Association Between Supine Hypertension and Orthostatic Hypotension in Autonomic Failure

David S. Goldstein, Sandra Pechnik, Courtney Holmes, Basil Eldadah, Yehonatan Sharabi

Abstract—Supine hypertension occurs commonly in primary chronic autonomic failure. This study explored whether supine hypertension in this setting is associated with orthostatic hypotension (OH), and if so, what mechanisms might underlie this association. Supine and upright blood pressures, hemodynamic responses to the Valsalva maneuver, baroreflex-cardiovagal gain, and plasma norepinephrine (NE) levels were measured in pure autonomic failure (PAF), multiple-system atrophy (MSA) with or without OH, and Parkinson’s disease (PD) with or without OH. Controls included age-matched, healthy volunteers and patients with essential hypertension or those referred for dysautonomia. Baroreflex-cardiovagal gain was calculated from the relation between the interbeat interval and systolic pressure during the Valsalva maneuver. PAF, MSA with OH, and PD with OH all featured supine hypertension, which was equivalent in severity to that in essential hypertension, regardless of fludrocortisone treatment. Among patients with PD or MSA, those with OH had higher mean arterial pressure during supine rest (109±3 mm Hg) than did those lacking OH (96±3 mm Hg, P=0.002). Baroreflex-cardiovagal gain and orthostatic increments in plasma NE levels were markedly decreased in all 3 groups with OH. Among patients with PD or MSA, those with OH had much lower mean baroreflex-cardiovagal gain (0.74±0.10 ms/mm Hg) than did those lacking OH (3.13±0.72 ms/mm Hg, P=0.0002). In PAF, supine hypertension is linked to both OH and low baroreflex-cardiovagal gain. The finding of lower plasma NE levels in patients with than without supine hypertension suggests involvement of pressor mechanisms independent of the sympathetic nervous system. (Hypertension. 2003;42:136-142.)

Key Words: hypotension, essential hypertension, Parkinson’s disease, autonomic nervous system, sympathetic nervous system, norepinephrine

Primary chronic autonomic failure has been classified clinically into 3 forms: pure autonomic failure (PAF), multiple-system atrophy (MSA), and autonomic failure in the setting of Parkinson’s disease (PD).1 All 3 forms typically include orthostatic hypotension (OH), wherein reflexive increases in sympathetic neurocirculatory tone fail to compensate adequately for decreased venous return to the heart.

Patients with primary chronic autonomic failure also often have supine hypertension.2 Because of widespread use of the salt-retaining steroid fludrocortisone to treat OH and literature documenting increases in blood pressure secondary to mineralocorticoid administration,3,4 supine hypertension in primary chronic autonomic failure might be a side effect of treating the OH and not part of the disease; however, supine hypertension has been reported in a substantial proportion of untreated patients.5 Analogously, levodopa is a mainstay in the treatment of PD, and based on literature that levodopa produces OH,6 OH in PD might be a side effect of treating the movement disorder and not part of the disease; however, OH occurs in at least some patients with PD who are off or have never been treated with levodopa.7

Recent studies therefore have supported the alternative views that supine hypertension does indeed reflect part of the pathophysiology of primary chronic autonomic failure and that OH does indeed reflect part of the pathophysiology of PD. In MSA with OH (formerly called the Shy-Drager syndrome),8,9 administration of trimethaphan, which inhibits ganglionic neurotransmission, evokes marked decreases in supine blood pressure, and administration of the α2-adrenoceptor antagonist yohimbine, which releases norepinephrine (NE) from sympathetic nerve terminals, evokes marked increases in supine blood pressure.2,10 These findings fit with the notion that in MSA, supine hypertension reflects a state of increased postganglionic sympathetic neurovascular tone. Meanwhile, at least a dozen recent studies have agreed on the remarkable finding that all patients with PD and OH have diffuse loss of sympathetic innervation throughout the left ventricular myocardium; more generalized loss of sympathetic noradrenergic nerves could easily cause or contribute to OH.7

This correlational, retrospective study addressed whether, in chronic autonomic failure, supine hypertension attends OH, and if so, what the bases for this relationship might be. We asked the following questions: (1) In PAF, MSA, or PD, is there an association between supine hypertension and OH?
Subject Characteristics

<table>
<thead>
<tr>
<th>Group</th>
<th>Gender, M/F</th>
<th>Age, y</th>
<th>Body Mass, kg</th>
<th>Levodopa</th>
<th>Fludrocortisone</th>
<th>Both</th>
<th>Neither</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAF</td>
<td>7/4</td>
<td>59±4</td>
<td>71±7</td>
<td>0</td>
<td>10</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>PD + OH</td>
<td>16/8</td>
<td>66±2</td>
<td>74±4</td>
<td>11</td>
<td>9</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>PD no OH</td>
<td>24/3</td>
<td>62±2</td>
<td>79±3</td>
<td>21</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>MSA + OH</td>
<td>15/11</td>
<td>61±2</td>
<td>79±3</td>
<td>10</td>
<td>13</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>MSA no OH</td>
<td>5/3</td>
<td>64±3</td>
<td>74±3</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Normal</td>
<td>22/9</td>
<td>60±1</td>
<td>78±3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>31</td>
</tr>
<tr>
<td>COI</td>
<td>5/12</td>
<td>54±1</td>
<td>71±3</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>EH</td>
<td>13/19</td>
<td>60±2</td>
<td>84±6</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>32</td>
</tr>
</tbody>
</table>

PAF indicates pure autonomic failure; PD + OH, Parkinson’s disease with orthostatic hypotension; PD no OH, PD without OH; MSA + OH, multiple-system atrophy with OH; MSA no OH, MSA without OH; COI, chronic orthostatic intolerance; and EH, essential hypertension.

(2) If so, does fludrocortisone treatment explain this association? (3) Do patients with OH have disruption of baroreflexes, as measured by low baroreflex-cardiovagal gain? and (4) If so, is there a link between the extent of baroreflex disruption and magnitude of supine hypertension? Regarding PD, we asked: (1) Does OH in PD result from levodopa treatment? (2) Is sympathetic denervation linked to baroreflex disruption? and (3) Might OH in PD require combined sympathetic denervation and baroreflex disruption? Finally, in both PD and MSA, we asked whether patients with supine hypertension had higher plasma NE levels during supine rest than did those without supine hypertension.

Methods

All subjects gave informed, written consent before participating in the study, which was approved by the Intramural Research Board of the National Institute of Neurological Disorders and Stroke.

Data were analyzed from a total of 61 consecutive, referred patients with neurogenic OH (data were excluded from 2 other patients with PAF deemed atypical because of neuroimaging evidence for intact cardiac sympathetic innervation).11 Eleven patients had PAF, 51 had PD (24 with OH, 27 without), and 34 had MSA (26 with OH, 8 without). Data were also included from ongoing databases of control subjects, including 32 middle-aged (≥45 years old) patients with essential hypertension, 12 middle-aged patients with chronic orthostatic intolerance, and 41 middle-aged, healthy volunteers. The healthy volunteers and patients with chronic orthostatic intolerance had about the same body mass as did the patients with OH; patients with essential hypertension had higher body mass (Table).

All of the patients with PAF, PD, or MSA were referred to the National Institutes of Health by their neurologists or internists, with confirmation of the referral diagnosis by clinical and laboratory evaluations, as follows. PD was diagnosed by 1 of 2 groups of findings. The first consisted of 3 classic components of PD—cogwheel rigidity, bradykinesia, and pill–roll tremor—combined with clear improvement in movement by levodopa treatment. The second consisted of parkinsonism (cogwheel rigidity, expressionless face, hypokinesia, stooped posture), without evidence of cerebellar degeneration or dementia, combined with neuroimaging evidence for loss of cardiac sympathetic innervation.12–14 MSA15 was identified by gradually progressive clinical worsening; absent or minimal autonomic abnormalities, such as erectile failure in men, urinary incontinence developing early in the clinical course, and slurred speech; and neuroimaging evidence for intact cardiac sympathetic innervation. Most patients with MSA also had marked OH, but a small subgroup did not. PAF was identified by the early development of severe, persistent, consistent OH without evidence of a secondary cause (eg, chronic debility, dehydration, diabetes, adrenal insufficiency, amyloidosis, multiple myeloma) and without clinical signs or symptoms of central neurodegeneration.19

We defined OH as a reproducible decrease in systolic blood pressure ≥20 mm Hg and a decrease in diastolic blood pressure≥10 mm Hg between 15 minutes of supine rest and 5 minutes of standing (or until symptomatic from hypotension after <5 minutes of standing). Normally, mean arterial pressure remains the same or increases slightly during upright posture, depending on factors such as gender and age.20–23

Blood pressure was measured noninvasively by using either a tonometric device placed on the radial pulse and calibrated against the brachial blood pressure measured by an automated cuff (Colin) or a photoplethysmographic device placed around a finger (Portapres, TNO Biomedical Instrumentation). The position of the arm was at heart level for both supine and upright measurements of blood pressure. Mean arterial pressure was calculated from the diastolic pressure plus one third the pulse pressure.

Baroreflex-cardiovagal gain was measured from the relation of the cardiac interbeat interval to systolic blood pressure during Phase II of the Valsalva maneuver.24 Normally, as blood pressure and pulse pressure fall because of decreased cardiac stroke volume, parasympathetic outflow to the heart decreases. Heart rate therefore increases, and the slope for the relation between the beat-to-beat electrocardiographic R-R interval and systolic blood pressure provides an index of baroreflex-cardiovagal gain. Most of the increase in heart rate during Phase II of the Valsalva maneuver results from decreased cholinergic outflow to the heart because atropinization virtually abolishes this increase.25

In the latter portion of Phase II, sometimes called Phase II L, blood pressure normally increases from its nadir owing to reflexive, sympathetically mediated cardiovascular stimulation; in Phase IV, the blood pressure normally “overshoots” the baseline value, presumably owing to ejection of a normal stroke volume into a reflexively constricted vasculature. A progressive decline in blood pressure during Phase II and absence of pressure overshoot in Phase IV therefore characterize patients with sympathetic neurocirculatory failure (Figure 1).26

Antecubital venous blood was sampled through an indwelling intravenous catheter, and vital signs were measured after at least 15 minutes of supine rest and after 5 minutes of standing upright. Plasma levels of catechols were assayed by batch alumina extraction followed by liquid chromatography with electrochemical detection, as described previously.27 The limit of detection for plasma NE was ~30 pmol/L.

Statistical testing included ANOVAs for comparisons between >2 groups; independent-means t tests for comparisons between 2 groups; and χ² calculations for analyzing frequency data. Mean values were expressed ±SEM. A value of P < 0.05 defined statistical significance.
Results

Association Between OH and Sympathetic Failure
Data for beat-to-beat blood pressure associated with performance of the Valsalva maneuver were obtained from 8 patients with PAF, 19 with MSA/Oh, 22 with PD/Oh, and 4 with PD lacking OH. Of the 49 patients with OH, 46 had abnormal pressure responses during Phases II_L and IV of the Valsalva maneuver, whereas of the 30 patients without OH, only 7 had abnormal blood pressure responses in both phases ($\chi^2=42$, $P<0.0001$; Figure 1). Data for increments in plasma NE levels after standing upright for 5 minutes were obtained from 3 patients with PAF, 7 with MSA/Oh, 18 with PD/Oh, and 24 with PD lacking OH. Of the 28 patients with OH, 20 had a $>60\%$ orthostatic increment in plasma NE, whereas of the 24 patients without OH, only 8 had a $<60\%$ increment ($\chi^2=13$, $P=0.0003$).

Thus, in general, in this study OH was associated with an abnormal blood pressure pattern characteristic of sympathetic neurocirculatory failure and with neurochemical evidence for deficient, sympathetically mediated NE release during orthostasis.

Association Between Supine Hypertension and OH
Mean arterial pressure (excluding data from patients with essential hypertension) varied with diagnosis ($F=6.1$, $P=0.0001$). The 3 groups with OH had significantly higher mean arterial pressure during supine rest than did the control group, with mean values at least as high as in the essential hypertension group (Figure 2). In contrast, the 2 groups with PD or MSA lacking OH did not have significantly higher mean arterial pressure than did the control group. Among patients with PD or MSA, those with OH had higher mean arterial pressure during supine rest ($109\pm3$ mm Hg) than did those lacking OH ($96\pm3$ mm Hg; $t=3.2$, $P=0.002$).

Drug Treatments
As indicated in Table, the groups with OH differed in terms of treatment with levodopa or fludrocortisone. Because levodopa has been thought to contribute to OH and fludrocortisone to supine hypertension, subgroups of patients being treated or not being treated with these drugs at the time of evaluation were compared in terms of supine mean arterial pressure, orthostatic change in mean arterial pressure, baroreflex-cardiovagal gain, supine plasma NE, and orthostatic change in plasma NE.

Among patients with PAF, MSA, or PD, the 32 on fludrocortisone had higher supine mean arterial pressure ($110\pm3$ mm Hg) than did the 64 off fludrocortisone ($100\pm2$ mm Hg; $P=0.0001$). Within the PAF, PD/Oh, and MSA/Oh groups, however, values for supine mean arterial pressure were clearly above normal, regardless of fludrocortisone treatment, and were similar to supine mean arterial
pressure in the essential hypertension group (108 ± 20 vs 105 ± 7 mm Hg in PAF; 116 ± 7 vs 98 ± 5 mm Hg in PD + OH; and 110 ± 5 vs 113 ± 6 mm Hg in MSA + OH). All 3 groups with PAF, PD + OH, and MSA + OH had large orthostatic decrements in mean arterial pressure (Figure 2). The levodopa-treated subgroups of PD + OH or MSA + OH patients did not differ from the corresponding levodopa-untreated subgroups in terms of the magnitude of orthostatic changes in mean arterial pressure, whether expressed in absolute or relative terms (−34 ± 4 mm Hg vs −38 ± 7 mm Hg; −0.28 ± 0.06 in PD + OH; and −31 ± 4 mm Hg vs −31 ± 4 mm Hg; −0.28 ± 0.04 in MSA + OH).

Baroreflex-Cardiovagal Gain
Baroreflex-cardiovagal gain varied with diagnosis (F = 5.8, P = 0.0001). Mean baroreflex-cardiovagal gain was markedly decreased in all 3 OH groups (Figure 3). The PD + OH group had much lower baroreflex-cardiovagal gain than did the PD group lacking OH or the control group, whereas the PD and MSA groups lacking OH did not have significantly lower mean baroreflex-cardiovagal gain than did the control group. Among patients with PD or MSA, those with OH had much lower mean baroreflex-cardiovagal gain (0.74 ± 0.10 ms/mm Hg) than did those lacking OH (3.13 ± 0.72 ms/mm Hg; t = 4.0, P = 0.0002). Most patients with supine hypertension and most patients with OH had baroreflex-cardiovagal gain that was < 2 ms/mm Hg (Figure 4).

Plasma NE
Plasma NE levels varied with diagnosis (F = 9.3, P = 0.0001), with markedly decreased mean plasma NE in PAF (Figure 3). The groups with PD + OH and MSA + OH had significantly lower mean plasma NE (1.31 ± 0.09 nmol/L) than did the corresponding groups lacking OH (2.33 ± 0.23 nmol/L; t = 4.66, P = 0.0001). The essential hypertension group did not differ significantly from the control group in mean plasma NE (data not shown).

Orthostatic fractional increments in plasma NE levels also varied with diagnosis (F = 5.2, P = 0.0003). The 3 groups with OH had much smaller orthostatic increments in plasma NE levels than did the control group, whether the increments were expressed in absolute or relative terms (Figure 5). Both MSA groups, with or without OH, had small, orthostatic, proportionate increments in plasma NE compared with those in the control group. As indicated in Figure 6, most patients with supine hypertension (excluding data from patients with essential hypertension) had plasma NE levels below the normal mean (χ² = 11, P = 0.001), as did most patients with OH (χ² = 51, P = 0.0001).

PD With Versus Without OH
The group with PD + OH had a significantly lower mean plasma NE level during supine rest (t = 2.6, P = 0.01), a...
smaller increment in plasma NE during standing (t=3.0, P=0.005), and lower mean baroreflex-cardiovagal gain (t=2.2, P=0.04) than did the group with PD lacking OH. Among 24 PD patients with both baroreflex-cardiovagal gain <2 ms/mm Hg and plasma NE <1.8 nmol/L, 19 had OH, whereas among 9 patients without either finding, only 1 had OH (χ²=13, P=0.0004). Nine patients had baroreflex-cardiovagal gain <2 ms/mm Hg or plasma NE <1.8 nmol/L but not both, and of these, only 2 had OH.

Discussion

The main overall finding in this study was that all patient groups with chronic primary autonomic failure and OH—PAF, MSA+OH, and PD+OH—also had supine hypertension, whereas patient groups with MSA or PD lacking OH did not, indicating an overall association between OH and supine hypertension in these diseases.

A simple potential explanation for this association would be the side effects of treatment. Treatment of OH with fludrocortisone might cause supine hypertension, and treatment of parkinsonism with levodopa might cause OH. The same severity of supine hypertension, however, occurred in PAF, MSA+OH, and PD+OH, regardless of fludrocortisone treatment, and the same severity of OH occurred in MSA+OH and PD+OH, regardless of levodopa treatment. Across all patients with PAF, MSA, or PD, those on fludrocortisone at the time of study clearly did have higher supine mean arterial pressure than did those off fludrocortisone; however, this could be explained by greater supine mean arterial pressure in the patients with OH, independent of fludrocortisone treatment. The finding of supine hypertension in PAF and MSA+OH, even in untreated patients, agrees with a recent report. We did not locate prior studies about supine hypertension in PD+OH.

In PD, there was no increased frequency of levodopa treatment in the group with OH. On the contrary, whereas most PD patients without OH were on levodopa, less than one half of PD patients with OH were on levodopa. In several patients, OH dominated the clinical picture, and we relied on cardiac sympathetic neuroimaging as an aid for the differential diagnosis of PD versus MSA, because in our experience and that of several other groups, PD+OH entails a postganglionic sympathetic noradrenergic lesion, with loss of sympathetic nerves throughout the left ventricular myocardium, whereas MSA+OH does not, sympathetic noradrenergic innervation of the heart remaining intact. The relation between the extent of OH, whether expressed as absolute or relative change in mean arterial pressure, and supine hypertension did not result from high blood pressure itself, because patients with essential hypertension, whose severity of supine hypertension was similar to those with OH, did not have OH.
Previous studies have not separately considered the unusual situation of MSA lacking OH. Overall, in MSA it has been proposed that supine hypertension reflects increased sympathetic neurovascular tone, because the patients have excessive depressor responses to ganglion blockade produced by intravenous trimethaphan, which reduces NE release from sympathetic nerve terminals, and excessive pressor responses to α2-adrenoceptor blockade produced by intravenous yohimbine, which increases NE release from the terminals. Although the present study was not designed to test this concept, the finding of lower, not higher, plasma NE levels in MSA+OH than in MSA lacking OH does not seem to fit well with the notion of increased release of NE from intact sympathetic nerve terminals as the explanation for supine hypertension in MSA.

In all 3 forms of chronic autonomic failure, markedly decreased baroreflex-cardiovascular gain attended the supine hypertension. Any of a variety of changes, alone or in combination, in functions of neuronal, hormonal, or autocrine-paracrine systems; intracellular processes; or arteriolar architectural changes (eg, increased wall-to-lumen ratios) could elicit supine hypertension in the absence of baroreflex buffering of blood pressure.

In PAF, increased sympathetic neurovascular tone probably does not explain the supine hypertension. Patients with PAF had markedly decreased plasma NE levels, even during supine rest, replicating the results of previous reports. PAF also features, if anything, attenuated blood pressure responses to trimethaphan and yohimbine. Such patients have “de-nervation supersensitivity,” with augmented vasoconstrictor and pressor responses to adrenoceptor agonists, and although the mechanism of this augmentation remains unclear, given the very low rate of release of NE from sympathetic nerves in the body as a whole, increased sympathetic nervation supersensitivity, sympathetic nerves in the body as a whole, and the lack of augmentation of blood pressure responses to trimethaphan or yohimbine, it is difficult to accept that supine hypertension in PAF results from “overcompensation” in the form of denervation supersensitivity of postsynaptic adrenoceptors, even coupled with baroreflex failure. We therefore favor the view that the association between supine hypertension and OH in PAF entails a hypertensive mechanism other than increased sympathetic nervous system outflows.

Supine hypertension in PD+OH might also require a mechanism independent of the sympathetic nervous system. Bases for OH in PD have not been studied as intensively as in PAF or MSA+OH. At least a dozen studies have noted, however, that all patients with PD and OH have a marked loss of sympathetic nerves throughout the left ventricular myocardium (studies have disagreed about the universality of this finding in PD lacking OH). The present finding of significantly lower plasma NE levels in PD with than without OH confirms previous reports and indicates a relatively smaller overall complement of sympathetic nerves in PD with than without OH. As in MSA+OH, in PD+OH, supine hypertension was associated with lower not higher plasma NE levels than in PD lacking OH.

Patients with PD+OH had lower NE levels than did the age-matched control subjects, and patients with PD lacking OH had higher NE levels than did the controls. The difference between PD+OH and PD lacking OH seems to have resulted from both an increase in NE release in the latter group and a decrease in the former. The abnormal blood pressure responses to the Valsalva maneuver indicated sympathetic neurocirculatory failure in all patients with PD+OH. Such abnormalities could reflect the loss of sympathetic terminals, as in PAF, or deficient baroreflexes, as in MSA+OH, or a mixture of both. The present findings provide some support for a combination of partial or heterogeneous sympathetic denervation and decreased baroreflex gain. Across all patients with PD, plasma NE levels were related inversely to baroreflex-cardiovascular gain, and most patients with PD+OH had both plasma NE < 1.8 mM/L and baroreflex-cardiovascular gain < 2 ms/mm Hg. There were some patients with PD, however, who had plasma NE levels > 1.8 mM/L and baroreflex-cardiovascular gain > 2 ms/mm Hg, or plasma NE levels > 1.8 mM/L and baroreflex-cardiovascular gain < 2 ms/mm Hg, but not both abnormalities. Most of these patients did not have OH. It appears, therefore, that in PD, for OH to be expressed might require both sympathetic cardiovascular denervation and disruption of baroreflexes.

**Perspective**

Because PAF entails generalized sympathetic denervation, PD organ-selective sympathetic denervation (especially noticeable in the heart), and MSA intact sympathetic innervation, one might speculate that in neurogenic OH, the relative contribution of the sympathetic nervous system to supine hypertension varies with the particular form of autonomic failure. On the other hand, considering the present findings that all 3 forms of OH entailed markedly attenuated plasma NE responses during orthostasis and very low baroreflex-cardiovascular gain, baroreflex failure might contribute to supine hypertension in these conditions.

The results might have practical implications in clinical neurocardiology. Clinicians should not feel reluctant to initiate levodopa treatment in patients with PD+OH, because such treatment does not necessarily worsen the OH. On the other hand, clinicians often face the dilemma of treating OH, knowing that such treatment can worsen supine hypertension. There might be a statistical increase in the long-term risk of cardiovascular morbid events due to worsened supine hypertension from treatment of neurogenic OH, but the risks of symptomatic orthostatic hypotension (eg, traumatic falls with hip fracture or subdural hematoma) are clear and immediate. On the basis of this reasoning, we treat OH vigorously, even at the expense of worsened supine hypertension, but the correctness of this practice remains to be established.

**References**


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An erratum has been published regarding this article. Please see the attached page for:
/content/42/4/e12.full.pdf
In the article by Goldstein et al in the August 2003 issue (Hypertension. 2003;41:136–142), a sentence in the abstract was incorrectly edited and resulted in a change of meaning. The sentence stated the following: “In PAF, supine hypertension is linked to both OH and low baroreflex-cardiovagal gain.” However, because this link is found also in two other forms of chronic autonomic failure, ie, multiple-system atrophy (MSA) and Parkinson’s disease (PD), the statement should have read as follows: “In chronic autonomic failure, supine hypertension is linked to both OH and low baroreflex-cardiovagal gain.” The Journal regrets this error.