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Vertigo in Children: Differential Diagnosis and Treatment

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ABSTRACT. A study of vertigo in 50 children showed that a careful neurological examination should be performed for all patients. A detailed family and personal history to find possible episodes of loss of consciousness or seizures should be obtained; 13 children with vertiginous seizures had a positive family history for seizures and 5 had febrile seizures in infancy. Electroencephalograms should be obtained in all instances of vertigo occurrence. Pediatrics, 59:833-838, 1977.

Vertigo has been a long-neglected symptom in children and very few etiologic classifications have been offered. Dizziness or "lightheadedness" may be nonspecific symptoms but true vertigo, described as a sudden sensation of twirling of the individual or of his surroundings, may be a manifestation of abnormality of the vestibular system.1

The most common cause of vertigo in adults is disease of the end organ, the labyrinth within the inner ear. The limited reports on children touch on certain of the possible causes. Involvement of peripheral pathways appears to be the cause of "benign paroxysmal vertigo"2,3 and "vestibular neuronitis,"4 whereas damage to the temporal lobe cortex may produce vertiginous seizures.5

This article discusses the incidence, differential diagnosis, and treatment of 50 pediatric patients who came to the outpatient department of the Bronx Lebanon Hospital Center with the complaint of vertigo. These patients were selected from 80 children referred to the Neurology Clinic between January 1972 and January 1975 with the complaint of "dizziness" because of our special interest in studying their symptoms.

MATERIALS AND METHODS

Each of the 50 patients had an ear, nose, and throat (ENT) examination and a complete neurological examination with emphasis on balance testing. Laboratory workup included complete blood count, urinalysis, and determination in blood or serum of sugar, urea, nitrogen, calcium, phosphate, electrolytes, and T3 and T4 in all patients. Skull and mastoid X-rays, electroencephalogram (EEG), and a hearing test were also performed.

Vestibular testing in all children consisted of electronystagmographic (ENG) recording of positional nystagmus, if present, and of the nystagmic response to perrotatory stimulation. A commercially available (Triacoustics) torsion swing which provides a 180° angular acceleration alternating to right and left at a rate of 60 revolutions per 10 seconds was used as the source of perrotatory stimulation. The nystagmus recorded is the result of stimulation of both labyrinths and represents a summation of responses. All children more than 3 years of age were additionally subjected to bithermal caloric irrigation at 30°C and 44°C of each ear. The technique was based on the method described by Fitzgerald and Hallpike6 in 1942, as

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TABLE I
TYPES OF VERTIGO FOUND IN 50 CHILDREN

<table>
<thead>
<tr>
<th>Type of Vertigo</th>
<th>No.</th>
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<tbody>
<tr>
<td>Central vertigo</td>
<td>42</td>
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<tr>
<td>Vertiginous seizures (idiopathic)</td>
<td>25</td>
</tr>
<tr>
<td>Post-meningitic vertigo</td>
<td>3</td>
</tr>
<tr>
<td>Post-traumatic</td>
<td>4</td>
</tr>
<tr>
<td>Migraine</td>
<td>5</td>
</tr>
<tr>
<td>Psychosomatic</td>
<td>5</td>
</tr>
<tr>
<td>Peripheral vertigo</td>
<td></td>
</tr>
<tr>
<td>Vestibular neuronitis</td>
<td>5</td>
</tr>
<tr>
<td>Paroxysmal benign vertigo</td>
<td>2</td>
</tr>
<tr>
<td>Congenital deafness</td>
<td>1</td>
</tr>
</tbody>
</table>

TABLE II
RESULTS OF EEG TRACINGS

<table>
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<tr>
<th>EEG</th>
<th>Diffuse Paroxysmal Discharge</th>
<th>Temporal Paroxysmal Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>11</td>
<td>14</td>
</tr>
<tr>
<td>ENG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>No response</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>EEG/ICC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No change</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Post slow</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Not done</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

modified by Jongkees and Philipszoon. The recording of the nystagmic response was performed on a one-channel dynograph ac recorder. Calculation of the results was based on Jongkee's formula to determine the presence of "labyrinthine preponderance, directional preponderance, no preponderance or no response." The term "labyrinthine preponderance" is used when one labyrinth responds to hot and cold water stimulation with a significantly greater intensity of nystagmus than the other labyrinth. A significant difference in response between right and left is more than 14%. Directional preponderance indicates that the nystagmus evoked by bithermal irrigation has significantly greater intensity (more than 18%) in one direction than in the other. "No preponderance" indicates equal responses from both labyrinths. "No response" means that no nystagmus was elicited during bithermal stimulation.

Two children in the series who were 2 and 3 years old, respectively, were subjected to ice-cold caloric stimulation instead of bithermal calorization according to a modification of Kobrak's method. A good response to ice-cold caloric stimulation was defined as nystagmus of between 180 to 250 seconds' duration. When the duration of nystagmus was between 90 to 180 seconds, the response was considered fair, and a response under 70 seconds' duration was considered poor. Twenty normal children were used as controls.

Twenty of the children in whom the diagnosis of vertiginous seizures or vestibulogenic seizures were suspected had electroencephalography (EEG) simultaneously with a 10-second and a 30-second ice-cold caloric stimulation of the labyrinths.

RESULTS

Neurological examination of the children showed that 3 had sensorineural hearing loss, 1 had mild mental retardation, 1 had a learning disability, 12 had minimal difficulty on balance testing such as the Romberg or stepping test, and a few had hyperactive reflexes. All ENT examinations results, except one, were normal. No metabolic abnormalities were detected.

Based on the results of the various tests, the children were separated into groups corresponding to the etiology of vertigo. Forty-two were diagnosed as having "central vertigo" and eight, "peripheral vertigo" (Table I). Those with vertiginous seizures were separated into two groups in accordance with the results obtained in the EEG tracings (Table II). The first group comprises 14 children ranging from 3 to 18 years of age. All complained of attacks of paroxysmal vertigo, occurring at times independently and at times accompanied by headaches, nausea, and vomiting, and culminating in loss of postural control and loss of consciousness with or without hypertonicity. A few had occasional focal seizures manifested by twitchings of the face and arm, inability to speak, and drooling. EEG studies showed unilateral or bilateral mid-posterior temporal paroxysmal spike and wave discharges and a right frontal temporal focus in two children. Brain scan results were negative. Five of the children had positional nystagmus, three had directional preponderance to the side of the epileptic focus on bithermal stimulation, and six had labyrinthine preponderance to the same side which showed the focus of paroxysmal discharges. Of the 14 children, 12 had simultaneous EEG recording and 10-second and 30-second irrigations of ear canals. There was no change in the EEG tracing in ten children whereas two showed slowing in the theta range and occasional phase reversals in the posterior temporal leads (bipolar...
recording), lasting for 30 to 40 seconds after the end of irrigation. Symptoms were controlled in all with anticonvulsant medication. All had normal hearing.

A second group of 11 children with vertiginous seizures, ranging in age from 3 to 14 years, was characterized by the presence of a diffusely abnormal EEG tracing with paroxysmal spike and wave discharges occurring synchronously from all leads. They suffered episodic headache and vertigo, most often expressed as a sensation of twirling and occasionally as a back and forth or sideways swaying of the body. These attacks were sometimes followed by loss of postural control and loss of consciousness culminating at times in a tonic or tonic-clonic generalized generalized seizure. One child had staring spells as well as vertiginous spells and an EEG compatible with petit mal.

Of the 11 children, 8 had a normal response to torsion swing and to bithermal caloric stimulation. Two showed no response to perrotatory, bithermal labyrinthine stimulation and ice-cold caloric stimulation of the labyrinths suggesting "dead labyrinths." One of the two had a unilateral poor response to ice-cold caloric irrigation of the ear canals (hypoactive labyrinth). One of the 11 children had directional preponderance on bithermal caloric stimulation although his EEG failed to demonstrate a paroxysmal focus. Eight children had a 10-second and 30-second ice-cold caloric irrigation of the labyrinths during EEG recording. There was no change in the EEG tracing in seven children, and one showed slowing in the theta range in the posterior temporal leads lasting around 40 seconds. These children all showed difficulty on some of the balance tests like extended Romberg, heel-toe-gait blindfolded or stepping test. They responded well to anticonvulsant medication. Hearing tests were normal in all.

Head trauma is a well-recognized cause of vertigo.10-13 Four male children 3 to 12 years of age had post-traumatic concussion vertigo. None had evidence of skull fracture. Attacks of vertigo and headache appeared a few days to a few weeks following the trauma. Two had a history of brain concussion, diffusely abnormal EEGs with posterior slowing, and normal response to bithermal calorization. Two had evidence of brain contusion, focal EEGs, and abnormal calorics (one had labyrinthine preponderance and the other, directional preponderance to the side of the EEG focus). All four had positional nystagmus. Three children required anticonvulsant medication for several months. The fourth has been receiving medication for several years because of recurrent vertiginous seizures most likely secondary to the trauma.

Three children, two males and one female, ages 7 to 13 years, suffered episodes of vertigo following meningitis. Both males developed sensorineural hearing loss, tinnitus, and labyrinthine preponderance in response to bithermal stimulation after mumps meningoencephalitis. One was mildly retarded and the other had a learning disability. The girl developed attacks of vertigo and focal seizures with a paroxysmal temporal EEG as a sequela of bacterial meningitis. Electronystagmography showed directional preponderance on bithermal caloric stimulation. She had no hearing loss and responded well to anticonvulsant medication. She can be considered as a case of "symptomatic" vertiginous seizures secondary to meningitis.

Two boys and three girls, aged 7 to 16 years, suffered severe vertigo as "auras" to migraine attacks. Occasionally, the vertigo would persist beyond onset of the severe headache, photophobia, nausea, and vomiting. There was no history of loss of consciousness. The headache and vertigo subsided in response to analgesics and ergotamine. All four children had strong family histories of migraine, normal EEGs, and abnormal responses to bithermal caloric stimulation.

Five adolescent girls and one adolescent boy had chronic headaches and daily episodes of vertigo without nausea, vomiting, or loss of consciousness. They were tense, anxious individuals with obvious emotional conflict and a depressive reaction. All physical and laboratory test results were normal and their symptoms subsided with psychiatric care and psychotropic medication.

Two girls, 2 and 3 years old respectively, experienced attacks of unsteadiness which caused them to seek stability, either by clinging to their mothers or by lying still on the floor for the entire duration of the attack. They appeared pale and scared and were ataxic and dysmetric when tested. No impairment of consciousness was noted in either. Nystagmus was observed in one during attacks. Both had normal EEG tracings. One child had a normal response to torsion swing stimulation, poor response to ice-cold caloric irrigation on the right, and a good response on the left. The other child had no nystagmic response to torsion swing stimulation, no response to ice-cold caloric irrigation on the right, and a poor response to irrigation on the left. After numerous daily episodes for several weeks, the attacks subsided spontaneously in one child and were significantly improved by administration of dimenhydrinate in
the other. These children had classical history and laboratory findings compatible with benign paroxysmal vertigo.

The diagnosis of vestibular neuronitis was established in three boys and two girls, ranging from 7 to 10 years of age, who had acute onset of vertigo and nausea without impairment of hearing or evidence of central nervous system (CNS) disturbance a few days following an upper respiratory infection and mild earache. Two had episodes of loss of postural control without loss of consciousness. Results of the neurological and ENT examinations were normal. All the children had normal EEG tracings. Two showed labyrinthine preponderance on bithermal caloric stimulation whereas three had normal vestibular examinations two weeks after onset of symptoms. The latter three children recovered spontaneously within ten days of onset of symptoms. The two other children were treated with dimenhydrinate.

A 7-year-old child had congenital bilateral profound sensorineural deafness. He was evaluated because of recurrent attacks of vertigo and nausea, at three- or four-month intervals, lasting for a couple of days and preceding upper respiratory infections (URI). The attacks had occurred over a period of three years without loss of consciousness but with postural balance affected. Electroencephalography results were normal. Electroystagmography showed absent responses to perrotatory and bithermal labyrinthine stimulation. There was a unilateral fair response to ice-cold caloric stimulation. The history of this child revealed delay in acquisition of motor milestones and some disorientation in space when swimming with his head submerged in the water. Neurological testing showed poor performance in the balance tests. It is very likely that the attacks of vertigo in this child were due to a buildup of endolymphatic pressure in the congenitally hypo-functional vestibular organs during incubation of an URI.

**DISCUSSION**

The present study indicates a high incidence of "central vertigo" (42 of 50) in children. A definite diagnosis of vertiginous seizures was established in 27 of the 50 children. A majority of these children whose EEG recordings contained unilateral or bilateral paroxysmal temporal lobe discharges had directional preponderance on bithermal labyrinthine stimulation which has been shown for children11 to indicate a CNS disturbance. Since the superior posterior part of the temporal lobe appears to be the cortical representation of the vestibular system,15-17 it is reasonable to assume that the directional preponderance demonstrated by electronystagmography in such children is the result of cortical temporal damage.

It is customary to believe that labyrinthine preponderance suggests the presence of a unilateral labyrinthine disease. The patients with vertiginous seizures had no evidence of peripheral disease and it is possible that the presence of labyrinthine preponderance in a few cases is indicative of hypersensitivity of the labyrinth in the presence of affected vestibular neurons at the level of the reticulocortical pathways.

The presence of labyrinthine and directional preponderance in a few patients with vertiginous seizures should probably be considered as if they had directional preponderance alone as demonstrated in another study by the authors (in press).

The excellent response to anticonvulsant therapy in all cases of vertiginous seizures proves that the symptom of vertigo can be controlled in a majority of cases just like any form of convulsive disorder, with depression of neuronal hyperexcitability.

The majority of children (8/11) in whom the EEG abnormalities were of a diffuse paroxysmal nature showed no abnormal response to vestibular stimulation, suggesting that their peripheral organs, as well as their cortical representations were intact. The sensation of vertigo and nausea experienced prior to the loss of consciousness may be regarded as the equivalent of an aura which could result from synchronized hyperactivity in the damaged reticular system of the brainstem acting as a major relay for vestibular pathways.18 Absent responses to labyrinthine stimulation in two children suggest abnormal labyrinthine organs. It is possible that in these two children transneuronal degeneration accounts for neuronal damage at the level of the reticular formation, where centrencephalic seizures of the vertiginous type probably originate.

An additional mechanism for seizure production was suggested by Behrman and Wyke19 in 1958. This type, designated "vestibulogenic seizure," depends on the initiation of convulsive activity in the reticulo-cortical system by abnormal activation of the reticular brainstem system from a hyperactive labyrinth. In three children with vertiginous seizures, ice-cold caloric stimulation of the labyrinths produced post-temporal slowing of the EEG. This in itself may not be sufficient evidence for vestibulogenic seizure but it is suggestive of the influence of peripheral
vestibular stimulation on previously damaged posterior temporal cortex.

The common presence of positional nystagmus following head trauma probably indicates the presence of a CNS insult involving the vestibulomesencephalic tract. It was a consistent finding in the three children with post-traumatic vertigo.

Post-meningitic vertigo is ascribed to damage of vestibular pathways secondary to the inflammatory process. The presence of peripheral organ involvement in bacterial meningitis has also been described. In two of three patients, the presence of sensorineural hearing loss and labyrinthine preponderance in response to bithermal caloric stimulation suggests involvement of both the peripheral organ (where both the vestibular and cochlear nerves are in close proximity to each other) as well as involvement of vestibular pathways. Indeed, two patients also had evidence of diffuse CNS damage, manifested by mental retardation in one child and minimal brain dysfunction with mild abnormalities in the EEG of the other. This latter patient, who developed vertigo and hearing loss after mumps meningoencephalitis, responded with worsening of his symptomatology when treated with dimenhydrinate and anticonvulsant medication. He improved when he received methyiphenidate, possibly because of this drug's effect at the level of the amine receptors of the reticular formation and the increase in level of arousal until dizziness was overcome.

Only 2 of the 50 children studied had a typical form of benign paroxysmal vertigo. This entity may be difficult to recognize since it occurs more frequently in young children who have difficulty in describing their symptomatology and the unobservant parent may not provide an adequate description to the examiner. The diagnosis is made in the presence of attacks of imbalance without loss of consciousness but with abnormalities during vestibular testing and a normal EEG tracing. Hearing deficits are not described in this syndrome, and they were absent in our patients.

No children with Meniere’s syndrome were seen in this series. However, five had symptomatology characteristic of vestibular neuronitis. This term was first introduced by Hallpike in 1949 and later described by Dix and Hallpike in 1952 and more recently discussed by Lindsay in 1967. It is characterized by recurring episodes of vertigo without auditory or CNS symptoms or signs but with reduced caloric excitability of the labyrinth. Symptoms appeared following a mild earache or signs of a mild viral illness in a majority of the cases described. A similar history was obtained in our patients.

Our present methods of evaluation do not permit a clear-cut differentiation between the syndrome of benign paroxysmal vertigo of younger children and vestibular neuronitis characterized by a subacute protracted course following an inflammatory episode in older children and adults.

**SUMMARY**

The high incidence of central vertigo in our study suggests that a careful personal and family history to note possible episodes of loss of consciousness or seizures should be obtained and a careful neurological examination should be performed in all cases. Thirteen children with vertiginous seizures had a positive family history for seizures and five children had a history of febrile seizures in infancy.

No case of brain tumor was included in this series but since the association of vertigo and brain tumors is known, the value of a careful neurological evaluation cannot be overemphasized. Presence of cranial nerve deficits or of cerebellar signs will alert the clinician to this diagnosis. An EEG test should definitely be obtained in all cases in which loss of consciousness or possible seizures have been reported.

Electronystagmography seems particularly indicated in cases of peripheral vertigo, which should be suspected in children with congenital deafness or in youngsters with a history compatible with benign paroxysmal vertigo or vestibular neuronitis. It is worth mentioning that none of the children had metabolic abnormalities which is compatible with the current pediatric experience that metabolic abnormalities in children more commonly produce dizziness than vertigo as opposed to reports of “metabolic vertigo” in adults (Pulec).

The use of combined EEG recordings with irrigation can be limited to investigative laboratories and does not seem to have practical value for the clinician in practice.

**REFERENCES**

4. Lindsay JR: Paroxysmal postural vertigo and vestibular

TRUE SCIENCE

... Medicine ... is a great science, for all its failings. I reject completely the conventional view which says that mathematics and physics are the true, basic sciences; that other sciences enjoy merit and intellectual vigour only to the extent that they rest on mathematics and physics. It is true that one can work "down" from medicine through pathology to genetics and cell biology, to biochemistry, to chemistry, and thence to physics and mathematics. Equally one can work "up" from biology and medicine to demography, sociology, economics, and political science. But these hierarchies represent descriptive conveniences and conventions, classifications of components in terms of size. They cannot be listings of value or of intellectual achievement. Biologists and doctors have nothing to be ashamed of in front of their colleagues in the physical sciences.

G. J. V. NOSSAL
Lancet 2:840, 1976

Noted by Student
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