Reflex vascular defects in the orthostatic tachycardia syndrome of adolescents

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Stewart, Julian M., and Amy Weldon. Reflex vascular defects in the orthostatic tachycardia syndrome of adolescents. J Appl Physiol 90: 2025–2032, 2001.—Dependent pooling occurs in postural orthostatic tachycardia syndrome (POTS) related to defective vasoconstriction. Increased venous pressure (Pv) >20 mmHg occurs in some patients (high Pv) but not others (normal Pv). We compared 22 patients, aged 12–18 yr, with 13 normal controls. Continuous blood pressure and strain-gauge plethysmography were used to measure supine forearm and calf blood flow, resistance, venous compliance, and microvascular filtration, and blood flow and swelling during 70° head-up tilt. Supine, high Pv had normal resistance in arms (26 ± 2 mmHg·ml⁻¹·100 ml−min) and legs (34 ± 3 mmHg·ml⁻¹·100 ml−min) but low leg blood flow (1.5 ± 0.4 ml·100 ml⁻¹·min⁻¹). Supine leg Pv (30 ± 2 vs. 13 ± 1 mmHg in control) exceeded the threshold for edema (isovolumetric pressure = 19 ± 3 mmHg). Supine, normal Pv had high blood flow in arms (4.1 ± 0.2 vs. 3.5 ± 0.2 ml·100 ml⁻¹·min⁻¹ in control) and legs (3.8 ± 0.4 vs. 2.7 ± 0.3 ml·100 ml⁻¹·min⁻¹ in control) with low resistance. With tilt, calf blood flow increased steadily in POTS with high Pv and transiently increased in normal Pv. Calf volume increased in all POTS patients. Arm blood flow increased in normal Pv only with forearm maintained at heart level. These data suggest that there are (at least) two subgroups of POTS characterized by high Pv and low flow or normal Pv and high flow. These may correspond to abnormalities in local or baroreceptor-mediated vasoconstriction, respectively.

METHODS

Patients and Controls

We studied 22 patients, aged 12–18 yr (19 girls, 3 boys), who were referred to our specialized center for investigation of symptoms of chronic orthostatic intolerance and had the POTS on head-up tilt (HUT) table testing. Patients with syncopeal episodes were specifically excluded. Thirteen normal control patients, aged 12–17 yr (10 girls, 3 boys), were also studied. Lightheadedness, nausea and vomiting, palpitations, fatigue, headache, blurred vision, abnormal sweating, and a sensation of heat while upright clinically characterized orthostatic intolerance with no other medical explanation for the symptoms. POTS patients complained of three or more symptoms of orthostatic intolerance for at least 3 mo. Technically adequate tracings were obtained from all patients with orthostatic tachycardia.

Control patients were recruited from adolescents referred for innocent heart murmur. Patients with a history of orthostatic intolerance were excluded. Adequate tracings were obtained from all normal controls. Electrocardiographic and echocardiographic evaluations were obtained in every patient.

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We excluded POTS patients and control subjects with Lyme disease or abnormalities in complete blood count, electrolytes, serum chemistries, thyroid-stimulating hormone, and urinalysis. All subjects were free of all obvious systemic illnesses, including cardiovascular disease, and were not taking neurally active or vasoactive medications. No trained athletes were enrolled. There were no completely bedridden patients. Informed consent was obtained, and the Committee for the Protection of Human Subjects (International Review Board) of New York Medical College approved all protocols.

**Definitions**

POTS was diagnosed by symptoms of orthostatic intolerance during upright tilt associated with an increase in sinus heart rate of >30 beats/min or to a rate of >120 beats/min during the first 10 min of tilt. Significant hypotension during HUT was occasionally observed and defined by a decrease in systolic blood pressure of ≥20 mmHg. Criteria were selected for consistency with the literature from adults (8, 12, 17) but may not be entirely appropriate for adolescents, in whom a higher heart rate may be more appropriate. We have used these criteria previously (20, 21). We partitioned patients a priori into two subgroups based on the presence or absence of calf venous hypertension defined by a supine resting calf Pv (see below) ≥20 mmHg. This pressure was chosen as the “high threshold” because we had never observed pressures ≥20 mmHg in normal subjects.

**Circulatory Evaluation**

Tests were performed in the laboratory during a single day. Our methods have been previously described (19–21). After an overnight fast, tests began between 9 and 10 AM. The electrocardiogram was monitored continuously and recorded to assess cardiac rhythm. Blood pressure was continuously monitored with an arterial tonometer (Collin Instruments, San Antonio, TX) placed on the right radial artery and was recalibrated every 5 min against an oscillometric sphyg- momanometer pressure. A respiratory impedance plethysmograph (Respirtrace 200, NIMS) was used to measure respiratory changes. Respiratory, electrocardiogram, and pressure data were interfaced to a personal computer through an analog-to-digital converter (DataQ Industries, Milwaukee, WI), and custom software was used to produce, display, and store R-R intervals, respiratory rate, strain-gauge output, and blood pressure (mean, systolic, diastolic, and phasic tracings) on a continuous basis.

**Peripheral Vascular Evaluation**

Mercury-in-Silastic strain-gauge plethysmography was used to measure simultaneous forearm and calf blood flow, the capacitance vessel compliance (volume-pressure) relation, and the microvascular filtration (flow-pressure) relation in the supine steady state in all patients. We also measured simultaneous forearm and calf size changes and blood flows during HUT. Methods were adapted from the work of Gamble et al. (3, 5–7) and have been used by us before (22), although previously only with the use of a single transducer. In brief, occlusion cuffs were placed around the limbs ~10 cm above a strain gauge of appropriate size attached to a Whitney-type strain-gauge plethysmograph (Hokanson). Strain gauges were positioned at approximately the maximum calf and forearm diameter. To determine limb blood flow, the cuff was inflated suddenly to a pressure of just below diastolic pressure, which prevented venous drainage. Wrist and ankle flow were prevented by inflating a smaller secondary cuff to above systolic blood pressure. Arterial inflow (in units of ml·100 ml tissue⁻¹·min⁻¹) was estimated as the rate of change of the rapid increase in limb cross-sectional area. Flow measurements were repeated in quadruplicate. After the return to baseline, we increased occlusion pressure slowly until limb volume change was just detected at cuff pressure just exceeding Pv (7). The value of Pv was also confirmed by the method of Gamble et al. (3, 5–7) in which the volume-pressure compliance curve (see below) was used to estimate the pressure at 0% change in volume (the pressure intercept). We used the mean arterial pressure (MAP) and Pv to calculate the supine arterial resistance to blood flow (in units of mmHg·ml⁻¹·100 ml tissue·min⁻¹) from (MAP – Pv)/flow. With cuffs deflated and the patient supine, the limb was progressively elevated, the elevation at the level of the strain gauge was measured, and the simultaneous decrease in limb cross section was recorded with each elevation. Heart rate and blood pressure remained unchanged throughout this procedure, suggesting that there was no systemic autonomic change. Pv at the strain gauge was estimated from the hydraulic formula $\text{P}_{\text{vaxt}} = 0.776 \times \Delta h$, where $P$ is pressure, $P_{\text{vaxt}}$ is Pv at rest, the constant 0.776 combines the pressure conversion factor from centimeters of blood to millimeters of mercury, and Δh is the height of the strain gauge above the table. This generated the descending portion of the capacitance vessel volume-pressure relation. After limbs were replaced at right atrial level, 10-mmHg steps in pressure starting at Pv to a maximum of 60 mmHg were used to produce progressive limb enlargement. This procedure generated the ascending limb of the capacitance vessel volume-pressure relationship and also the microvascular filtration flow-pressure relationship. A modified linear least squares analysis was used to separate venous filling from filtration, as we have done previously (22).

Once the volume response was partitioned into contributions from filling of capacitance vessels and contributions from microvascular filtration, the complete curvilinear limb volume vs. pressure relation for capacitance vessels was constructed using these data combined with the limb elevation data. The linear filtration rate vs. pressure relation was calculated. Volume change is expressed in normalized units of milliliter volume change per 100 ml tissue, and filtration is expressed in normalized units of milliliters filtered per 100 ml tissue per millimeters of mercury. Capacitance and compliance properties primarily reside in venous vessels, whereas microvascular filtration occurs in capillaries and venules. Under normal resting conditions at normal Pv, veins are partially filled. Congestion cuff pressure only causes an increase in limb size once the resting pressure is exceeded. The slope of the filtration-pressure relation was used to estimate the microvascular filtration coefficient in units of milliliters per 100 ml per minute per millimeters of mercury. The intercept with the pressure axis of the filtered flow-pressure graph is denoted $P_1$, and represents the mean pressure within the microvascular filtration units (capillaries and venules) at which microvascular filtration begins.

**Upright Testing**

After supine vascular measurements were complete, the subjects were tilted to 70° for a maximum duration of 30 min or until syncope, presyncope, or intolerable symptoms with or without significant hypotension occurred. An electrically driven tilt table (Cardiosystems 600, Dallas, TX) with a footboard for weight bearing was used. The right arm bearing the tonometer and the left arm bearing the strain gauge were maintained at heart level via table supports at all angles of
tint. Baseline recordings of blood pressure and heart rate in the supine position were obtained near the end of a 30-min resting period. The forearm and calf strain gauges were used to measure the change in forearm and calf size with time throughout tilt and to measure forearm flow by intermittent venous occlusion to 50 mmHg every minute and calf blood flow by intermittent venous occlusion to 75 mmHg every minute. Although calf venous filling was increased during upright tilt, we were able to define and to measure the rate of change of a further rapid increase in limb cross-sectional area representing calf blood flow. Similar techniques have been used by other investigators (6, 14). Patients with a positive test were returned immediately to the supine position, and the test was terminated. No pharmacological potentiation was employed. Comparison of flows and size change between POTS patients and control can be difficult because POTS patients do not usually complete 30 min of tilt. However, in all patients, we obtained at least 5 min and most often 10 min of recording.

**Statistics**

Data were compared by two-way analysis of variance for repeated measures. When significant interactions were demonstrated, the ratio of F values was converted to a t-distribution using Scheffe’s test, and probabilities were thereafter determined. A Bonferroni correction was used to correct for small samples. All results are reported as means ± SE. Statistically significant differences are reported for P < 0.05.

**RESULTS**

**Data in the Supine Position**

Heart rate and blood pressure. Resting supine heart rate was significantly (P < 0.05) higher in normal-Pv POTS (77 ± 3 beats/min) and high-Pv POTS (74 ± 1 beats/min) compared with controls (66 ± 2 beats/min). MAPs and phasic pressures were not different supine (72 ± 1 mmHg in normal-Pv POTS, 71 ± 1 mmHg in high-Pv POTS, and 75 ± 2 mmHg in controls).

Compliance relationship and microvascular filtration. The capacitance vessel pressure-volume relationships are depicted in Fig. 1. Curves for arms are similar for all groups. There was a trend toward reduced distensibility and capacitance in the range of 10–30 mmHg for both POTS groups compared with controls that did not reach statistical significance. However, compliance, defined as the rate of change of volume with pressure, was not different for POTS compared with control subjects.

The microvascular filtration coefficient was not different for POTS compared with controls. For the forearm, results are 0.009 ± 0.001, 0.010 ± 0.002, and 0.013 ± 0.004 ml·100 ml⁻¹·min⁻¹·mmHg⁻¹ in normal-Pv POTS, high-Pv POTS, and controls, respectively; for the calf, results are 0.007 ± 0.001, 0.005 ± 0.001, and 0.006 ± 0.001 ml·100 ml⁻¹·min⁻¹·mmHg⁻¹ for normal-Pv POTS, high-Pv POTS, and controls, respectively.

P1, and Pv. Resting P1 is shown in Fig. 2. P1 was significantly (P < 0.05) decreased in arms and legs of high-Pv patients compared with control subjects and significantly (P < 0.05) decreased in the calves of normal-Pv patients. Arm P1 was 11 ± 1 mmHg in normal-Pv POTS, 10 ± 1 mmHg in high-Pv POTS, and 10 ± 1 mmHg in controls. Leg P1 was significantly (P < 0.05) increased at 30 ± 2 mmHg in high-Pv POTS compared with 11 ± 1 mmHg in normal-Pv POTS and 13 ± 1 mmHg in controls. The highest recorded leg P1 in a control subject was 17 mmHg.

Peripheral flow and resistance. Supine blood flow and peripheral resistance are depicted in Fig. 3. Resting flow was significantly (P < 0.05) increased in arms and legs for normal-Pv patients compared with control and was significantly (P < 0.05) decreased in the legs of high-Pv patients compared with control. Both forearm and calf peripheral resistance were significantly (P < 0.05) decreased in normal-Pv patients compared with control but were not different from control for high-Pv patients, despite increased resting calf Pv values.

**Data During Upright Tilt**

Data are presented for the first 10 min of upright tilt during which syncope did not occur in any patient.
Limb volume changes. Figure 4 depicts forearm and calf volume changes during the course of upright tilt. Both POTS groups had a significantly (P < 0.05) greater increase in calf size compared with control patients. Increases occurred despite a shorter time of tilt (13 ± 2 vs. 30 min for controls, P < 0.05).

Forearm volume increased significantly (P < 0.05) in normal-Pv POTS, decreased significantly (P < 0.05) in controls, and remained unchanged in high-Pv POTS patients compared with baseline supine values in each group.

Figure 5 shows peripheral blood flow data averaged during upright tilt. Time 0 data are for resting supine condition. As depicted in Fig. 5A, forearm blood flow increased markedly albeit transiently above baseline in normal-Pv POTS. Because the forearm remained at right atrial level, there was little gravitational change in Pv or arterial blood pressure and thus no change in local stimulation of vasoconstriction. The situation was different for calf blood flow as depicted in Fig. 5B. Calf blood flow briefly increased in normal-Pv POTS above baseline values and then declined to approximately equal baseline levels. However, Pv POTS calf blood flow always exceeded control calf blood flow. Calf blood flow in high-Pv POTS patients increased significantly (P < 0.05) from low resting levels to exceed control subjects' flow (P < 0.05).

**DISCUSSION**

Compliance and Microvascular Filtration Do Not Account for Pooling

Neither arm nor leg compliance relation differed from control in the POTS groups, although there was a trend toward greater distensibility in control subjects. Similarly, microvascular filtration coefficients were not different for POTS patients compared with control subjects. This suggests that neither increased venous compliance and capacitance nor increased microvascular filtration coefficients account for dependent pooling in our POTS patients.
Our results showed that high-Pv patients had resting Pv averaging 30 mmHg, which, on average, exceeded $P_i$ at 19 mmHg for this group. Because we measured comparable microvascular filtration coefficients in patients and controls, this suggests that the lower limbs in high-Pv POTS patients are edematous when supine. Because $P_i$ is decreased in POTS patients and is determined by the balance of Starling forces and by lymphatic interstitial drainage, further work will be required to define interstitial properties in these patients.

**Peripheral Flow and Resistance: Impaired Baroreflex-mediated Sympathetic Vasoconstriction in Normal-Pv POTS and Impaired Local Vasoconstriction in High-Pv POTS?**

Normal-Pv POTS patients had increased supine resting forearm and calf flow and decreased supine resting forearm and calf resistance compared with control subjects. This implies decreased resting vascular tone related either to decreased sympathetically mediated vasoconstrictive tones or structural modifications (e.g., opening of arteriovenous connections). We hypothesize that baroreceptor-mediated sympathetic vasoconstriction is compromised, but local vasoconstriction is not. The response to HUT supports this speculation. When the calf is placed upright, calf flow initially increases briskly, because resting vasoconstriction is deficient, but then decreases, following a time course of several minutes, consistent with activation of local mechanisms of vasoconstriction (9, 10, 28, 29). Preliminary data supporting the interpretation of intact venoarteriolar reflex are shown in Fig. 6. After determination of baseline MAP, baseline calf Pv, and baseline calf flow, the occlusion cuff was rapidly inflated to a steady 50 mmHg to invoke the venoarteriolar reflex, and flow was remeasured. Calf resistance [calculated as $(MAP - Pv)/flow$] increased in four supine, normal-Pv patients and also in four supine control subjects with no change in heart rate or blood pressure. This was a local response. Baroreflex

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**Fig. 4.** Percent change in arm (A) and leg (B) volume from baseline supine values during head-up tilt for POTS patients and control subjects. Arm volumes increased significantly ($P < 0.05$) above baseline only in POTS patients with normal Pv and decreased significantly ($P < 0.05$) below baseline in controls. Leg volumes increased significantly ($P < 0.05$) above baseline in all POTS patients and control subjects. Leg volumes were significantly ($P < 0.05$) larger in all POTS patients compared with control. Values are means ± SE. *$P < 0.05$ compared with control. # $P < 0.05$ compared with baseline supine values.

**Fig. 5.** Flow in the forearm (A) and calf (B) averaged over patient groups during head-up tilt. Forearm flow is significantly ($P < 0.05$) elevated at rest compared with control in normal-Pv POTS and increases significantly ($P < 0.05$) further above baseline during tilt in normal-Pv POTS patients. Calf flow is higher than control ($P < 0.05$) in normal-Pv POTS patients and increases briefly above baseline ($P < 0.05$) in normal-Pv POTS patients ($P < 0.05$) and then returns to resting levels. Calf flow increases significantly ($P < 0.05$) throughout tilt in high-Pv POTS patients from resting low flow to levels significantly ($P < 0.05$) greater than control. Values are means ± SE. *$P < 0.05$ compared with control. # $P < 0.05$ compared with baseline supine values.
changes were not evoked. There is evidence to suggest that these local responses may account for one-half of the constrictor response to gravity (29). The arm, maintained at cardiac level in the present experiments, has no such gravitational stimulus, and, therefore, inadequate vasoconstriction in normal-Pv patients is not compensated by local reflex vasoconstriction in the arm. This may account for the sustained increase in arm blood flow during HUT shown in Fig. 5 in normal-Pv patients and the consequent increase in arm volume as shown in Fig. 4.

High-Pv POTS patients had decreased resting supine calf blood flow with normal supine calf resistance. Increased Pv should activate the venoarteriolar reflex (9, 10, 28, 29), if intact, resulting in increased calf resistance. Calf resistance is not increased in these patients, suggesting that the venoarteriolar reflex may be impaired. Pv is increased in calves but not forearms, we believe, because of the effects of sustained dependency of legs but not arms. We hypothesize that local vasoconstriction is compromised in high-Pv POTS, but baroreceptor-mediated sympathetic control is not. The response to HUT supports this speculation. When placed upright, the forearm is maintained at heart level, thereby defeating gravitationally stimulated local mechanisms. Arm flow does not increase in high-Pv patients. Leg flow, however, increases during early orthostasis because local vasoconstriction is defective and gravitational stimulation is invoked. Leg flow rates in high-Pv patients increase to approximate leg flow observed in normal-Pv patients with defective sympathetic innervation (Fig. 5). Preliminary data supporting the interpretation of defective venoarteriolar reflex are shown in Fig. 6. After determination of resting MAP, Pv, and flow, the occlusion cuff was inflated to a steady 50 mmHg to invoke the venoarteriolar reflex, and flow was remeasured. The maneuver produced a decrease in calculated calf resistance in three of four high-Pv patients and essentially no change in a fourth (which is abnormal), whereas resistance increased in normal-Pv patients and in controls as expected for competent venoarteriolar reflex.

Pathophysiological Hypotheses and Comparison with the Literature: The Normal-Pv Group

Our results from the normal-Pv (high resting flow) patients are consistent with hypotheses regarding the pathophysiology of POTS involving distal extremity denervation, or the so-called “long tract syndrome” (12, 23). The results suggest that this concept needs to be extended to include tracts to the arm, because there is decreased resistance in forearm as well as calf and swelling in the forearm as well as calf during orthostatic challenge. Clinical differences between the upper and lower extremities may relate to chronic dependence of the lower extremities rather than any intrinsic difference in sympathetic function. Evidence for the denervation hypothesis also appears in the earlier work of Streeten et al. (23–25), who defined a disease state of “hyperadrenergic orthostatic hypotension” to contrast with the hypoadrenergic findings in autonomic failure. Streeten et al. demonstrated persistent orthostatic tachycardia and orthostatic intolerance in hypotensive patients that was related to apparent denervation hypersensitivity of the superficial veins of the foot but not the hand. They theorized that lower limbs had enhanced responses to administered exogenous norepinephrine or phenylephrine because the numbers of $\alpha_1$-receptors had increased. A problem with this explanation for POTS is that superficial veins respond poorly to arterial baroreceptor stimuli and more to thermal stimuli or local vasoregulation. Thus the work of Streeten et al. could also be interpreted as supporting the hypothesis of defective local vasoconstriction in POTS patients. Contributions from active limb vasoconstriction to the orthostatic response are still controversial. Our data suggest that a contribution from passive elastic properties of capacitance vessels is not likely to be important to pooling in POTS. On the other hand, our data suggest that arterial constrictor defects account for increased passive venous filling.

Others have proposed sympathetic vasoconstrictive deficiency in chronic orthostatic intolerance. Thus Jacob et al. (11, 13) noted decreased blood volume in some patients with this syndrome, which was associated with increased plasma catecholamines. Furlan et al. (4) demonstrated increased muscle sympathetic nerve activity at rest, which did not increase appropriately with orthostatic stress. This resulted in blunted sympathetic response to orthostasis, consistent with inappropriate vasoconstriction to orthostatic stress. They found, as we did, that orthostatic tachycardia was most consistent with vagal withdrawal in their patients with chronic orthostatic intolerance. The most recent publi-
cation of Jacob et al. (12) more directly supports the idea of defective peripheral vasoconstriction. Altered norepinephrine spillover was observed in arms and legs accentuated on orthostasis in lower extremities. This work did not distinguish between impaired local and baroreceptor-mediated sympathetic defect and is thus compatible with either form of vasoconstrictor defect that we have hypothesized.

Support for a vasoconstrictor defect also comes from work by Brown and Hainsworth (1, 2). These investigators noted failure of vasoconstriction during orthostatic challenge of syncopal patients that was associated with capillary fluid shifts during orthostasis. The hypothesis of altered vasoconstrictor reserve has also been recently reviewed by Schondorf and Wieling (18).

In pediatric patients, defective vasoconstriction has been described in several reports during standing in children with orthostatic intolerance who had features in common with patients with POTS. Thus Jong-de Vos van Steenwijk et al. (15) reported decreased systemic vascular resistance with higher heart rates in children with syncope during orthostatic challenge, whereas Tanaka et al. (26) reported a similar decrease in resistance occurring in a group with “instantaneous” orthostatic hypotension. Patients with instantaneous orthostatic hypotension have persistent tachycardia and share other similarities with POTS patients. The studies lend support to the hypothesis of impaired vasoconstriction in orthostatic intolerance. Mechanical factors may also be important, including arterial wall and mooring structure composition. A recent paper describes enhanced incidence of the chronic fatigue syndrome in patients with the Ehlers-Danlos syndrome in which mechanical vascular properties are known to be affected (16).

Defective Local Sympathetic Vasoconstriction: the High-Pv Group

These patients are an interesting subgroup of POTS patients. The venoarteriolar reflex has been well described by Henriksen and co-workers (9, 10). Although it was originally believed to exert a relatively small effect on peripheral resistance and arteriolar vasoconstriction, that belief has been modified because of work by Vissing et al. (28). These authors have shown, at least for the cutaneous circulation, that the venoarteriolar reflex may be the most important mechanism for cutaneous arteriolar vasoconstriction during orthostasis. Furthermore, their data suggest that the venoarteriolar reflex may be the only significant effector of cutaneous arteriolar vasoconstriction during orthostasis, contributing primarily to closure of terminal arteriolar sphincters. The same is not true for muscle vasculature (27). Visible “pooling” comprises primarily blood and fluid within the skin. Therefore, it is likely that, if there were a defective venoarteriolar reflex, it would significantly compromise the response to orthostasis by causing increased arteriolar, capillary, and venous blood pressure, thus potentially resulting in interstitial edema.

Importance

This is the first report of a venous hypertensive variant of POTS. We speculate that venous hypertension relates to impaired venoarteriolar reflex. It also provides circulatory measurements in support of the vasoconstrictor defect hypothesis of POTS.

Limitations

We have presented one interpretation of our data. However, our interpretation generates predictions that can be tested. For example, lower extremity blood pressure is not increased by lower body negative pressure, which might yield a method to separate effects of increased arterial pressure on the myogenic reflex from increased venous transmural pressure with consequently quite different flow results. Such an approach has been adopted by Vissing and coworkers (27, 28).

Flow measurements rather than resistance calculations were obtained during upright tilt. We agree that resistance measurements are the most appropriate. During our standard HUT at 70° Pv, measurements in POTS patients were not feasible because tilt testing was often brief, tumultuous, and lacking even a quasi-steady state. We could only reliably obtain the flow data, which are presented. Therefore, we cannot definitely conclude that resistance is decreased while subjects are upright. However, because MAP was not different in POTS patients compared with control subjects and because it is unlikely that Pv is much less than control, it may be fair to speculate that arterial resistance decreases in POTS patients in whom arterial flow is increasing.

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REFERENCES


