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Tilt Test for Diagnosis of Unexplained Syncope in Pediatric Patients

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ABSTRACT. Thirty-five teenage patients with a history of presyncope or syncope underwent passive head-up tilting to reproduce symptoms of syncope. If tilting alone did not induce syncope, isoproterenol infusion was given to increase heart rate to 150 to 160 beats per minute. In 80% of patients with a history of syncope, identical symptoms could be reproduced during tilting: an abrupt fall in blood pressure combined with profound nodal bradycardia, ranging from 32 to 86 beats per minute. These symptoms were quickly reversed by returning the patient to the supine position. For patients with frequent occurrences of syncope, especially when there was a history of trauma sustained during these episodes, a therapeutic regimen of either β blockers or 9α-fluorocortisol was begun. The mechanism of this common cause of syncope in childhood is neurocardiogenic in response to venous pooling and catecholamine-induced tachycardia. The tilt test is an excellent and cost-effective test for the workup of unexplained syncope in children.

Syncope in the pediatric age range is for most parents and practicing physicians an alarming symptom. The child, usually a teenager, will typically be hospitalized for extensive workup. Neurology consult, electroencephalogram, computed tomographic scan, Holter monitoring, even “routine” cardiac catheterization and electrophysiologic studies usually yield little, if any, useful information. Already in 1982, Kapoor et al1 called for a more cost-effective approach to the diagnostic workup. Although syncope occurs in all age-groups, there appear to be two major groups in which it occurs: teenagers2 and adults older than 50 years.3-5 In recent years the head-up tilt test has been reported as a useful diagnostic test for adults.5-10 To date we have found no study using the tilt protocol for the pediatric age-group. We modified this protocol slightly; it is an outpatient procedure for evaluation of patients with presyncope or syncope. In this paper we report the initial results of this test and our therapeutic approach to this problem.

METHODS AND PATIENT SELECTION

Thirty-five patients (17 boys, 18 girls; 8 to 19 years of age) were referred for evaluation; 6 of these patients had spells of dizziness or blacking out (loss of vision without loss of muscle tone); 29 had episodes of complete loss of consciousness. Most of these patients had repeated episodes; some had sustained minor injuries when falling; one girl had such frequent episodes that she was unable to attend school. These episodes occurred while patients were in the upright position and were not accompanied by focal neurologic symptoms. In some cases episodes occurred in context with physical exertion. The past history of these patients was otherwise unremarkable. All patients had a thorough physical examination. Whereas some patients were referred for the tilt test after extensive neurologic testing was negative, more recent referrals had only an electrocardiogram and echocardiogram. None of our patients had cardiac disease. In three families more than one member had episodes of syncope.

Tilt Protocol

Our aim was to develop a useful, efficient tilt protocol. We found it impractical to use prolonged supine resting periods or extensive (60 minutes) tilt periods. We also found no use for partial tilt such as 30° or 45°. Our tilt protocol (Table 1) takes 30
TABLE 1. Tilt Protocol*

1. Supine resting state for 10 min.
2. Tilt to 60° for 10 min; record symptoms.
3. Return patient to supine position; after 2–3 min start isoproterenol infusion at 1 µg/min; when isoproterenol effect is apparent and consistent, re-tilt to 60°.
4. 60° tilt for 5–10 min; if no symptoms, increase isoproterenol to 2 µg/min for 2–3 min, then 3, 4, and, maximally, 5 µg/min until a heart rate of 150–170 beats/min is reached.
5. When you decide to terminate the test, discontinue isoproterenol first (if symptoms permit); after isoproterenol effect wears off, return patient to supine position.
6. Record recovery for 3–5 min.

* Record blood pressure and electrocardiographic rhythm strip every 2 min (time and heart rate should be printed on strip). The test may be terminated whenever symptoms require it, but try to record a full tilt response, preferably with loss of consciousness. When patient shows signs of incipient syncope (drop in heart rate), turn on electrocardiographic recorder to record rhythm and rate as well as time.

to 60 minutes, depending on how rapidly a “positive” test can be induced. An intravenous drip is needed for administration of isoproterenol. Throughout the test the electrocardiogram is continuously monitored. A lead with a clearly visible P wave is selected. The electrocardiogram, including time and heart rate, is printed out every 2 minutes. Blood pressure readings (by cuff) are recorded every 2 minutes, together with comments such as degree of tilt, medication, patient symptoms, etc. The tests were done in a radiologic suite where motorized tables that tilt from the supine to the upright position are standard equipment.

Definitions and Test Interpretation

The vasodepressor response consists of presyncope or syncope. Presyncope are vasovagal symptoms, subjective and objective: the patient does not feel well; may experience dizziness, with or without blackout; is cold, sweaty; has a thready pulse and low blood pressure; but does not lose consciousness. In syncope, presyncopal symptoms are followed by loss of muscle tone and consciousness. Test results can be negative (normal), positive, or borderline.

The typical normal test result in a teenage patient is characterized by a resting heart rate of 60 to 70 beats per minute (sinus rhythm) and a blood pressure of 110/70 mm Hg in supine position (Fig. 1). Upon 60° upright tilt the heart rate rises to 80 to 90 beats per minute with a small, transient fall in blood pressure. Return to the supine position reestablishes baseline values. Isoproterenol infusion of 1 µg/min increases the heart rate to 100 to 110 beats per minute. Adding 60° tilt increases the heart rate further, but sinus rhythm is present at all times. The heart rate can be increased to 140 to 160 beats per minute with higher isoproterenol doses. After discontinuation of isoproterenol and return to the supine position, resting values return. The blood pressure changes little throughout the test.

A positive result (Fig. 2), either without or with isoproterenol infusion, shows initially the same normal increase in heart rate during 60° tilt. A few
minutes later, however (typically within 3 to 10 minutes), presyncope supervenes. Nodal bradycardia with a full vasovagal response is observed. These symptoms, which are reproducible, are quickly reversed by return to the supine position.

A borderline result has unusually wide swings in blood pressure and heart rate (>15 mm Hg and >15 beats per minute); the patient feels dizzy when asked, but is without visible signs of a vasovagal reaction.

RESULTS

Twenty-six patients had positive results, 3 studies were borderline, and 7 were negative. In positive studies the average heart rate during the vasodepressor reaction was 58 beats per minute; the rhythm was (with one single exception) nodal. The acuteness of the full reaction was striking: the precipitous drop in heart rate often occurred within 10 to 15 seconds.

Of the 6 patients with a history of presyncope (Table 2), 3 had a normal tilt response, 1 response was borderline, and 2 were positive. By contrast, a positive response was seen in 24 of the 29 patients with a history of syncope, 1 patient had a borderline response, and 4 had negative results.

The resting heart rate (Table 3) and the initial response to passive tilt were identical for positive and negative responders. During isoproterenol infusion the heart rates of patients with negative or borderline responses rose to 130 to 160 beats per minute. In patients with positive tilt responses maximal heart rates prior to presyncope were 76 to 130 beats per minute (with two exceptions of 150 and 160 beats per minute).

Of 25 positive studies (Table 4), the test was terminated during profound presyncope in 13 patients and during full syncope in 12 patients. The vasodepressor reaction became apparent 4 to 9 minutes after start of tilt for those patients who did not receive isoproterenol infusion.

β-Blockade therapy was prescribed for 15 patients who had positive responses to tilting. Repeat tilt studies were done in 6 patients. Five of these had a normal (negative) response. The sixth patient was markedly improved. Before therapy she had often experienced several syncopal episodes per day, but she had none during therapy (10 months); blackout without loss of consciousness occurred occasionally. During therapy she tolerated tilting alone normally. During isoproterenol infusion (2 μg/min) presyncope developed only with sinus bradycardia (62 beats per minute): In none of these patients did isoproterenol infusion achieve heart rates of more than 130 beats per minute.

DISCUSSION

Syncope caused by vasodepressor reaction (neurocardiogenic syncope) is an old problem; its differential diagnosis has been reviewed. Other causes of syncope commonly seen in adult patients (ventricular arrhythmias, atrioventricular block, sick sinus syndrome) or associated diseases (hypertension, myocardial infarction, or diabetes) are virtually absent in childhood. None of our patients had underlying heart disease. Malignant vasovagal syncope resulting in extended asystole is probably a rare exception. Erect position has been used to induce vasodepressor responses but can cause problems when syncope ensues. The head-up tilt, without or with the additional challenge with isoproterenol, has been a simple and effective tool in diagnosing patients who are prone to vasodepressor reactions. We consider it the first step in the workup of an older child with syncope. Once this diagnosis has been confirmed, the patient and family can be reassured that this is not a disease in the usual sense, but a normal albeit paradoxical and extreme physiologic reaction.
TABLE 3. Heart Rates During Tilt Study*

<table>
<thead>
<tr>
<th>Tilt Response Normal or Borderline</th>
<th>Tilt Response Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine (mean ± SD)</td>
<td>68 ± 11</td>
</tr>
<tr>
<td>60° tilt (mean ± SD)</td>
<td>91 ± 11</td>
</tr>
<tr>
<td>Maximal heart rate with isoproterenol (range)</td>
<td>130–160</td>
</tr>
<tr>
<td>Maximal heart rate prior to syncope (range)</td>
<td></td>
</tr>
<tr>
<td>24 patients</td>
<td>76–130</td>
</tr>
<tr>
<td>2 patients</td>
<td>150–160</td>
</tr>
<tr>
<td>Minimal heart rate during syncope (range)</td>
<td>32–86</td>
</tr>
</tbody>
</table>

* Results are given in beats per minute.

TABLE 4. Conditions Inducing Presyncope or Syncope*

<table>
<thead>
<tr>
<th>Tilt Only</th>
<th>Isoproterenol Infusion (µg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Presyncope</td>
<td>6</td>
</tr>
<tr>
<td>Syncope</td>
<td>7</td>
</tr>
</tbody>
</table>

* Results are numbers of patients.

The mechanism of this reaction begins with reduced left ventricular filling, secondary to pooling of blood in the lower parts of the body during tilting. This reduction in left ventricular volume, especially when combined with the inotropic effect of isoproterenol, results in stimulation of left ventricular mechanoreceptors. Increased afferent neural activity causes reflex slowing of heart rate and vasodilation, a paradoxical response seen in those patients who are prone to the vasodepressor response. This hypothetical sequence of events is the basis for treatment of these patients with β blockers (blockade of inotropic and chronotropic effects of catecholamines).

It is striking to observe the sequence of these physiologic reflexes: there is the normal rise in heart rate by 10 to 20 beats per minute in response to tilting, with little change in blood pressure; this level of tachycardia is maintained for several minutes until a precipitous seemingly simultaneous fall in blood pressure and heart rate supervenes. The electrocardiogram shows complete cessation of sinus activity and profound nodal bradycardia. Vasovagal symptoms are evident. Return to the supine position reverses this process promptly. It is equally fascinating to observe the wide range of susceptibility and expression of this vasovagal reaction. Some patients exhibit the full vasovagal response with tilt alone; others have a positive tilt response only with the additional isoproterenol-induced tachycardia; again others show extensive fluctuation in heart rate and blood pressure without a full-fledged vasodepressor reaction. This "borderline" response indicates a transient instability in heart rate control which we consider an incomplete expression of a vasodepressor response.

It is of interest that patients with a history of only presyncope usually had a normal (negative) tilt response. For patients with a history of syncope the demonstration of a positive response, duplicating the child's known symptoms under controlled circumstances, is diagnostic and reassuring for all involved parties. It should be noted that for most patients presyncope became evident at relatively low heart rates, 76 to 130 beats per minute.

THERAPY

Whereas pacemaker implantation may have merit in selected patients, most authors have used β blockade to interrupt the physiologic sequence leading to neurocardiogenic syncope. This approach has been successful. After acute or chronic autonomic blockade, vasodepressor symptoms could no longer be induced or were significantly attenuated.

Whether a "positive tilter" needs to be given medication will depend on the frequency of vasodepressor reactions. For the child who occasionally passes out in church or under similar circumstances, only reassurance is used. The patient, who is usually quite aware of presyncopal symptoms, should lie down rather than fight this reaction with willpower. However, for more frequent episodes, especially when associated with physical exertion, or when there is a history of trauma sustained during syncope, we have used medication as well as reassurance. Seventeen of our patients have had drug therapy. Atenolol (25 or 50 mg every day) is our first choice (12 patients); long-acting propranolol (80 mg every day) was used for 3 patients. For 2 patients, who wish to be very active in sports, we...
opted for 9α-fluorocortisol (0.1 mg every day) and salt supplement as blood volume expander. All patients except 1 have shown complete suppression of syncope. In 1 child, who had neurocardiogenic syncope even while receiving 50 mg of atenolol, we changed the medication to metoprolol (50 mg every day). As this is a preliminary report the duration of drug therapy and the possible difference between β blockers and 9α-fluorocortisol are unanswered questions.

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5. Abi-Samara F, Maloney JD, Fouad-Tarazi FM, Castle LW. The usefulness of head-up tilt testing and hemodynamic investigations in the workup of syncope of unknown origin. *Pace*. 1988;11:1202-1214

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