Concern that vestibular stimulation may induce seizures in seizure-prone children has been based on hearsay and unconfirmed clinical impressions of practicing therapists. To clarify this issue, we took electroencephalographic recordings of seizure-prone children before, during, and after specific vestibular stimulation. Ten children with seizure histories, 5 to 15 years of age, were exposed to warm and cold caloric vestibular stimuli. Electroencephalographic activity was recorded before, during, and after each vestibular stimulus; recordings were rated and compared prevestibular and postvestibular stimulation. Electronystagmographic recordings were also taken. Results show that vestibular stimulation does not accentuate the abnormal brain wave pattern in seizure-prone children. Six of 10 subjects had a significant reduction in paroxysmal activity ($p < .02$). Possible explanations for clinical reports of vestibular induced seizures are given, with suggestions for precautions when applying vestibular stimulation to seizure-prone children.

Key Words: Vestibular function tests, Seizures, Pediatrics.

Increased use of vestibular stimulation in clinical settings has generated concern over potential dangers of indiscriminate application of this form of sensory input. Specifically, clinicians have reported seizure activity in seizure-prone children after application of vestibular stimulation. Some clinicians have questioned the advisability of allowing children with a seizure history to participate in playground activity that produces a vestibular component of stimulation.

Electroencephalographic (EEG) disturbances may be divided into two categories. A nonparoxysmal disturbance is one that is an integral part of the overall basic pattern of the background rhythm of electrical brain activity. Nonparoxysmal activity may include a focal, or generalized, excessive slowing of the rhythmical electrical activity of the brain, as well as voltage asymmetries and excessively fast rhythmical activity. A paroxysmal disturbance is one that arises abruptly out of the basic pattern in a bursting fashion. Paroxysmal activity may be observed in EEG recordings as high voltage discharges (spikes), slow wave volleys, and complexes of spikes and slow waves. It is this paroxysmal abnormality that is associated with subclinical seizures and overt clinical epileptic crises.

Behrman and Wyke differentiate between vestibular and vestibulogenic seizures. The former arise from cortical projection areas of the labyrinth, but are not the result of vestibular end organ stimulation. Vestibulogenic seizures arise from the reticular brainstem area as a result of vestibular end organ stimulation. The few published reports of EEG response in seizure-prone subjects show mixed results. Molnar and associates reported that 25 of 36 subjects were affected by vestibular stimulation with 16 of the 25 showing a decrease in the number of paroxysms recorded. Barac used both rotatory and caloric stimulation in a sample ranging in age from 10 to 61
### TABLE 1
Data about Seizure-Prone Subjects

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age (yrs)</th>
<th>Seizure History</th>
<th>Medication</th>
<th>Developmental History</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>13</td>
<td>febrile seizures, stress seizures</td>
<td>mephobarbital</td>
<td>unremarkable; all-A student (on track team)</td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>psychomotor seizures</td>
<td>primidone, carbamazepine, phenobarbital</td>
<td>some problem with hyperactivity in past</td>
</tr>
<tr>
<td>3</td>
<td>11</td>
<td>grand mal seizures for 4 years</td>
<td>phenobarbital, ethosuximide</td>
<td>learning disability, held back one year in school</td>
</tr>
<tr>
<td>4</td>
<td>8</td>
<td>grand mal</td>
<td>phenytoin, carbamazepine</td>
<td>hyperactive</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>classic petit mal</td>
<td>trimethadione, ethosuximide, phenytoin, phenobarbital</td>
<td>mental retardation</td>
</tr>
<tr>
<td>6</td>
<td>10</td>
<td>grand mal seizures since birth</td>
<td>valproic acid, carbamazepine, phenobarbital</td>
<td>problem with hand-eye coordination, behavioral problems in learning disability classroom, IQ 64-77 (borderline)</td>
</tr>
<tr>
<td>7</td>
<td>13</td>
<td>grand mal seizures, absence spells</td>
<td>hydroxyzine pamoate, valproic acid, carbamazepine</td>
<td>significant developmental delay, severe hyperactive mentally retarded</td>
</tr>
<tr>
<td>8</td>
<td>12</td>
<td>vestibular seizures, paroxysmal vertigo</td>
<td>valproic acid</td>
<td>episodes of dizziness of unknown etiology, hyperactive, adequate in school although immature attention span, problems with spatial relationships, home problems, poor visuomotor coordination, learning disability?</td>
</tr>
<tr>
<td>9</td>
<td>8</td>
<td>staring spells, lapses of consciousness</td>
<td>ethosuximide</td>
<td>unremarkable</td>
</tr>
<tr>
<td>10</td>
<td>15</td>
<td>right focal motor seizures</td>
<td>phenobarbital, carbamazepine, valproic acid</td>
<td>clumsy gait—internal rotation and planovagus feet, learning disability, near average IQ (88), minimal brain dysfunction</td>
</tr>
</tbody>
</table>

He concluded that vestibular stimulation seldom provided epileptic discharges not previously present in the resting, waking EEG. He also reported a tendency, during vestibular stimulation, toward modification of EEG patterns in an asymmetrical, although not necessarily pathological, way. In all these studies the subjects were predominantly adults, 18 years of age and older.

The cited literature is equivocal as to vestibular stimulation effects on seizure or abnormal brain wave activity. Peripheral sensory stimulation, including vestibular stimulation, can function as an activating mechanism in seizure activity. However, reflex seizures (seizures activated by peripheral sensory stimulation of any sensory modality), represent less than 1 percent of all seizures in children and adults. Reflex seizures induced by vestibular stimulation, or vestibulogenic seizures, are very rare clinical events. Paradoxically, peripheral sensory stimulation has been reported to arrest the development of a seizure in a number of instances.

Because the literature revealed a limited number of observations of EEG response to vestibular stimulation in children this study was undertaken to clarify clinical impressions. Specifically, we hypothesized that semicircular canal stimulation would have no effect on EEG abnormalities in seizure-prone children.

**METHOD**

**Subjects**

Eight girls and two boys, between the ages of 5 and 15 years, were studied. Each subject had a type of seizure disorder confirmed by a history of abnormal EEGs and clinical seizures. Ongoing anticonvulsant medication was not altered for purposes of this study. None of the subjects had received vestibular stimulation therapy before. Table 1 lists age, seizure history, drug(s), and developmental history of each subject.

**Procedure**

Scalp electrodes were placed over the bilateral frontal, central, occipital, and temporal areas of the subject. The subject was placed supine, with the head in the midline, raised 60 degrees from the supporting surface. This placed the horizontal semicircular canals in the vertical plane. Intrahemispheric chain linked
bipolar montages were recorded on a Beckman, model CG, EEG polygraph.* A prestimulation period of 5 to 10 minutes was recorded to establish a baseline of EEG activity. Horizontal semicircular canal stimulation was then provided by conventional caloric stimulation. Distilled water (250 cc) at 30°C and 44°C was introduced, over a 30-second period, into the external auditory meatus in the following order: cold water left ear, cold water right ear, warm water left ear, warm water right ear.14 The poststimulation period consisted of the 30-second period during which the caloric stimulation was applied, plus 120 seconds after the cessation of the stimulus for each stimulation period. The effectiveness of the stimulus was evaluated by monitoring the electronystagmogram (ENG) that replaced one of the EEG lines of the prestimulation recording. The EEG was recorded continuously throughout the entire poststimulation period, which lasted approximately 10 minutes.

Caloric stimulation was used for two reasons: 1) to eliminate the effects of other afferent stimuli such as proprioceptive and tactile stimuli that are present during rotatory vestibular stimulation and 2) to reduce the amount of artifact on the EEG recordings resulting from the crossing of EEG leads during rotatory stimulation.

The room lights were dimmed and the subjects were instructed to keep their eyes closed during the stimulation and poststimulation periods. Subjects were also asked to perform simple mental arithmetic during and following stimulation to maintain mental arousal. Both of these conditions are necessary to facilitate nystagmus.

All EEG recordings were scored by three different raters; two experienced EEG technicians and a practicing neurologist. They used a scale of 0 to 4, with 0 being normal and 4 being severely abnormal (Fig. 1). Tracings were scored separately by each rater for paroxysmal and nonparoxysmal abnormalities in both the prestimulus and poststimulus conditions. Interrater reliability was $r = .96$ ($p < .01$) using Pearson's product-moment correlational analysis (Fig. 2).

RESULTS

Six subjects showed a decrease in paroxysmal EEG activity from prestimulation to poststimulation recording. These same six showed no change in nonparoxysmal activity. Four subjects demonstrated no nonparoxysmal or paroxysmal activity at any time (Tab. 2). The mean ratings of nonparoxysmal and paroxysmal activity for all 10 subjects prestimulation and poststimulation are shown in Figure 3. A paired $t$-test analysis showed the decrease in paroxysmal activity significant at the $p < .02$ level. Decreases in nonparoxysmal activity were not significant. There was some variance in individual changes in paroxysmal ratings with subjects #2, #4, #9, and #10 showing marked decreases from prestimulation to poststimulation ratings (Fig. 4). The ENG recordings were within normal limits for all 10 subjects, indicating an effective stimulus and a physiologically normal end organ.

The EEG recordings from subjects #3 and #10 are examples of variations in response (Tab. 2). For

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* Beckman Instruments, Inc, Fullerton, CA 92634.

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**FIG. 1.** Scale used to rate presence of paroxysmal and nonparoxysmal activity in EEG recording of the subjects.

**TABLE 2**

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Change in Nonparoxysmal EEG Activity</th>
<th>Changes in Paroxysmal EEG Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>2</td>
<td>O</td>
<td>↓</td>
</tr>
<tr>
<td>3</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>4</td>
<td>O</td>
<td>↓</td>
</tr>
<tr>
<td>5</td>
<td>O</td>
<td>↓</td>
</tr>
<tr>
<td>6</td>
<td>O</td>
<td>↓</td>
</tr>
<tr>
<td>7</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>8</td>
<td>NO</td>
<td>NO</td>
</tr>
<tr>
<td>9</td>
<td>O</td>
<td>↓</td>
</tr>
<tr>
<td>10</td>
<td>O</td>
<td>↓</td>
</tr>
</tbody>
</table>

* O = no change, ↓ = decrease, NO = nonparoxysmal or paroxysmal activity not observed at any time.

**FIG. 2.** Mean rating for all subjects as determined by each rater (R1, R2, R3) for all experimental conditions (prestimulation and poststimulation, nonparoxysmal and paroxysmal). Interrater reliability was >.95.
subject #3, an 11-year-old boy with a four-year history of grand mal seizures, response before and after caloric stimulation was normal (Fig. 5). Subject #10, a 15-year-old girl with right focal motor seizures, showed almost continuous paroxysmal activity in the 10-minute prestimulation period. In the 10-minute

![Fig. 3](image)

**Fig. 3.** Mean rating for all subjects and all raters for each experimental condition. The degree of paroxysmal abnormality decreased significantly ($p = .02$) while the non-paroxysmal activity remained unchanged.

![Fig. 4](image)

**Fig. 4.** Mean rating change (poststimulation score minus prestimulation score) for paroxysmal disturbances averaged across all raters for each subject. Subjects 1, 3, 7, and 8 showed no change and were scored as having a normal EEG in the prestimulation period. Remaining subjects showed marked decreases in abnormal activity.

![Fig. 5](image)

**Fig. 5.** The EEG of subject #3 was interpreted as normal both before (above) and after (below) caloric stimulation of the horizontal semicircular canals. The only change was some reduction in the alpha activity and the introduction of some faster activity in the beta range characteristic of arousal. Key: O—alpha waves; □—beta waves; ?—simple math questions (asked of subject periodically to maintain his arousal). Alpha waves usually appear in posterior head regions. Beta waves are a lower amplitude, higher frequency wave than alpha waves and appear in anterior regions of the head.
poststimulation period only one paroxysmal disturbance was recorded (Fig. 6).

**DISCUSSION**

Results of this investigation indicate that semicircular canal stimulation does not accentuate the nonparoxysmal and paroxysmal abnormalities seen in the EEG recordings of seizure-prone children. In fact, a significant reduction in paroxysmal activity was observed during and after semicircular canal stimulation. Nonparoxysmal activity remained unchanged throughout the poststimulation period. Barac reported a suppression of epileptic foci in seizure-prone subjects, but in an asymmetrical manner depending upon the duration of the semicircular canal stimulation. There was no asymmetry of cortical response to semicircular canal stimulation in our 10 subjects. The EEG changes we found were induced bilaterally.

Clinical impressions relating semicircular canal stimulation and seizures are not supported by the data collected in this study. Available literature, supported by our findings, indicates that a seizure directly induced by semicircular canal stimulation is an extremely rare event. The Gastaut and Tassinari study of triggering mechanisms in epilepsy indicated that the few cases of epileptic discharge triggered by vestibular stimulation were too doubtful to be included as one of the causes of sensory-induced seizures. Some untrained observers may have mistakenly identified tonic activation of vestibulospinal reflexes as a seizure. In our experience children who have little or no experience in being spun and who are seizure or nonseizure prone often demonstrate a tonic activation of extensor groups that may last up to 5 seconds after the cessation of spinning. This reflex response is mediated by the vestibulospinal tracts.

An attempt was made in our study to isolate semicircular canal stimulation from other forms of sensory stimulation. The subject was made as comfortable as possible in a quiet EEG recording studio. We routinely explained, in detail, all parts of the procedure, and attempted to reassure the subject throughout the recording session. We eliminate the usual noise, hustle, and bustle of an active therapy clinic. Other sensory modes could be responsible for

![Fig. 6. The EEG of subject #10 showed moderate to moderately severe generalized abnormality as well as a moderately severe paroxysmal disturbance in the prestimulation condition (above). Key: O—paroxysmal activity; □—generalized abnormality. In the poststimulation condition (below) the generalized abnormality is unchanged, but the paroxysmal disturbance showed marked improvement (that is, only one paroxysm was recorded in the poststimulation period as noted in circled area). "Face scratched" by patient at this point in recording; "Wrong answer" given to question (?) asked just before the only poststimulation seizure.](image-url)
clinically reported seizures. Fatigue and excitement, both of which may be experienced by a child in a clinical setting, are more closely linked to seizure activity than is vestibular stimulation.

Seizures could result from semicircular stimulation with eyes open in a lighted room. The rhythmical interruption of light impinging on the retina can trigger the seizure. Most EEG laboratories routinely use a stroboscopic light at 8 to 10 Hz with patients suspected of having seizures in an attempt to activate epileptic foci. Nystagmus, generated by semicircular canal stimulation, can approach a frequency of 10 Hz. A similar stroboscopic effect would be produced when visual targets are intermittently held and lost during the alternating slow and fast phases of nystagmus. Our subjects kept their eyes shut, eliminating the chance of any visual, stroboscopic activation of latent abnormal EEG activity.

Hyperventilation is also a potential activating mechanism of latent abnormal brain wave patterns in EEG laboratories. Vestibular stimulation may increase respiration rate, which can possibly lead to short periods of hyperventilation. Some seizure-prone children may demonstrate subclinical or clinical paroxysmal recordings as a result of hyperventilation. If either hyperventilation or photogenic seizures are suspected as being present, the therapist should contact the child's neurologist to determine if these activating mechanisms were used in previous EEG recordings and whether paroxysmal effects resulted.

The neurophysiological substrate for the reduction of paroxysmal activity in 6 out of 10 subjects after vestibular stimulation may be in the cerebellum. In Snider's review of the literature related to cerebellar influences on seizure discharges, he reports that electrical excitation of either the cerebellar nuclei (fastigial) or cortex can alter or stop seizure discharges initiated in the neocortex. Snider suggests that the inhibitory functions of the Purkinje cells of the cerebellum are somehow recruited by the electrical stimulation and exert control on abnormal cerebral paroxysms through cerebellar reticular or cerebellar thalamic connections. Vestibular stimulation through direct and indirect connections to fastigial nuclei and cerebellar cortex could recruit these cerebellar inhibiting mechanisms and cause a reduction in paroxysmal disturbances present in various areas of the neocortex.

CONCLUSIONS

Caloric stimulation of the horizontal semicircular canals of 10 seizure-prone subjects, aged 5–15 years, showed no facilitation of abnormal EEG activity. In fact, data from this study showed a significant decrease in paroxysmal abnormalities in six of the subjects with no change in the EEG activity of the remaining four subjects. If clinical reports of seizures associated with spinning are correct, however, then it may be advisable to restrict a subject’s vision during and shortly after cessation of spinning to reduce the possibility of photogenic seizures. Also, children should be monitored for signs of hyperventilation during and shortly after spinning. Therapists should question the neurologist concerning previous EEG recordings where photic and other activation methods were used. This study and some of the available literature do not discount the possibility that certain seizure-prone children may experience a vestibulogenic seizure.

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