Effects of Midodrine Hydrochloride on Blood Pressure and Cerebral Blood Flow During Orthostasis in Persons With Chronic Tetraplegia

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Objective: To determine the mean arterial pressure (MAP) and middle cerebral artery mean blood flow velocity (MFV) responses to 5 and 10mg midodrine during head-up tilt (HUT) in persons with tetraplegia.

Design: Prospective dose-response trial.

Setting: James J. Peters Veterans Administration Medical Center.

Participants: Persons (N=10) with chronic tetraplegia (duration of injury=23±11y).

Intervention: A dose titration study was performed over 3 testing days: control (no drug), 5mg midodrine (5mg), or 10mg midodrine (10mg) during 30 minutes of baseline (predrug/no drug), 30 minutes of supine rest postdrug/no drug, 15 minutes of progressive HUT (5 minutes at 15°, 25°, 35°), and 45 minutes of 45° HUT.

Main Outcome Measures: MAP and MFV response to midodrine supine and during HUT.

Results: Ten milligrams of midodrine significantly increased MAP while supine and during the HUT maneuver. Of note, the mean increase in MAP during HUT with 10mg was a result of a robust effect in 2 persons, with minimal change in the remaining 8 study subjects. The reduction in cerebral MFV during HUT was attenuated with 10mg.

Conclusions: These findings suggest that midodrine 10mg may be efficacious for treatment of hypotension and orthostatic hypotension in select persons with tetraplegia. Although midodrine is routinely prescribed to treat orthostatic hypotension, the results of our work suggest limited efficacy of this agent, but additional studies in a larger sample of subjects with spinal cord injury should be performed.

Key Words: Hypotension, orthostatic; Midodrine; Rehabilitation; Spinal cord injuries; Tilt-table test.

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IN PERSONS WITH SPINAL cord injury, in addition to motor and sensory deficits, partial or complete interruption of autonomic cardiovascular innervation results in dysregulation of blood pressure. Although the etiology may vary, the prevailing thought is that blood pressure disorders in persons with tetraplegia derive from decentralized sympathetic neural cardiovascular control, and significantly reduced plasma norepinephrine levels have been reported during HUT.1-3 As a consequence of impaired sympathetic cardiovascular innervation, individuals with tetraplegia are prone to chronic hypotension with exacerbations during periods of orthostasis.4-8 It is well established that OH hinders the rehabilitation process during the acute and subacute phases of SCI.9-11 but may also hamper the resumption of independence and functional activities in persons with chronic SCI.5,10 We recently reported significantly reduced memory and marginally reduced attention and processing speed and executive function in hypertensive persons with chronic SCI compared with normotensive counterparts,12 as previously reported in the non-SCI population.13,14 Thus, chronic hypotension and OH might be expected to limit significantly the quality of life in persons with SCI, and treatment options should be considered.

Although many persons with tetraplegia are hypertensive, they often remain clinically asymptomatic; and, for this reason, treatment strategies are not routinely considered as part of clinical care. In fact, there is a striking disparity between the available treatment options for hypertension compared with hypotension in the general population: hypertension has 119 U.S. Food and Drug Administration–approved medications, and hypotension just 1, midodrine hydrochloride. Of note, the safety and efficacy of midodrine for the treatment of chronic hypotension and OH has not been formally tested in the SCI population. The results of several case reports on the use of midodrine to treat OH in person’s with SCI suggest improved blood pressure and reduced symptoms of cerebral hypoper-
fusione (ie, dizziness, fatigue, blurred vision, syncope, lightheadedness). Reduction in the cerebral symptoms of hypotension should be reflected in preserved CBF during orthostasis after midodrine administration, although this has not been reported in the SCI population.

The objective of this study was to determine the dose-response for blood pressure and CBF after midodrine administration while supine and during a HUT maneuver in persons with chronic tetraplegia. We hypothesized that systemic blood pressure would be increased in a dose-response manner after midodrine administration, and the increase in systemic blood pressure would be associated with a dose-response increase in CBF.

METHODS

Subjects
Ten subjects with chronic tetraplegia volunteered to participate; the demographics of the study group are presented (table 1). No study subject had a history of cardiovascular disease, none were prescribed medications with known cardiovascular or autonomic effects, and none were current smokers. All study participants were neurologically stable for at least 9 years postinjury and were recruited from the Center of Excellence for the Medical Consequences of Spinal Cord Injury at the James J. Peters Veterans Affairs Medical Center. The study protocol was approved by the local institutional review board with strict adherence to the standards established in the Helsinki Declaration. Written informed consent was obtained before performing the study procedures.

Study Procedures
The study time line is presented (fig 1); 10 subjects with tetraplegia underwent 3 separate days of testing: visit 1, no drug (control); visit 2, midodrine 5mg; visit 3, midodrine 10mg. On arrival at the laboratory, between 8:00 and 10:00 AM, subjects were transferred to the tilt table in the supine position for a minimum of 20 minutes for instrumentation. Three ECG electrodes were applied to the chest for continuous heart rate monitoring. Blood pressure was measured at the brachial artery by a trained clinician by using a standard adult blood pressure cuff placed around the left upper arm. CBF was estimated from MFV of the left MCA by using TCD ultrasound technology. After instrumentation, baseline data were collected for heart rate, blood pressure, and MFV at 0 and 20 minutes. Midodrine hydrochloride 5 or 10mg was administered orally with a glass of water 30 minutes into testing; subjects were not given any medication on the control visit, and additional supine data were collected at 35, 45, and 55 minutes prior to the tilt maneuver. Because the blood pressure effects of midodrine are generally demonstrated within minutes and peak effects are expected within an hour of administration, the progressive HUT maneuver began 30 minutes after midodrine administration, and subjects remained in the orthostatic position for a total of 60 minutes thereafter (ie, 90min after midodrine administration). The tilt table was padded and motorized; restraining straps were used on the lower extremities and trunk to ensure subject safety and were padded to avoid stimulation of sympathetic spinal reflexes during testing. Adjustment of the tilt table to the desired angle was accomplished in less than 5 seconds, and the progressive HUT maneuver consisted of 5 minutes at each intermediate angle of tilt (15°, 25°, 35°) and 45 minutes at 45°. A progressive HUT maneuver was used to allow adequate time for activation of the renin-angiotensin system, the predominant mechanism used for blood pressure control in individuals with tetraplegia. Subjects were questioned at 10-minute intervals for the presence of the symptoms of hypotension and cerebral hypoperfusion (ie, dizziness, fatigue, blurred vision, syncope, lightheadedness).

Heart rate was monitored continuously with an ECG signal recorded from a 3-lead configuration. Electrodes were placed at the distal right and left clavicle, and the recording electrode was placed in the left lateral fifth intercostal space (V-5) by using standard skin-abrading and hair-shaving methods as needed. Manual blood pressure was measured by auscultation at the brachial artery and was recorded by a trained clinician twice at baseline, 3 times postdrug while the subject was in the supine position, and once at each angle of progressive HUT, and 4 measurements were taken at 15-minute intervals while

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex</th>
<th>Age (y)</th>
<th>HT (cm)</th>
<th>WT (kg)</th>
<th>BMI</th>
<th>DOI (y)</th>
<th>Level</th>
<th>AIS</th>
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<tr>
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<td>66</td>
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<td>2</td>
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<td>180</td>
<td>66</td>
<td>20.2</td>
<td>30</td>
<td>C4-5</td>
<td>ASIA grade C</td>
</tr>
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<td>3</td>
<td>Man</td>
<td>36</td>
<td>165</td>
<td>70</td>
<td>25.8</td>
<td>10</td>
<td>C5-6</td>
<td>ASIA grade A</td>
</tr>
<tr>
<td>4</td>
<td>Woman</td>
<td>55</td>
<td>168</td>
<td>62</td>
<td>22.1</td>
<td>39</td>
<td>C7</td>
<td>ASIA grade B</td>
</tr>
<tr>
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<td>Man</td>
<td>38</td>
<td>188</td>
<td>77</td>
<td>21.8</td>
<td>13</td>
<td>C4-5</td>
<td>ASIA grade A</td>
</tr>
<tr>
<td>6</td>
<td>Man</td>
<td>47</td>
<td>180</td>
<td>82</td>
<td>25.1</td>
<td>23</td>
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<td>ASIA grade B</td>
</tr>
<tr>
<td>7</td>
<td>Man</td>
<td>42</td>
<td>165</td>
<td>84</td>
<td>30.8</td>
<td>13</td>
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<td>ASIA grade A</td>
</tr>
<tr>
<td>8</td>
<td>Man</td>
<td>43</td>
<td>163</td>
<td>59</td>
<td>22.1</td>
<td>26</td>
<td>C5-6</td>
<td>ASIA grade A</td>
</tr>
<tr>
<td>9</td>
<td>Woman</td>
<td>35</td>
<td>173</td>
<td>51</td>
<td>17.2</td>
<td>17</td>
<td>C5-6</td>
<td>ASIA grade A</td>
</tr>
<tr>
<td>10</td>
<td>Man</td>
<td>57</td>
<td>183</td>
<td>62</td>
<td>18.4</td>
<td>39</td>
<td>C5-7</td>
<td>ASIA grade B</td>
</tr>
</tbody>
</table>

Abbreviations: AIS, American Spinal Injury Association Impairment Scale; ASIA, American Spinal Injury Association; HT, height; WT, weight; BMI, body mass index; DOI, duration of injury; Level, level of SCI.
the subject was at 45°. MAP (mmHg) was calculated from brachial pressures as follows:

\[
\frac{\text{systolic} + (2 \times \text{diastolic})}{3}
\]

To determine MFV, a TCD probe was operated at a frequency of 2.0MHz to visualize the left MCA, and insonation was through the temporal window. The MCA was identified by the target depth (45-55mm), sound and direction of flow (i.e., toward the probe), the characteristic spectral waveform, relatively faster flow velocity compared with other cerebral vessels, and compression of the common carotid artery, which resulted in an appropriate reduction in MCA flow velocity. Once the MCA was visualized, a head harness was used to secure the probe position for the duration of testing, and MFV (cm/s) was calculated as follows:

\[
\frac{\text{peak systolic velocity} + (2 \times \text{end diastolic velocity})}{3}
\]

MCA MFV is an accepted surrogate of CBF, which has been validated and reported by several investigators.\textsuperscript{19-21} TCD signals were channeled and stored on a hard drive for future analysis by using customized data acquisition and analysis programs written with LabVIEW graphic software for instrumentation.\textsuperscript{4}

**Data Analysis**

Data are reported as mean \(\pm\) SD. Repeated-measures analysis of variance was used to determine within-subject differences in mean heart rate, blood pressure, and MFV responses to no drug, 5mg, and 10mg midodrine. A repeated-measures analysis of variance was used to determine significant dose (no drug, 5mg, 10mg), condition (baseline, postdrug, transition, HUT), and interaction effects for heart rate, blood pressure, and MFV. Fisher post hoc analyses were used to explore significant omnibus effects further. Significance was set at the .05 alpha level.

**RESULTS**

Demographic characteristics of the study group are presented (see table 1). Subjects were between 27 and 57 years of age and a minimum of 9 years postinjury, with levels of lesions between C4 and C7; 9 were motor-complete American Spinal Injury Association Impairment Scale grades A and B; all were nonambulatory. Although 1 individual (patient 3) developed significant symptoms of syncope that led to early termination of the HUT maneuver on the no drug visit, and there was a trend toward reduced orthostatic symptoms after midodrine administration, in general, OH symptoms reporting did not differ significantly among the study visits (table 2).

The MAP, heart rate, and MFV response to study visit and condition are presented (fig 2). There was a significant main effect for condition and interaction effects for MAP, heart rate, and MFV. In general, heart rate was increased and MAP and MFV tended to be reduced during the HUT maneuver regardless of the study visit. MAP was increased during the supine postdrug period and during the transition with 10mg midodrine compared with the control visit \((P<.001)\), and the interaction effect was significant for MAP \((P<.01)\) and MFV \((P<.05)\). The main effect for study visit was not significant for MAP, heart rate, or MFV. NOTE. The letters a, b, c, d are 4 time points of data collection at 45° HUT. Abbreviation: bl, baseline.

Table 2: Symptoms Reporting During HUT

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No Drug</th>
<th>5mg</th>
<th>10mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleepy</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Nausea</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Lightheadedness</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Yawning</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Fatigue</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pallor</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Dizziness</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Heated</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Blurry vision</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neck pain</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>8</td>
<td>2</td>
</tr>
</tbody>
</table>

Fig 2. The response to study visit: control (open circles), 5mg (closed triangles) and 10mg (closed squares), and condition (bl, postdrug, transition, HUT) for MAP (A, mmHg), heart rate (B, bpm) and MFV (C, cm/s). There were significant condition main effects for MAP, heart rate, and MFV \((P<.0001)\), and the interaction effect was significant for MAP \((P<.01)\) and MFV \((P<.05)\). The main effect for study visit was not significant for MAP, heart rate, or MFV. NOTE. The letters a, b, c, d are 4 time points of data collection at 45° HUT. Abbreviation: bl, baseline.
Mean data for the heart rate, blood pressure, and MFV response to dose and condition are presented (Table 3). There were no differences for baseline supine heart rate, blood pressure, or MFV. Supine diastolic blood pressure and MAP were increased after 10mg midodrine compared with the control visit ($P < .05$). During the 45° HUT maneuver, systolic blood pressure and MAP were increased after 10mg midodrine compared with the control visit ($P < .05$). There were no significant effects for heart rate, blood pressure, or MFV when comparing 5mg midodrine and the control visit, or between the 5-mg and 10-mg doses of midodrine.

The mean change from baseline in heart rate, MAP, and MFV is presented (Fig 3). On average, the change in MAP was increased after 10mg midodrine during the supine postdrug period and during the sustained HUT maneuver compared with the control visit ($P < .05$). There were no differences among study visits for the change in heart rate. However, 10mg midodrine attenuated the fall in MFV during HUT compared with the control visit ($P = .05$).

Although, on average, MAP was increased after the 10-mg dose of midodrine during supine rest and HUT compared with the control visit, individual responses varied (Fig 4). During the supine period, 10mg midodrine (see Fig 4A) seemed to increase MAP in most subjects compared with their control visit; during the HUT maneuver, however, the data are less convincing (see Fig 4B). It is apparent that 2 individuals had an augmented response to 10mg midodrine during HUT (subjects 5 [closed triangle] and 9 [+] ). Also, 1 subject appeared to respond better to the 5-mg dose than to the 10-mg dose of midodrine (subject 1 [closed square]), and 1 subject had the highest MAP response to HUT during the control visit (subject 11 [open circle]).

**DISCUSSION**

Individuals with tetraplegia are a model of decentralized sympathetic cardiovascular control, which may result in chronic hypotension with exacerbations during periods of orthostasis. The extent of autonomic impairment and the cardiovascular consequences may or may not relate to the level of SCI. Although midodrine is the most commonly prescribed pharmacologic agent for treatment of OH in this population, convincing scientific data are lacking. This is the first systematic investigation to document the effects of midodrine hydrochloride on systemic blood pressure during orthostasis in individuals with tetraplegia. The results suggest that, compared with the control visit, the 5-mg dose of midodrine had little effect on increasing MAP during a HUT maneuver. Although the 10-mg dose of midodrine increased MAP during HUT compared with the control HUT maneuver, individual responses varied considerably and do not support the general use of this medication for treatment of OH in this population.

Several case reports have been published on the effects of midodrine to increase orthostatic blood pressure and reduce symptoms of OH in individuals with SCI. The results of these case studies suggest that midodrine is safe and efficacious

### Table 3: Mean Data at Supine, Postdrug, and 45° HUT

<table>
<thead>
<tr>
<th></th>
<th>No Drug</th>
<th>5mg</th>
<th>10mg</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Heart rate (bpm)</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Supine</td>
<td>59±13</td>
<td>57±8</td>
<td>62±13</td>
<td>NS</td>
</tr>
<tr>
<td>Postdrug</td>
<td>60±14</td>
<td>57±10</td>
<td>59±11</td>
<td>NS</td>
</tr>
<tr>
<td>HUT</td>
<td>70±15</td>
<td>64±9</td>
<td>67±11</td>
<td>NS</td>
</tr>
<tr>
<td><strong>SBP (mmHg)</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td>98±5</td>
<td>97±5</td>
<td>102±12</td>
<td>NS</td>
</tr>
<tr>
<td>Postdrug</td>
<td>102±11</td>
<td>102±11</td>
<td>107±15</td>
<td>NS</td>
</tr>
<tr>
<td>HUT</td>
<td>87±15</td>
<td>97±17</td>
<td>109±22</td>
<td>&lt; .05</td>
</tr>
<tr>
<td><strong>DBP (mmHg)</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td>64±6</td>
<td>61±8</td>
<td>63±7</td>
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<tr>
<td>Postdrug</td>
<td>64±9</td>
<td>66±10</td>
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<tr>
<td>HUT</td>
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<td>64±7</td>
<td>72±17</td>
<td>NS</td>
</tr>
<tr>
<td><strong>MAP (mmHg)</strong></td>
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<tr>
<td>Postdrug</td>
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<td>82±8</td>
<td>NS</td>
</tr>
<tr>
<td>HUT</td>
<td>70±13</td>
<td>73±9</td>
<td>84±17</td>
<td>&lt; .05</td>
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<tr>
<td><strong>MCA MFV (cm/s)</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
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<td>Supine</td>
<td>55±12</td>
<td>52±10</td>
<td>55±15</td>
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</tr>
<tr>
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<td>HUT</td>
<td>48±8</td>
<td>45±10</td>
<td>52±13</td>
<td>NS</td>
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</table>

NOTE. Data are mean ± SD.

Abbreviations: DBP, diastolic blood pressure; MCA MFV, middle cerebral artery mean flow velocity; NS, not significant; SBP, systolic blood pressure.

*Versus no drug.
for use in individuals with SCI to treat OH associated with rehabilitation activities during the acute period after injury. In the only placebo-controlled randomized trial, 10mg midodrine increased systolic blood pressure during maximal exercise in 3 individuals with chronic tetraplegia; 1 individual did not respond to the medication. Based on these limited data, however, several review articles suggest that midodrine should be considered for use in individuals with SCI for treatment of OH. Our data indicate that midodrine may have utility for the management of OH in select patients with SCI. However, the effects of midodrine on supine, seated, and upright blood pressure should be determined on an individual basis in persons with SCI to document the therapeutic effect prior to prescribing this agent.

Midodrine is an alpha-1 agonist that exerts effects via activation of the alpha-adrenergic receptors on the vascular walls, thereby increasing vessel tone and blood pressure. Midodrine is the only U.S. Food and Drug Administration–approved medication for the treatment of OH, and its efficacy has been documented in conditions of central and peripheral autonomic impairment. There is evidence to suggest that midodrine elicits a larger blood pressure effect in conditions of postganglionic (ie, multiple system atrophy) disorders compared with preganglionic (ie, pure autonomic failure, etc.) disorders. The utility in persons with SCI, a preganglionic sympathetic disorder, is therefore uncertain, although there is evidence of increased sensitivity to an alpha-1 agonist in 1 individual with SCI compared with 6 controls without SCI. As such, alpha-1 agonists may have clinical utility for treatment of hypotension and OH in the SCI population. It should be appreciated that, because of alpha-1 receptor hyperresponsiveness, blood pressure elevations during AD may be exacerbated with midodrine administration in persons with high cord lesions. Although several reports have documented the utility of midodrine to increase the likelihood of ejaculation during sexual stimulation in persons with SCI, the observed increase in blood pressure with midodrine administration and ejaculation was not augmented compared with ejaculation alone. In contrast, AD associated with bladder distension was reported in 2 individuals with tetraplegia due to increased sphincter tone after midodrine administration for the treatment of OH. Because AD is often unpredictable and the blood pressure elevations may be severe and life-threatening, caution and close clinical observation should be maintained when prescribing midodrine for the treatment of OH in individuals with SCI.

Compromise in cerebral blood flow is presumed to be responsible for the clinical symptoms of hypotension (ie, dizziness, fatigue, blurred vision, syncope, lightheadedness), and there is documentation of reductions in these symptoms after treatment with midodrine in individuals with neurogenic OH and SCI. Midodrine reportedly does not cross the blood-brain barrier; however, direct evidence of the effects of midodrine on CBF is limited. Significantly reduced MCA MFV has been reported in persons with chronic hypotension, and improvement in CBF was documented after acute administration of midodrine. In contrast, a 4-week treatment with midodrine (4mg/d) did not significantly improve CBF during HUT in hemodialysis patients with OH. The present data suggest that the reduction in MCA MFV during HUT was attenuated with 10mg midodrine compared with the control HUT, which was most likely related to increased systemic blood pressure. However, because only 1 subject developed significant symptoms of cerebral hypoperfusion during the control HUT maneuver, we were unable to determine improvement in the clinical symptoms of OH after midodrine administration.

An increased prevalence of OH has been reported in the SCI population, and the diagnosis of OH has been as high as 74% during orthostatic maneuvers and mobilization in the acute rehabilitation setting. It is also well established that the risk of developing OH during orthostatic maneuvers is increased in high cord lesions. The mechanisms responsible for OH in persons with SCI have been investigated and include loss of descending supraspinal sympathetic control associated with low circulating plasma norepinephrine and epinephrine levels, morphologic changes within the sympathetic nervous system, increased nitric oxide production in association with vascular vasodilatation. The etiology of OH in persons with SCI is multifactorial, and treatment approaches should reflect the variety of pathologic mechanisms. Prior to prescribing midodrine for treatment of hypotension and OH in patients with SCI, nonpharmacologic remedies should be considered. These include using abdominal and leg binders, increasing daily salt and water intake, raising the head of the bed 10° to 20° at nighttime, and minimizing daily intake of diuretics such as caffeine and alcohol.

Study Limitations

Because of safety concerns, our study was designed as a dose-response trial of 5mg and 10mg doses of midodrine compared with a control HUT maneuver. A matching placebo tablet was not used and the order of testing was not randomized, which are limitations to data interpretation. In addition, controls without SCI were not recruited, and, therefore, the blood pressure and MFV responses during HUT after mido-

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For further analysis, please refer to the original article in the Arch Phys Med Rehabil Vol 91, September 2010.
midodrine in the SCI population cannot be compared with a response of a control without SCI. The total number of subjects recruited was relatively small, and extrapolation to a broader more heterogeneous group of individuals with chronic SCI may not be appropriate. Furthermore, because the blood pressure responses varied among this small cohort of subjects, these preliminary results do not support the utility of midodrine for treatment of OH in persons with SCI. Finally, caution should be exercised prior to prescribing midodrine to individuals with SCI who experience frequent bouts of AD.

CONCLUSIONS

To our knowledge, this study is the first systematic report on the efficacy of midodrine for treatment of OH in individuals with SCI. The findings suggest that mean blood pressure was increased during HUT after 10mg midodrine compared with the control HUT maneuver; the 5-mg dose of midodrine did not significantly increase orthostatic blood pressure. The significant increase in MAP during HUT was largely a result of an augmented response in 2 subjects; the other 8 individuals demonstrated a change in MAP during tilt that was similar to the study visit without medication. Furthermore, an augmented MAP response to 5mg compared with 10mg midodrine was observed in 1 subject. Thus, because of the variability in response to midodrine and the heterogeneous etiology of hypotension in the SCI population, medications with different mechanisms of action should be considered and developed for use in the treatment of hypotension and OH in this population.

References


Suppliers
a. Ivy Biomedical Systems Inc, 11 Business Park Dr, Branford, CT 06405-2959.
b. GE Healthcare Information Technologies, 8200 W Tower Ave, Milwaukee, WI 53223-3219.
c. Terumo Cardiovascular Systems, 1311 Valencia Ave, Tustin, CA 92780-6447.
d. National Instruments, 11500 N Mopac Expy, Austin, TX 78759-3504.