases. Dix and Hallpike and also Harrison have stressed the occurrence of a febrile illness at the onset of vestibular neuronitis in adults or the association of upper respiratory tract infection, usually antral. No specific conclusions are drawn by them in regard to the ætiology of vestibular neuronitis, but it is implied that infection is an important factor. Infection is not an obvious ætiological factor in the cases of benign paroxysmal vertigo of childhood. However, having regard to the prevalence of upper respiratory tract infections in children generally, this factor cannot be dismissed. The possibility of a specific neurotropic virus might be considered but there is no supporting evidence. It is speculative to suggest that the vestibular pathway may have a special vulnerability in childhood rendering it liable to this particular disorder. The ætiology is obscure.

The mechanism of symptom production is of considerable interest. The features of the attacks suggest that sudden spontaneous recurrent discharges within the vestibular neurone are responsible. It is implied that the underlying lesion is irritative as well as destructive, and that the duration of the liability to attacks corresponds with the duration of the irritative phase as there is evidence, already mentioned, that the destructive lesion often persists after the attacks cease and is permanent. The mechanism of symptom production suggested is analogous to that responsible for lightning pains in tabes dorsalis and sensory radicular neuropathy, i.e. brief spontaneous recurrent discharges arising in a peripheral sensory neurone as a result of an irritative lesion.

The importance of the recognition of benign paroxysmal vertigo of childhood is stressed. The attacks are alarming. The importance rests chiefly on the completely benign prognosis. Reassurance may be confidently offered. Extensive and unnecessary investigations are avoided. The condition is readily recognized and the diagnostic features have been described. An adequate history is imperative.

#### SUMMARY

Attention is drawn to the problem of vertigo occurring in childhood. The features of a particular variety, benign paroxysmal vertigo of childhood, are described.

An analysis of the case material is presented.

The differential diagnosis is discussed, particularly from functional disorder, epilepsy, and those varieties of paroxysmal vertigo occurring in adult life.

The site and pathology of the lesion are discussed and a mechanism of symptom production is postulated.

The importance of the recognition of the condition is emphasized.

## APPENDIX

## SYNOPSIS OF ILLUSTRATIVE CASE HISTORIES

*Case I (G. F.).*—An English boy, he had his first attack while living in England at the age of 7 years. He was perfectly well and was walking to the cinema with a friend when he suddenly dropped to the ground, extremely giddy. His friend walked on until he noticed that the patient was no longer with him and turned back to find him lying on the ground. He was quite conscious. He recovered in less than a minute and was able to proceed to the cinema. He had five attacks in the next five years at intervals of about a year. He then had several attacks within a period of twelve months and the attacks then ceased spontaneously at the age of 13 years, the duration of liability to the attacks having been six years. All the attacks were similar. They were of very sudden onset and without warning. Describing the attacks he said "In an instant I am very giddy and everything seems to be turning in a clockwise direction." The attacks were frequently associated with sweating, and he felt cold and might have epigastric discomfort. The duration of the attacks was usually seconds and not longer than a minute. He was quite definite that there was no impairment of consciousness, and that he was perfectly aware of everything that was going on during the attacks. There was no headache during or after the attacks. He continued to have the attacks after he came to Australia and he had one while watching television. He suddenly threw his hands in the air and said "I am giddy" and shut his eyes. There were no precipitating factors. There was no history of otitis media. There were no abnormal neurological signs. The electroencephalogram was normal, and radiological studies of the skull including special views of the petrous temporal bones were normal. There was no personal or family history of epilepsy. The tympanic membranes were normal in appearance. Hearing was clinically normal and this was confirmed by an audiogram carried out by the Commonwealth Acoustic Laboratory. The caloric responses showed severe canal paresis on the right and complete canal paresis on the left which have persisted.

*Case II (G. J.).*—This girl's attacks began at the age of 3 years. They occurred at intervals of about a fortnight. At first they were minutes in duration but later lasted for seconds only. They continued for a year and ceased spontaneously at the age of 4 years. All the attacks were similar. They were of sudden onset. A typical attack took place while she was sitting in a chair, when suddenly she cried out "I'm falling; the walls are falling." There was no impairment of consciousness. There were no precipitating factors. There was no history of otitis media. The electroencephalogram was normal. Radiological studies of the skull were normal. There was no personal or family history of epilepsy. The tympanic membranes were normal. Hearing was clinically normal and this was confirmed by a normal audiogram. The caloric tests showed a severe right canal paresis and a moderate left canal paresis. The tests were done on three separate occasions between 1958 and 1961.

*Case III (R. D.).*—This boy had his first attack at the age of 2½ years. His attacks recurred at intervals of about a month. They were mostly of seconds' duration but could be as long as one or two minutes. They ceased after about six months at the age of 3 years. He was always perfectly well before the attacks. The onset was very sudden. In a typical attack he complained that "Everything around me is spinning." He would fall in the attacks and did hurt himself on occasions. There was no impairment of consciousness. There were no precipitating factors. There was no history of otitis media. There were no abnormal neurological signs. The electroencephalogram was normal and radiological studies of the skull were normal. There was no personal or family history of epilepsy. The tympanic membranes were normal. Hearing was clinically normal. The caloric tests were not done during the period of

liability to the attacks but subsequently showed complete bilateral canal paresis which was permanent.

*Case IV* (J. P.).—This girl had her first attack at the age of 3 years. The attacks occurred at intervals of about a month. Their duration was less than a minute. They ceased spontaneously after a few months. The attacks occurred in a setting of good health. They were sudden in onset. In a typical attack she suddenly complained "The house is going around." She was pale and frightened and could not stand unsupported. Nystagmus was reliably observed in a number of attacks. There was no impairment of consciousness. There were no precipitating factors. There were no abnormal neurological signs. The electroencephalogram was normal. Radiological studies of the skull were normal. There was no personal history of epilepsy. The tympanic membranes were normal. Hearing was clinically normal. An audiogram was normal. The caloric tests showed a severe canal paresis on the right.

*Case V* (S. M.).—This boy had his first attack at the age of  $2\frac{1}{2}$  years. The attacks recurred at intervals of four to six weeks. They lasted for minutes. They continued for a period of about a year and ceased spontaneously at the age of  $3\frac{1}{2}$  years. The attacks were of sudden onset and occurred in a setting of good health. He was observed by his mother to be "walking all over the place" and during the attacks he complained "I can't walk, Mummy, I can't walk." He became pale during the attacks and in some attacks he vomited. There was no impairment of consciousness. There were no precipitating factors. There were no abnormal neurological signs. The electroencephalogram was within normal limits and radiological studies of the skull including special views of the petrous temporal bones were normal. There was no personal or family history of epilepsy. The tympanic membranes were normal. Hearing was clinically normal and this was confirmed by an audiogram. The caloric tests showed bilateral moderate canal paresis.

*Case VI* (L. M.).—This girl had her first attack at the age of 20 months. The attacks occurred at intervals of about two or three months. Their duration was minutes. They ceased spontaneously at the age of 8 years, the duration of the liability to the attacks having been six years. All the attacks were similar. They were of sudden onset. She complained of a feeling that she was spinning and that the "floor was moving." There was no impairment of consciousness. There were no precipitating factors. There was no history of otitis media. There were no abnormal neurological signs. An early electroencephalogram was dysrhythmic but two subsequent records within the next five years were normal. Radiological studies of the skull including special views of the petrous temporal bones were normal. She did have two infantile convulsions at the age of 8 months. The tympanic membranes were normal. Hearing was clinically normal and this was confirmed by an audiogram. The caloric tests showed a moderate right canal paresis which persisted.

*Case VII* (C. R.).—A girl aged 2 years and 9 months, her attacks began at the age of 18 months. Her mother heard a scream and looked out to see her fall off her tricycle. She tried to walk and kept falling over. She was perfectly well before the onset of the attack and recovered completely within five to ten minutes. The next attack was six months later. Her mother again heard her scream and she fell over. She recovered completely within a few minutes. There was another attack some three months later and then the attacks subsequently recurred about every ten days. They came on either sitting or standing. She now complains during the attacks that "The house is going round." She grasps her mother's hand to be picked up. Her mother has let her go but finds that she falls over without support. Within a couple of minutes she is perfectly well and goes off again to play. After one attack she asked her mother for her paints and then her mother inquired if the house was still going round and she said "No." On occasions she has lain on the floor and screamed.

Sweating has been noted during some attacks. Consciousness is retained during the attacks. There was no history of ear trouble or upper respiratory tract infection. There was nothing of significance in the past health or family health. Her hearing is normal. There was no abnormality on neurological or general examination. The X-ray appearances of the skull including Stenver's views were normal. The electroencephalogram was normal. The caloric responses showed complete bilateral canal paresis.

*Case VIII (L. F.).*—A boy seen recently had his first attack at the age of 2 years. He had had three attacks in six months. Their duration was less than a minute. In his first attack he said "The bathroom has gone away." In the second attack he said "The room has gone away" and broke out in a sweat and was noted to be very pale. In the third attack he was being carried down the street when he suddenly complained that the road appeared to be "going round and round." He was put down and would have fallen without support. In each attack he clutched his mother and kept close to her. Nystagmus was observed in each attack and was well described. It persisted only for the duration of the giddiness. There was no impairment of consciousness. There were no precipitating factors. There were no abnormal neurological signs. The electroencephalogram was normal. Radiological studies of the skull were normal. There was no personal history of epilepsy. A cousin of his father had epilepsy. The tympanic membranes were normal. Hearing was clinically normal. The caloric tests showed a complete left canal paresis.

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