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RESEARCH ARTICLE

# Altered venous capacitance as a cause of postprandial hypotension in multiple system atrophy

**Motoko Takamori**
**Masaaki Hirayama**
**Rei Kobayashi**
**Hiroki Ito**
**Naoki Mabuchi**
**Tomohiko Nakamura**
**Norio Hori**
**Yasuo Koike**
**Gen Sobue**

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M. Takamori, MD · M. Hirayama, MD
H. Ito, MD · T. Nakamura, MD
N. Hori, MD · G. Sobue, MD (✉)
Dept. of Neurology
Nagoya University Graduate School of Medicine
Nagoya 466-8550, Japan
Tel.: +81-52/744-2385
Fax: +81-52/744-2384
E-Mail: sobueg@med.nagoya-u.ac.jp

R. Kobayashi, MD
Dept. of Neurology
Nagoya Medical Center
Nagoya, Japan

N. Mabuchi, MD
Dept. of Neurology
Okazaki Municipal Hospital
Aichi, Japan

Y. Koike, MD
Dept. of Health Science
Nagoya University Graduate School of Medicine
Nagoya, Japan

**Abstract** Patients with multiple system atrophy (MSA) often have clinically significant postprandial hypotension (PPH). To elucidate the cause of insufficient cardiac preload augmentation that underlies PPH, we recorded calf venous capacitance (CVC) by strain-gauge plethysmography, in 17 MSA patients and eight healthy controls before and after oral glucose ingestion. Among 17 MSA patients, nine who showed a decrease in systolic blood pressure exceeding 20 mmHg and were diagnosed with PPH. MSA patients without PPH showed a significant decrease in CVC and a significant increase in cardiac output after oral glucose ingestion, as did controls; those with MSA exhibiting PPH showed a significant increase in CVC and no significant change in cardiac output. The change in CVC correlated positively with the decrease in systolic and diastolic blood pressure after glucose ingestion, and also correlated negatively with the increase in cardiac output. Physiologically, PPH is prevented by a decrease in venous capacitance, which increases circulating blood volume and cardiac output. In some MSA patients, failure of venous capacitance to decrease may induce PPH.

**Key words** postprandial · hypotension · multiple system atrophy · venous capacitance · autonomic nervous system

## Introduction

Postprandial hypotension (PPH) was first reported as a clinical problem in a patient with parkinsonism [20], although a change in blood pressure provoked by eating already had been recognized [5, 21]. Currently PPH is known to occur frequently among patients with autonomic failure caused by multiple system atrophy (MSA), pure autonomic failure (PAF) [16, 17], Parkinson's disease [18, 25], and diabetes mellitus [13]; PPH even may occur in hypertensive patients and healthy elderly subjects [14]. Like orthostatic hypotension (OH), PPH can be a major clinical problem because of various alarming, sometimes serious symptoms such as dizziness, nausea, lightheadedness, weakness, syncope, falls, angina pectoris, and cerebral ischemia [13, 26]. Various reported studies have sought to clarify the mechanism of PPH. Food ingestion increases portal blood flow

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and may cause hypotension [19, 24]. In normal healthy subjects, cardiac output increases postprandially while blood pressure does not decrease; in patients with PPH, cardiac output fails to increase [10, 16]. Cardiac output can be increased by increasing preload (venous return), decreasing afterload (systemic vascular resistance), and/or increasing myocardial contractility ($\beta$1 sympathetic stimulation). In a previous study, we found decreased afterload (vascular resistance) in PPH despite unchanged cardiac output, while healthy controls showed a postprandial increase in cardiac output in the absence of a significant increase in heart rate (HR) [6, 10]. If myocardial contractility augmentation by $\beta$1 sympathetic excitation were a major contribution to the normal postprandial increase in cardiac output, HR would be expected to increase more. Based on these observations, we hypothesized that the preloading effect of venous compliance is a crucial physiologic factor preventing PPH. However, no reported studies or anecdotal accounts have described venous compliance in patients with PPH. Venous compliance is pressure dependent value, so the measurement of venous compliance needs frequent examinations and takes some times [7, 23], and standard methods have not been established. PPH is a phenomenon which changes in minutes, so the measurement of venous compliance is difficult in PPH. We therefore substituted calf venous capacitance (CVC) for venous compliance [3], assessing its involvement in the pathophysiology of PPH.

**Table 1** Clinical features and autonomic function parameters in MSA patients with PPH, MSA patients without PPH, and controls

| | MSA with PPH ($n$ = 9) | MSA without PPH ($n$ = 8) | Controls ($n$ = 8) |
|---|---|---|---|
| Age (years) | 59.6 (9.0) | 60.5 (8.3) | 48.6 (10.2) |
| Gender (M:F) | 4:5 | 5:3 | 7:1 |
| Disease duration (years) | 3.4 (1.6) | 4.6 (3.4) | |
| *Baseline* | | | |
| SBP (mmHg) | 147 (18)$^{\$}$ | 125 (12) | 121 (15) |
| DBP (mmHg) | 82 (9) | 78 (6) | 70 (11) |
| HR (bpm) | 74 (12) | 71 (9) | 71 (10) |
| Clinical PPH | 2/9 (22%) | 0/8 (0%) | 0/8 (0%) |
| *Change after glucose ingestion* | | | |
| • SBP (mmHg) | −35 (12)$^{\dagger}$ | −2 (5) | 0 (7) |
| • DBP (mmHg) | −21 (7)$^{\dagger}$ | −6 (6) | 1 (6) |
| • HR (bpm) | 4 (6) | 1 (4) | −1 (5) |
| OH | 9/9 (100%)* | 6/8 (75%)** | 0/8 (0%) |
| Clinical OH | 7/9 (78%)$^{\ddagger}$ | 1/8 (13%) | 0/8 (0%) |
| *Change after 60° head up* | | | |
| • SBP (mmHg) | −47 (20)* | −29 (22)*** | −3 (9) |
| • DBP (mmHg) | −25 (11)* | −12 (21) | 1 (6) |
| NE supersensitivity | 8/9 (89%) | 4/8 (50%) | n.e. |
| *Change after NE infusion* | | | |
| • SBP (mmHg) | 31 (6) | 23 (15) | n.e. |
| • DBP (mmHg) | 19 (10) | 13 (17) | n.e. |
| NE (pmol/l) | 992 (610) | 1520 (654) | 1778 (824) |
| AVP (pg/ml) | 1.83 (1.57) | 0.98 (0.64) | 1.22 (0.70) |

*$p$ < 0.001 vs. controls. **$p$ < 0.01 vs. controls. ***$p$ < 0.05 vs. controls. $^{\dagger}p$ < 0.001 vs. both MSA without PPH and controls. $^{\ddagger}p$ < 0.05 vs. MSA without PPH, <0.001 vs. controls. $^{\$}p$ < 0.05 vs. MSA without PPH, <0.01 vs. controls. MSA, multiple system atrophy; PPH, postprandial hypotension; OH, orthostatic hypotension; NE, norepinephrine; AVP, arginine vasopressin; n.e., not evaluated

## Methods

### Subjects

The 17 consecutive probable MSA patients participating in this study included nine men and eight women (mean age at examination ± SD, 59.8 ± 7.9 years, range, 46–73; mean illness duration, 4.2 ± 2.6 years, range, 2–10). Probable MSA was diagnosed according to the criteria established by a consensus statement concerning the diagnosis of MSA [4]. Criteria for exclusion from this study were presence of cardiovascular disease, diabetes mellitus, peripheral neuropathy, and severely impaired motor function defined as a Modified Rankin Scale score exceeding 4 (inability to walk independently with or without a crutch). Eight normal healthy control subjects also were studied (Table 1). The ethics committee of the Nagoya University School of Medicine approved this study in full. We obtained informed consent from all subjects prior to study participation.

### Glucose loading test

#### Protocol

The test began at 9:00 AM in our laboratory, at an ambient temperature of 25°C. All medications and oral intake were withheld after the night before the study. Subjects lay supine on a bed and were monitored at rest for 30 min (baseline period), and then for another 30 min after oral ingestion of 75 g of glucose in 225 ml of water. Systolic blood pressure (SBP), diastolic blood pressure (DBP), HR, cardiac output, and arterial blood flow in the lower legs (LBF) were measured continuously. CVC was measured twice, at baseline and at 30 min after glucose ingestion. We defined PPH as a decrease in SBP following glucose administration that exceeded 20 mmHg [13]. Clinical PPH was defined as sensation of dizziness, visual disturbance, or a gradual fading of consciousness after food ingestion during the study or previously.

### Hemodynamic measurements

SBP, DBP, and HR were measured in the right radial artery at the wrist by tonometry (BP-508; COLIN, Aichi, Japan), which noninvasively determined the subject's beat-to-beat blood pressure. The electrocardiogram (ECG) was monitored. Cardiac output and LBF were monitored by impedance plethysmograph (4134; NEC San-ei, Tokyo, Japan). CVC was measured using a mercury-filled silastic strain-gauge plethysmograph (EC-5R; Hokanson Inc., Washington, USA). The strain gauge was positioned around the midpoint of the right calf to measure change in lower limb volume. After a blood pressure cuff at the right ankle had been inflated to 200 mmHg and had rendered the sole of the foot temporarily ischemic, a thigh blood pressure cuff was inflated additionally to 40 mmHg for 2 min, while the change in lower limb volume was recorded. CVC was defined as volume increase (ml/100 ml) [2, 3, 12]. CVC was

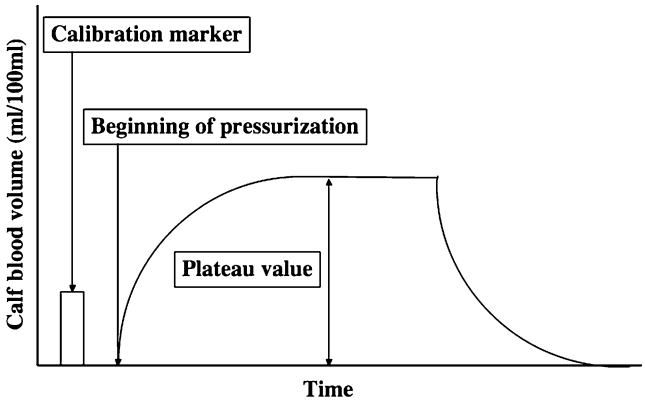


**Fig. 1** Illustration of limb volume curve. Calf volume changes following applied thigh blood cuff pressurization. Calf venous capacitance was calculated from plateau value divided by the height of the calibration marker. This marker represents a 1% change in the electrical resistance of the conductor inside the gauge, which is equal to a 1% limb volume

calculated by measuring the plateau value of limb volume curve after thigh blood pressure cuff was inflated to 40 mmHg and dividing this value by the height of calibration marker; this marker represents a 1% change in the electrical resistance of the conductor inside the gauge, which is equal to a 1% change in limb volume (Fig. 1). The change of blood pressure can influence the plateau time, but it does not influence the plateau value except when SBP goes down to less than 40 mmHg. Cardiac output and LBF were presented here as percentage variation from the baseline value, because the measurement of absolute value was not possible by impedance method.

#### Head-up tilting

On a different day than the hemodynamic tests, head-up tilting was carried out with the subject in a supine position on a tilt table with a foot-plate support. The table was tilted in a stepwise manner (20°, 40°, and 60° for 5 min each). We diagnosed OH when the decrease of SBP during head-up tilting exceeded 20 mmHg, or the decrease of DBP during head-up tilting exceeded 10 mmHg [15]. Clinical OH was defined as sensation of dizziness, visual disturbance, or a gradual fading of consciousness after standing during the study or previously.

#### Denervation supersensitivity to norepinephrine (NE)

To assess sensitivity to NE, we administered an intravenous NE infusion at a rate of 3 $\mu$g/min for 3 min. A diagnosis of the denervation supersensitivity was made when SBP increased more than 25 mmHg [9].

#### Plasma norepinephrine (NE) and arginine vasopressin (AVP)

To determine plasma NE and AVP, venous blood was sampled after rest in the supine position for at least 30 min.

Both NE infusion tests and examinations of plasma NE and AVP were performed on the same day as head-up tilting. NE infusion tests were not performed in the healthy controls.

#### Statistical analysis

The Fisher's exact probability test was used to compare the usage of vasopressor between the group with MSA inducing PPH and with MSA without PPH. The Mann–Whiney *U*-test was used to assess differences for continuous variables between the group with MSA inducing PPH and with MSA without PPH. Scheffé's *F*-test was used for post hoc testing between three groups (MSA with PPH, MSA without PPH, and controls). Wilcoxon signed-ranks tests were used to assess differences in cardiac output and CVC between determinations at baseline and after glucose ingestion. Relationships between change in CVC and decrease in SBP and DBP, between change in CVC and increase in cardiac output, were analyzed using Spearman's correlation coefficient by rank. Calculations were performed using the StatView statistical software package (version 5.0; Abacus Concepts, Berkeley, CA). Statistical significance was defined by $p < 0.05$. Values are presented as the means ± SD.

## Results

Clinical features and autonomic functions in each group are summarized in Table 1. Of 17 MSA patients, nine were diagnosed with PPH positive, while the other eight were PPH negative. All eight controls were PPH negative.

### Clinical features

No significant difference was evident in illness duration or age at examination between the MSA group with PPH and the MSA group without PPH. Controls were slightly younger than subjects in the other two groups, but the age difference was not significant. Four out of nine MSA patients with PPH took vasopressor before the examination. One took droxidopa, one amezium, one took both droxidopa and midodrine, and another both droxidopa and amezium. Only one out of eight MSA patients without PPH took vasopressor, both doroxidopa and midodrine before the examination. More patients with PPH used vasopressor than patients without PPH, although the difference was not significant.

### Response to glucose loading test

SBP at baseline in MSA with PPH was significantly higher than in MSA without PPH or in controls ($p < 0.05$ and $p < 0.01$, respectively). DBP or HR at baseline was not significantly different between MSA with PPH, MSA without PPH, and controls. The change in SBP after glucose ingestion in MSA with PPH was significantly greater than in MSA without PPH or in controls (both $p < 0.001$). The change in DBP after glucose ingestion in MSA with PPH was

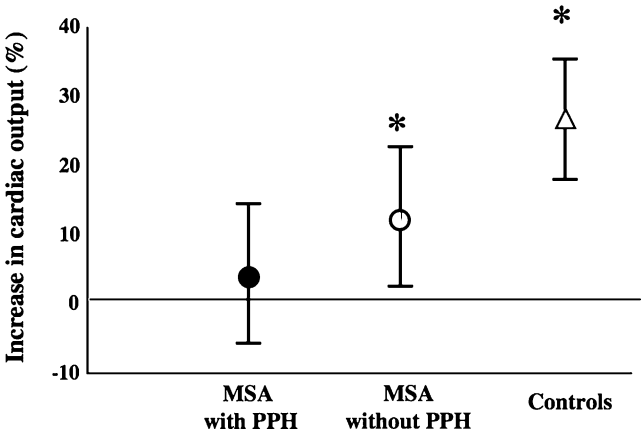


**Fig. 2** Increase in cardiac output after glucose ingestion in multiple system atrophy (MSA) patients with postprandial hypotension (PPH), MSA patients without PPH, and normal control subjects. Values are means, with error bars indicating SD. *$p < 0.05$ vs. baseline

significantly greater than in MSA without PPH or in controls (both $p < 0.001$). The change in HR after glucose ingestion was not significantly different between the three groups (Table 1).

Cardiac output increased significantly in MSA without PPH (13 ± 10%, $p < 0.05$) and in controls (26 ± 9%, $p < 0.05$), while output did not change significantly in MSA with PPH (4 ± 10%; Fig. 2). After glucose ingestion, CVC decreased significantly in MSA without PPH (from 2.20 ± 0.88% to 1.84 ± 0.60%, $p < 0.05$) and in controls (from 2.04 ± 0.24% to 1.72 ± 0.36%, $p < 0.05$), but increased significantly in MSA with PPH (from 1.80 ± 0.72% to 2.08 ± 0.88%, $p < 0.05$; Fig. 3). The change in CVC correlated positively with the decrease

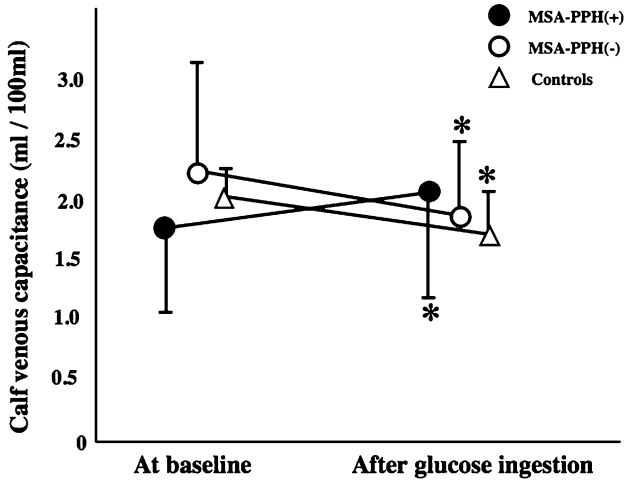


**Fig. 3** Change in calf venous capacitance after glucose ingestion in multiple system atrophy (MSA) patients with postprandial hypotension (PPH), MSA patients without PPH, and normal control subjects. Values are means, with error bars indicating SD. *$p < 0.05$ vs. baseline

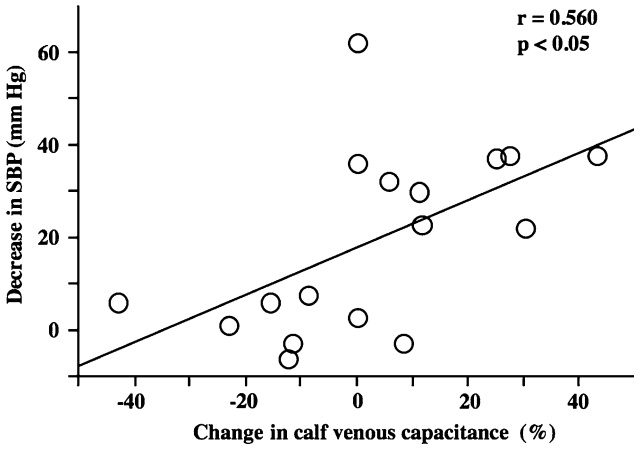


**Fig. 4** Change in calf venous capacitance showed significant positive correlation with decrease in systolic blood pressure (SBP) after glucose ingestion across the whole MSA population ($r = 0.560$, $p < 0.05$)

in SBP ($r = 0.560$, $p < 0.05$; Fig. 4), and with the decrease in DBP ($r = 0.524$, $p < 0.05$; Fig. 5) after glucose ingestion, and correlated negatively with the increase in cardiac output ($r = -0.583$, $p < 0.05$; Fig. 6). The increases of LBF after glucose ingestion were 1 ± 19% in MSA with PPH, 0 ± 37% in MSA without PPH, and 3 ± 9% in controls, and there were no significant differences among three groups.

## Other autonomic function tests

All nine MSA patients with PPH demonstrated OH. Frequency of OH in MSA with PPH (100%) and in MSA without PPH (75%) was significantly higher than in controls (0%); ($p < 0.001$, $p < 0.01$, respectively).

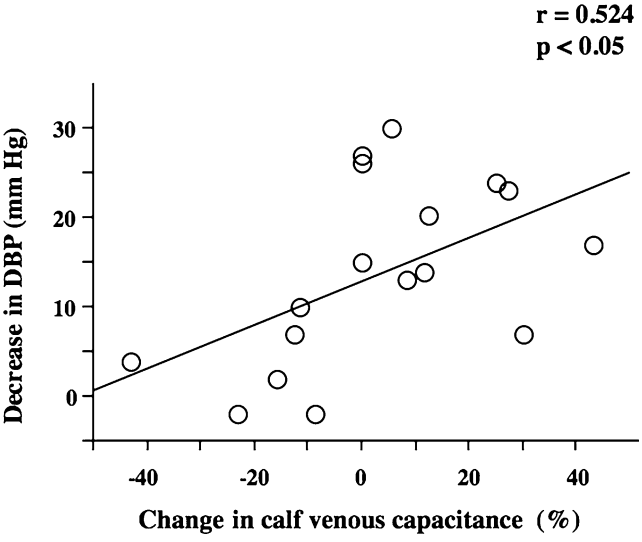


**Fig. 5** Change in calf venous capacitance showed significant positive correlation with decrease in diastolic blood pressure (DBP) after glucose ingestion across the whole MSA population ($r = 0.524$, $p < 0.05$)

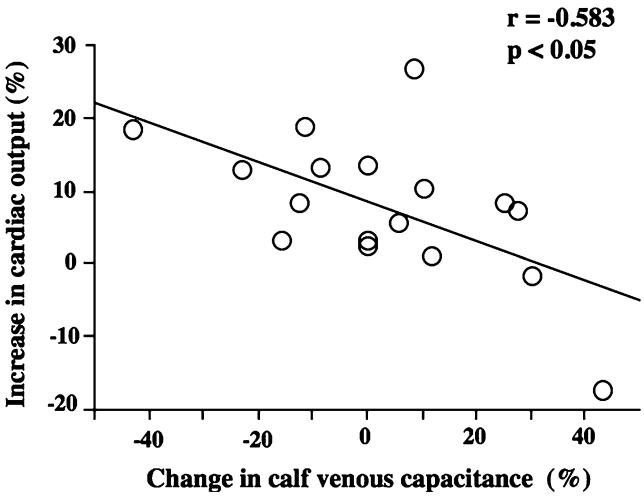


**Fig. 6** Change in calf venous capacitance showed significant negative correlation with increase in cardiac output after glucose ingestion across the whole MSA population ($r = -0.583$, $p < 0.05$)

Frequency of OH was slightly higher in MSA with PPH than in MSA without PPH, but the difference was not significant. NE supersensitivity was demonstrated more frequently in MSA with PPH than in MSA without PPH, but the difference was not significant. Plasma NE was slightly lower in MSA with PPH than in MSA without PPH or in controls, but the difference was not significant. Plasma NE levels were not significantly correlated with HR and with CVC changes after glucose ingestion across the whole MSA population. Plasma AVP in MSA with PPH was slightly but not significantly higher than in MSA without PPH or in controls (Table 1).

## Discussion

Reduced CVC and increased cardiac output after glucose ingestion were demonstrated in MSA without PPH and in controls, while in contrast CVC increased after ingestion in MSA with PPH. The change in CVC showed a negative correlation with the increase in cardiac output. Furthermore, the change in CVC after glucose ingestion correlated positively with the decrease in SBP and DBP. The venous system has been found to normally contain 64% of total blood volume while showing great distensibility, as to represent a reserve compartment for circulating blood volume [22]. Regulation of venous capacitance thus plays an important role in homeostasis of the systemic circulation system. A reduction of venous capacitance increases effective intravascular volume that results in an increase in cardiac output. A physiologic decline in venous capacitance can contribute importantly to systemic compensation for increased splanchnic blood flow induced by a meal; failure of venous capacitance reduction to occur is one of the factors underlying PPH, especially in MSA.

The precise mechanism of PPH has not been fully clarified. As presently understood, the causative sequence would begin with food ingestion inducing an increase in splanchnic blood flow [19, 24], and release of vasodilatory gastrointestinal peptides such as neurotensin [1, 8, 16]; as a result, systemic blood pressure tends to decrease. In normal subjects, the sympathetic nervous system is then activated to prevent systemic hypotension. However, this compensatory system is compromised in patients with autonomic failure, this defect eventually can result in PPH. Previous studies have shown evidence of impaired sympathetic compensation in PPH patients, including diminished baroreflex function, blunted compensatory increases in cardiac output, impairment of physiologic increases in muscle sympathetic nerve activity [6], and insufficient peripheral vasoconstriction [10, 13, 16]. These response defects are compatible with our previous finding that simultaneous treatment with $\beta_1$ and $\alpha_1$ agonists sufficiently increased cardiac output and vascular resistance to prevent PPH [11]. The present study implicates defective regulation of venous capacitance as an important contributor to PPH. A notable finding in this study is that CVC after glucose ingestion not only failed to decrease, but actually increased in MSA with PPH. The mechanism resulting in elevation is not clear, and further studies are necessary.

In this study HR did not change in MSA patients, even when SBP decreased significantly. This compromised physiologic response suggests that impaired baroreflex function also contributes to PPH in MSA. Thus, PPH appears to result from an interplay of several mechanisms.

MSA with PPH showed somewhat more frequent NE supersensitivity than MSA without PPH, while falling short of significance. NE denervation supersensitivity suggests that postganglionic sympathetic neurons are affected in this MSA subgroup. Thus, MSA with PPH had more severely impaired autonomic function than MSA without PPH. As the illness progresses, MSA patients have increasing difficulty maintaining an upright posture, so PPH becomes a more troublesome symptom than OH. The frequency of clinical PPH was low even in the group diagnosed as PPH by glucose loading test. PPH is a symptom that patients often fail to notice, although it can lead to severe clinical problems such as cardiac and cerebral ischemia [13, 26]. Not only patients but also caregivers and health care staffs need to keep alert to possible consequences of PPH.

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