Haemodynamic effects of increasing angle of head up tilt

A Zaïdi, D Benítez, P A Gaydecki, A Vohra, A P Fitzpatrick

Abstract

Objective—To investigate the haemodynamic effects of varying the angle of head up tilt.

Methods—20 healthy subjects (12 female, eight male; mean (SD) age 33.6 (8.4) years) underwent head up tilt for five minutes to each of four angles of tilt in random order, with a five minute rest period at the horizontal between each angle. Forearm blood flow was measured using intermittent occlusion mercury strain gauge plethysmography at two and five minutes. Subjects underwent continuous monitoring of heart rate, systolic blood pressure (SBP), and diastolic blood pressure (DBP) by Finapres and cardiac output and stroke volume by impedance cardiography. Each variable was measured at two and five minutes, averaged over the period of blood flow measurement.

Results—Every haemodynamic variable at each angle was significantly different from supine values. Head up tilt produced progressive increases in heart rate (11–21%), SBP (12–21%), and DBP (20–33%) with increasing tilt angle. However, although 45° produced significantly less haemodynamic effect, there were no significant differences for angles between 60° and 90°. Cardiac output fell on head up tilt by 17–20% and stroke volume by 28–34%, but increasing tilt angle produced no significant additional reduction in cardiac output and stroke volume because of increases in heart rate and vasoconstriction.

Conclusions—Angles < 60° produce significantly less haemodynamic effects than steeper angles. Increasing tilt angle beyond 60° produces no apparent additional effect on cardiac output or sympathetic tone. Increasing tilt angle beyond 60° confers no additional orthostatic stress and may not affect the sensitivity and specificity of head up tilt testing as previously thought. Sixty degrees of tilt is a more practical angle for support of a syncopal patient and is recommended.

(Heart 2000; 83: 181–184)

Keywords: head up tilt test; vasovagal syncope; thoracic impedance

Head up tilt testing is established as the key investigation in the diagnosis of vasovagal syncope. However, despite the widespread use of tilt testing, there is no agreement on a standard protocol. Various angles and durations of tilt are used, with or without provocative agents, including isoprenaline,1–4 11 and glyceryl trinitrate.5 Most early tilt studies used passive 60° tilt but this required up to 60 minutes of head up tilt to achieve satisfactory sensitivity.7–10 Steeper angles of tilt have been used to reduce the duration of the test,7 4–11 and the perception has grown that steeper levels of tilt confer additional orthostatic stress, increasing peripheral venous pooling and β adrenergic stimulation,12 thereby increasing the sensitivity of tilt testing at the expense of reduced specificity. However, although there have been studies comparing the effects of different tilt angles using the development of syncope or presyncope as the end point,4 13 the haemodynamic effects of increasing the angle of tilt remain unclear. In this study we investigated the haemodynamic effects of varying tilt angle to determine the most appropriate angle for head up tilt testing.

Methods

SUBJECT POPULATION

We studied 20 healthy controls (12 female, eight male; mean (SD) age 33.6 (8.4) years) with no history of syncope or presyncope. All subjects were hospital medical personnel. None of the subjects had a history of cardiovascular disease or was taking any cardioactive drugs. Informed consent was obtained in each case.

TILT TESTING PROTOCOL

Tilt testing took place in quiet, dimly lit room with subjects in a postabsorptive state. Subjects were comfortably restrained on an electric tilt table with foot plate support. Surface electrocardiography (Hewlett-Packard 78351A ECG monitor, Hewlett-Packard Co, Andover, Massachusetts, USA) was used for continuous monitoring of heart rate and rhythm. Beat to beat blood pressure was monitored continuously with a non-invasive finger tip photo-plethysmographic device (Finapres 2300 BP monitor, Ohmeda, Englewood, Colorado, USA). Thoracic impedance was continuously monitored using the BoMed NCCOM3-R7S impedance cardiograph (BoMed Medical Manufacturing Co, Irvine, California, USA), using the technique for non-invasive measurement of cardiac output and stroke volume previously described by Kubicek et al.14 Forearm blood flow was measured by intermittent cuff occlusion mercury-in-Silastic strain gauge plethysmography17 (EC4 plethysmograph, DE Hokanson Inc, Bellevue, Washington, USA).

Analogue signals from each recorder were collected through BNC connectors by a 16 bit data acquisition card (National Instruments PCI-MIO-16XE-50) plugged into the PCI bus.
Table 1  Cardiovascular response to varying angle of head up tilt

<table>
<thead>
<tr>
<th>Tilt angle</th>
<th>HR (beats/min)</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
<th>CO (l/min)</th>
<th>SV (ml)</th>
<th>FBF (%/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>62.2 (8.1)</td>
<td>122.4 (22.6)</td>
<td>69.4 (12.9)</td>
<td>6.4 (1.9)</td>
<td>104.8 (34.4)</td>
<td>2.43 (1.34)</td>
</tr>
<tr>
<td>45°</td>
<td>59.1 (11.3)</td>
<td>137 (23.2)</td>
<td>83.5 (15.7)</td>
<td>5.3 (1.6)</td>
<td>75.8 (20.7)</td>
<td>1.96 (1.22)</td>
</tr>
<tr>
<td>60°</td>
<td>72.7 (11.3)</td>
<td>138.7 (19.5)</td>
<td>88.1 (13.5)</td>
<td>5.3 (1.6)</td>
<td>72.6 (21.4)</td>
<td>1.55 (0.81)</td>
</tr>
<tr>
<td>75°</td>
<td>73.9 (10.6)</td>
<td>141.3 (26.2)</td>
<td>88.2 (17.3)</td>
<td>5.1 (1.6)</td>
<td>68.9 (20.6)</td>
<td>1.56 (0.74)</td>
</tr>
<tr>
<td>90°</td>
<td>75.2 (11.0)</td>
<td>148.6 (25.5)</td>
<td>92 (17.1)</td>
<td>5.2 (1.7)</td>
<td>69.5 (21.9)</td>
<td>1.31 (0.86)</td>
</tr>
</tbody>
</table>

F(4,60) = 34.5, p < 0.001; 21.8, p < 0.001; 50.7, p < 0.001; 12.1, p < 0.001; 35.9, p = 0.001; 8.8, p = 0.001

*Comparison between supine and different angles of tilt made separately for each haemodynamic variable using single factor repeated measures analysis of variance.

CO, cardiac output; DBP, diastolic blood pressure; FBF, forearm blood flow; HR, heart rate; SBP, systolic blood pressure; SV, stroke volume.

Table 2  Change in haemodynamics from supine to each angle of tilt

<table>
<thead>
<tr>
<th>Tilt angle</th>
<th>% HR</th>
<th>% SBP</th>
<th>% DBP</th>
<th>% CO</th>
<th>% SV</th>
<th>% FBF</th>
</tr>
</thead>
<tbody>
<tr>
<td>45°</td>
<td>11.1</td>
<td>11.9</td>
<td>20.3</td>
<td>−17.2</td>
<td>−27.7</td>
<td>−19.3</td>
</tr>
<tr>
<td>60°</td>
<td>16.9</td>
<td>13.3</td>
<td>26.9</td>
<td>−17.2</td>
<td>−30.7</td>
<td>−36.2</td>
</tr>
<tr>
<td>75°</td>
<td>18.8</td>
<td>15.4</td>
<td>27.1</td>
<td>−20.3</td>
<td>−34.3</td>
<td>−35.8</td>
</tr>
<tr>
<td>90°</td>
<td>20.9</td>
<td>21.4†</td>
<td>32.6†</td>
<td>−18.8</td>
<td>−33.7</td>
<td>−46.1†</td>
</tr>
</tbody>
</table>

*Significant difference from 45°; †significant difference from 60° (p < 0.05).

% CO, percentage change in cardiac output after tilting; % DBP, percentage change in diastolic blood pressure after tilting; % FBF, percentage change in forearm blood flow after tilting; % HR, percentage change in heart rate after tilting; % SBP, percentage change in systolic blood pressure after tilting; % SV, percentage change in stroke volume after tilting.

Discussion

HAEMODYNAMIC EFFECTS AT ANGLES BELOW 60°

The ACC expert consensus document on tilt table testing for assessing syncope concluded that angles below 60° provide insufficient orthostatic stress and result in a lower yield of positives in patients with syncope.18 There is compelling evidence to support this view. Haemodynamic studies in healthy controls have shown no significant haemodynamic effect at angles below 30°.19 At angles between 30° and 60° there was a linear response in cardiac output, diastolic blood pressure, and heart rate, with no significant change in systolic blood pressure.16 17 while at angles beyond 60°, there was no significant change in cardiac output.13 This is reflected in the apparent loss of sensitivity in tilt testing at angles below 60°. Fitzpatrick et al reported that passive tilting to 45° reproduced syncope in only three of 10 patients with positive tests at 60°20 while Sanden-Jensen et al showed that 30° tilt failed to reproduce syncope in healthy controls with typical vasovagal reactions at 60° using a saddle support.21

CLINICALLY USEFUL TILT ANGLES

For angles between 60° and 80°, the conclusion of the ACC expert consensus document was that there was no substantial difference in terms of test outcome in the absence of pharmacological provocation, but that there was a higher positive yield at steeper tilt angles with isoprenaline provocation. This implies that tilt angles beyond 60° confer additional orthostatic stress, but the evidence for this is circumstantial. In general, series of head up tilt testing have shown a higher yield with 80° tilt than with 60° tilt.14 15 However, positive
rates of 75% have been achieved using passive 60° tilt for 60 minutes, which compares favourably with most studies using 80°, and it seems probable that patient selection rather than additional orthostatic stress accounts for much of the apparent superiority of 80° tilt. Furthermore, the majority of series at 80° have used provocative agents, which clearly increase the positive yield of tilt testing.1–11 There are no satisfactory studies comparing the haemodynamic effects of head-up tilting at 60° and 80°. However, Voice et al compared the susceptibility to syncope at the two most widely used angles for clinical tilt testing by randomising patients with two or more episodes of unexplained syncope to 45 minute passive tilt at 60° or 80°. The positive yield was higher at 80° (54% compared with 42% at 60°), but neither the positive rate nor the time to syncope were significantly different and it was concluded that there was probably little difference between the two angles.

ISOPRENALINE STUDIES

The best evidence for the superiority of 80° comes from studies of head-up tilt in healthy controls, particularly with isoprenaline provocation. Natale et al randomised healthy controls to tilt at 60°, 70°, or 80° (20 minutes passive 50% followed by tilt with isoprenaline infusion to raise heart rate by 20%). During baseline tilt, there was reduced specificity with increased tilt angle (positive rate 8% at 60°, 8% at 70°, 20% at 80°) but the difference in the positive rate between the different tilt angles was not significant. Isoprenaline infusion produced similar results (positive rate 4% at 60°, 4% at 70°, 28% at 80°), with significant differences between 80° and the two lower angles, albeit with small numbers of false positives. However, the specificity of 80° tilt with isoprenaline has been shown to be as high as 92% in other studies.22 Similarly, Kapoor et al pooled the results of 14 tilt test series using isoprenaline at various angles between 60° and 90° and showed that the proportion of positive tests rose progressively with increasing tilt angle, suggesting that orthostatic stress increases with tilt angle.23 However, the data for 60° and 90° were from single small studies and the lack of a uniform protocol for patient selection and investigation raises concern about patient selection bias.

PATHOPHYSIOLOGY OF HEAD UP TILT

The pathophysiology of tilt induced syncope remains incompletely understood but it is thought that the primary effect of orthostasis is the redistribution of up to 1000 ml of blood from the central cardiovascular compartment to the capacitance vessels of the lower limbs. This leads to a progressive fall in venous return, which may be excessive in patients predisposed to vasovagal syncope, resulting in inappropriate activation of left ventricular mechanoceptors and paradoxical reflex sympathetic withdrawal.1 It has been assumed that by increasing the tilt angle the degree of gravity mediated displacement of the circulating volume to the peripheries is also increased. However, the redistribution of blood is directly related to the sine of the angle of tilt,24 which rises in an approximately linear fashion between 30° and 60° but changes very little at angles beyond 60° (sin 60° = 0.87, sin 80° = 0.98). This is in keeping with previous haemodynamic studies at angles below 60° and with our present results, and suggests that there is a “plateauing” of haemodynamic effects at 60°, rendering higher angles of tilt unnecessary. The haemodynamic change seen in our control subjects is similar to previous studies using passive 90° tilt in controls, where stroke volume fell by approximately 28% on tilt with a 30% increase in peripheral vascular resistance.25

CONCLUSIONS

Sixty degrees of tilt is the most practical angle for supporting syncopal patients. Increasing tilt angle beyond 60° produces no additional orthostatic stress. This suggests that passive tilt at 60° for up to 60 minutes is the optimum protocol for achieving the combination of high specificity with low sensitivity in head-up tilt testing.

Aortic valve tumour trapped in the right coronary ostium

A 48-year-old man with a history of rectocolitis was admitted to a coronary care unit because of an inferior myocardial infarction. Revascularisation was not performed because of the absence of pain at the time of admission and delay (six hours) after the onset of thoracic pain. The patient had no risk factors for atherosclerosis apart from smoking, which he had stopped two years ago.

Transthoracic cross-sectional echocardiography revealed an abnormal structure behind the right coronary aortic cusp. This was confirmed by multiplane transoesophageal echocardiography, which showed a pedunculated tumour overhanging the right coronary ostium. Because of the risk of embolisation, coronary angiography was not performed and surgical removal of the tumour was attempted. At surgery, behind the right coronary cusp, only remnants of the tumour were found and excised. Peroperative videoscopy detected the tumour trapped in the right coronary ostium. The tumour (7 mm diameter) was extracted with a Fogarty catheter. Histopathology showed a fibroma with areas of attached thrombus. One month after surgery, coronary angiography was performed, which showed single vessel disease with a 70% distal stenosis of the right coronary artery. Because of inferior akinesia of the left ventricle and negative stress test, coronary revascularisation was not attempted.

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