Application of USAF G-Suit Technology for Clinical Orthostatic Hypotension: A Case Study

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Introduction: The purpose of this study was to determine the effectiveness of a USAF anti-gravity suit (G-suit) on the stability of a patient with chronic orthostatic hypotension. Methods: A 37-yr-old female with a history of insulin-dependent diabetes mellitus (IDDM) and symptomatic orthostasis was evaluated and the results were compared with those of non-diabetic controls, matched for age, height, and weight. Cardiac vagal tone was assessed by determination of standard deviation of 100 R-R intervals (R-R SD). We assessed the carotid-cardiac baroreflex response by plotting R-R intervals (ms) at each of eight neck pressure steps with their respective carotid distending pressures (mm Hg). Heart rate and blood pressure were recorded in response to the Valsalva maneuver (VM) performed at an expiratory pressure of 30 mmHg to assess integrated baroreflex responses. Blood pressures and heart rate were measured during three 5-min stand tests to assess orthostatic responses: a) without G-suit; b) with noninflated G-suit; and c) with inflated G-suit (50 mm Hg). Results: The IDDM patient had minimal baseline cardiac vagal tone (R-R SD = 8.5 ms) compared with the average response of a control group of 24 subjects with orthostatic stability (R-R SD = 67.2 ± 7.1 ms). Carotid-cardiac baroreflex response was virtually non-existent in the IDDM patient (Gain = 0.06 ms · mm Hg^{-1}) compared to the control subjects (4.4 ± 0.8 ms · mm Hg^{-1}). VM responses corroborated the lack of cardiac baroreflex response in the IDDM patient, while blood pressure changes during the VM were similar to those of the controls. Upon standing, the IDDM patient demonstrated severe orthostatic hypotension (90 mm Hg SBP) and tachycardia without the G-suit. The G-suit, with and without pressure, reduced hypotension and tachycardia during standing. Conclusion: These results demonstrate successful application of Air Force technology as a useful alternative to pharmacologic intervention in the treatment of a patient with autonomic dysfunction leading to supine hypertension and orthostatic hypotension.

PERHAPS THE MOST common cause of secondary autonomic dysfunction leading to postural hypotension is diabetes (5). Postural hypotension is a disturbance of systemic blood pressure that affects approximately 10% of the diabetic population (17). Nonpharmacologic measures such as elastic stockings, increasing salt intake, orientation to avoid abrupt rise from bed, and head-up tilt during sleep are designed to counter hypotension during assumption of the upright posture. However, these countermeasures are often ineffective due to various physiological mechanisms underlying the pre-existing conditions. Symptomatic orthostasis due to autonomic insufficiency is often disabling and may require pharmacological intervention, which frequently causes side effects. The usual pharmacologic treatment of patients with orthostatic hypotension is designed to expand plasma volume. However, the concern with this approach is that many patients with orthostatic hypotension secondary to diabetes have normal plasma volume and can become hypervolemic and develop supine hypertension during long-term drug therapy (28). Thus, alternative therapeutic methods that can minimize supine hypertension and defend against acute orthostatic hypotension must be considered for treatment.

Antigravity garments (G-suit) have been used successfully to prevent pooling of blood in the lower extremities during orthostatic challenges, in emergency treatment of hemorrhagic shock, and as a tool for studying physiological characteristics of orthostatic insufficiency. However, effective application of G-suits for idiopathic postural hypotension has produced conflicting results (3,4,18, 22,25). Assessment of these G-suit treatments from previous investigations has been greatly limited by the absence of data on underlying physiological dysfunctions associated with orthostatic pathology and pharmacological treatments. Therefore, the purpose of this study was to assess the effectiveness of abdominal and lower body pressure exerted by an anti-G suit on orthostatic instability of a diabetic patient with chronic supine hypertension and orthostatic hypotension, and determine specific physiological deficiencies of blood pressure regulation that were associated with her condition.

HISTORY

A 37-yr-old female (height 173 cm and weight 77.1 kg) with insulin dependent diabetes mellitus (IDDM) and a history of postural hypotension was assessed. Although there are no uniform diagnostic criteria, orthostatic hypotension is often defined as a fall in systolic pressure of greater than 20–30 mm Hg when arising from the supine

position (5,19). The patient complained of frequent episodes of dizziness and fainting while in the upright position accompanied by chest pain, and was admitted to Jupiter Medical Center for treatment. The patient was maintained on the following medications over the 21 d preceeding the experiments: sucralfate 1 gm q12h (antiulcer); ranitidine hydrochloride 150 mg q12h (gastric acid inhibitor); enteric-coated aspirin 325 mg qd; verapamil 40 mg q8h (antihypertensive); cefadroxil monohydrate (antibiotic) 500 mg bid; furosemide 20 mg qd (diuretic); fludrocortisone 0.1 mg qAM (volume expander) and insulin as required. Fludrocortisone was withheld 3 days prior to the experiments and verapamil was withheld the day of testing. At the time of the experiment, hematology and blood chemistries were: WBC = 11.2; RBC = 3.87; Hb = 11.4; Hct = 33.9; Na = 139; K = 4.0; Cl = 103; CO_2 = 28; BUN = 28; Creatinine = 0.7. Blood glucose concentration during a 30-d period averaged 200 ± 90 (SD) with a range from 40 to 421.

Subjects, with characteristics comparable for age, height and weight to those of the patient and who underwent similar measurements during previous experiments conducted in our laboratory, were selected to serve as controls. All subjects included in the studies gave their written consent to serve as subjects for this investigation after they had been informed of all procedures and risks. All procedures were approved by the Human Research Review Board of NASA-Kennedy Space Center.

METHODS

Experimental protocol: At the end of a 30-min baseline control period in the supine posture, an antecubital blood sample was drawn to measure plasma norepinephrine (NE), arginine vasopressin (AVP), plasma renin activity (PRA), and atrial natriuretic peptide (ANP), and plasma volume was measured. Following the measurement of plasma volume, the stimulus-response relationship of the carotid-cardiac baroreflex was measured followed by the assessment of heart rate and blood pressure responses to a Valsalva maneuver. At 15 min after the baroreflex tests, the patient underwent three 5-min stand tests separated by 15 min in the supine position.

Carotid-cardiac baroreflex function: Non-invasive measurement of the carotid-cardiac baroreflex stimulus-response relationship was achieved as previously described (14). Hypo- and hypertensive stimuli were delivered to the carotid baroreceptors by a Silastic neck chamber covering the area of the carotid arteries. An initial pressure of 40 mmHg was delivered to the chamber and maintained for four R waves. With each succeeding R wave, the pressure sequentially stepped to approximately 25, 10, −5, −20, −35, −50, and −65 mm Hg followed by a return to ambient pressure. To avoid respiration-related variations of cardiac vagal outflow, we applied neck-pressure changes while the patient held her breath at mid-expiration. A test session consisted of five successful applications of the neck pressure sequence. Resting systolic (SBP) and diastolic (DBP) blood pressures were measured in the brachial artery using sphygmomanometry immediately prior to and following the protocol. Average values for R-R interval at each

pressure step were plotted against carotid distending pressures (CDP = SBP less neck chamber pressure). The stimulus-response carotid-cardiac baroreflex relationship was reduced to the calculation of maximum slope to provide an index of reflex sensitivity (determined by application of least squares linear regression analysis to every set of three consecutive points on the stimulus-response relationship).

Valsalva maneuver: The patient performed three Valsalva maneuvers at a controlled expiratory pressure of 30 mm Hg in the supine posture. Each trial included a 30-s baseline period of quiet breathing and a 15-s strain period, followed by a 2-min post-strain period. The patient blew into a mouthpiece connected by a short plastic tube to a calibrated pressure transducer. A small leak in the system prevented occlusion of the glottis. Continuous heart rate was measured by a standard electrocardiogram. Beat-to-beat mean arterial pressure (MAP) was estimated with a non-invasive finger plethysmographic device (Finapres, Ohmeda, Englewood, CO). Excellent estimates of directly measured intraarterial pressures during Valsalva maneuvers have been demonstrated with this device (20). Aortic pressure during Valsalva straining was considered to be mean arterial pressure less expiratory pressure. Expiratory pressure was used as an index of intrathoracic pressure changes during Valsalva maneuvers.

Heart rate variability: Prior to the measurement of carotid-cardiac baroreflex function, the magnitude of respiratory sinus arrhythmia (heart rate variability) was assessed by calculating the standard deviation of 100 R-R intervals during normal breathing.

Stand test: The subject was fitted with the standard Air Force G-suit (USAF CSU-13B/P). The G-suit consists of five interconnected bladders (2 thigh, 2 calf, and 1 abdominal) with inflow at the abdominal bladder. Three stand tests were conducted in succession in the following order: 1) without the G-suit (control); 2) wearing the G-suit without pressure; and 3) wearing the G-suit with 50 mm Hg pressure. Each stand test began with the patient lying for 15 min followed by 5 min of active standing. The patient was instructed to stand still with her feet placed 12 in apart, with her weight evenly distributed, and to refrain from moving. Thus, contractions of the leg muscles were restricted to those required to support stationary standing. Blood pressure and heart rate were measured continuously throughout standing. Antecubital venous blood samples were collected without stasis before the initial stand test and after the initial and third stand periods.

Plasma volume and hormone measurements: Plasma volume was determined using a modified Evans blue dye dilution technique. After the patient was stabilized in the supine position for 30 min, an intravenous injection of 11.5 mg of dye diluted with isotonic saline solution (2.5 ml) was administered. The dye from a 10-min post-injection blood sample, recovered from the plasma with a wood-cellulose powder (Solka-Floc SW-40A, James River Corp., Berlin, NH) chromatographic column, was compared with a standard dye solution at 615 nm with a spectrophotometer.

Antecubital venous blood samples were taken without stasis during the postinjection Evans blue dye sampling

(baseline) and at the end of the first stand test (without G-suit) and third stand test (with inflated G-suit). Radioimmunoassay procedures were used to analyze plasma for arginine vasopressin (AVP, Instar Nuclear Corp.), atrial natriuretic peptide (ANP, Peninsula Laboratories, Inc.), and plasma renin activity (PRA, Biotecx RIA kit). For the determination of AVP, samples were extracted using ODS-silica columns and then assayed using a disequilibrium RIA procedure. Spiked recovery was 92%, sensitivity was 0.5 pg/ml, within-assay coefficient of variability (CV) was 2.8% and between-assay CV was 9.9%. For determination of ANP, acidified heparinized plasma was extracted using C^{18} Sep-pak columns and the eluates were evaporated to dryness and then assayed by competitive binding radioimmunoassay. Recovery was 90%, sensitivity of the assay was 2.0 pg/ml, within-assay CV was 5.9% and between-assay CV was 7.3%. Measurement of PRA was performed by a RIA competitive binding procedure using a specific antibody, a radiolabeled antigen, a pure sample of antigen used as a reference standard, and a separation medium. The amount of unlabeled antigen in the sample being analyzed was determined by comparing the assay results to a standard curve prepared with known amounts of the unlabeled antigen. Recovery efficiency was 96%, sensitivity was 0.1 $ng \cdot ml^{-1} \cdot hr^{-1}$, within-assay CV was 2.7% and between-assay CV was 5.5%. Plasma norepinephrine (NE) concentrations were measured by high performance liquid chromatography (Waters Model 460). The within-assay coefficient of variability was 1.4% for NE and 1.9% for E; between-assay coefficient of variability was 3.8% for NE and 10.5% for E.

RESULTS

At the end of a 30-min supine rest period, clinical descriptive data was recorded to establish baseline values. The subject had a supine blood pressure of 175/95 mm Hg and a resting heart rate of 97 bpm. Resting plasma volume was 2609 ml and heart rate variability (R-R standard deviation) was 8.5 ms.

Baroreflex responses: The patient's carotid baroreflex stimulus-response relationship is illustrated in **Fig. 1,** depicting the average R-R interval response as a function of carotid distending pressure. The maximum slope of the stimulus-response relationship was 0.06 $ms \cdot mm$ Hg^{-1} and range of R-R response was 10 ms. Thus, heart rate decreased from 95.5 bpm at a SBP of 135 mm Hg to 94.0 bpm at SBP of 240 mm Hg. The cardioacceleration limb of the carotid-cardiac baroreflex was assessed from the changes in baseline R-R (630 ms) to minimum R-R (628 ms), which represents a change in heart rate of only 0.3 beats per min (from 95.2 to 95.5 bpm) during carotid pressure reduction of ~40 mm Hg.

Valsalva maneuver: Mean HR and BP responses to the Valsalva strain are illustrated in **Fig. 2** and **Table I.** The MAP response did not demonstrate a typical four-phase response. At the beginning of the maneuver (phase 1), there was a rapid increase of 14 mm Hg in MAP. Typically, phase I is followed by a biphasic blood pressure response (phase II) consisting of an early decline in MAP (early phase II) followed by an elevation in MAP (late phase II) (Fig. 2B). In the IDDM patient, MAP decreased
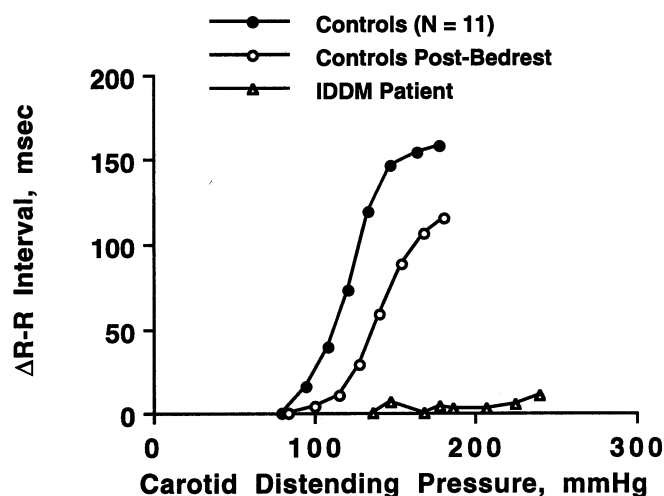

Fig. 1. Stimulus-response relationship of the carotid-cardiac baroreflex in the IDDM patient (open triangles) compared to the average response of 11 healthy subjects before (closed circles) and after (open circles) 30 d of bedrest. Data modified from Convertino et al. (5).

continuously throughout phase II without demonstrating a late phase II increase in MAP (Fig. 2A and Table I). Phase III occurred at the release of pressure and was characterized by a rapid decrease in MAP, usually followed by a phase IV rise and overshoot in MAP (Fig. 2B). There was no phase IV rise and overshoot in MAP in the IDDM patient (Fig. 2A). There was virtually no reflex heart rate response to changes in MAP in early phase II and phase IV.

Orthostatic responses: The SBP and HR responses to orthostatic challenge are illustrated in **Fig. 3.** Without the G-suit, SBP fell from 160 mm Hg to 94 mm Hg upon standing. SBP remained between 47 and 59 mm Hg below baseline to as low as 101 to 107 mm Hg during the final 3 min of standing. HR initially rose from 91 to 123 bpm during the first minute, but decreased to 104 to 110 bpm during the final 3 min of standing. With the non-inflated (0 mm Hg) and inflated (50 mm Hg) G-suit, the reduction in SBP was 31-33 mm Hg, but remained at or above 125 throughout standing. While blood pressure remained normal during G-suit application, standing heart rate rose 13-16 bpm and remained between 100 to 108 bpm throughout the test.

Hormone responses: Vasopressor hormone responses to standing are presented in **Table II.** There was virtually no response in PRA upon standing both with and without the G-suit. NE and AVP increased by 195% and 73%, respectively, in response to standing without the suit, and by 207% and 91%, respectively, while wearing the G-suit. ANP decreased by 22% after standing without the suit and by 38% after donning the G-suit.

DISCUSSION

To test the effectiveness of an anti-G pressure suit on providing functional stability to an IDDM patient with severe postural hypotension, we measured changes in heart rate, blood pressure and vasoactive hormones in response to a series of stand tests. In order to identify the capability of the G-suit to overcome cardiovascular compromise in such a patient, we also conducted a series
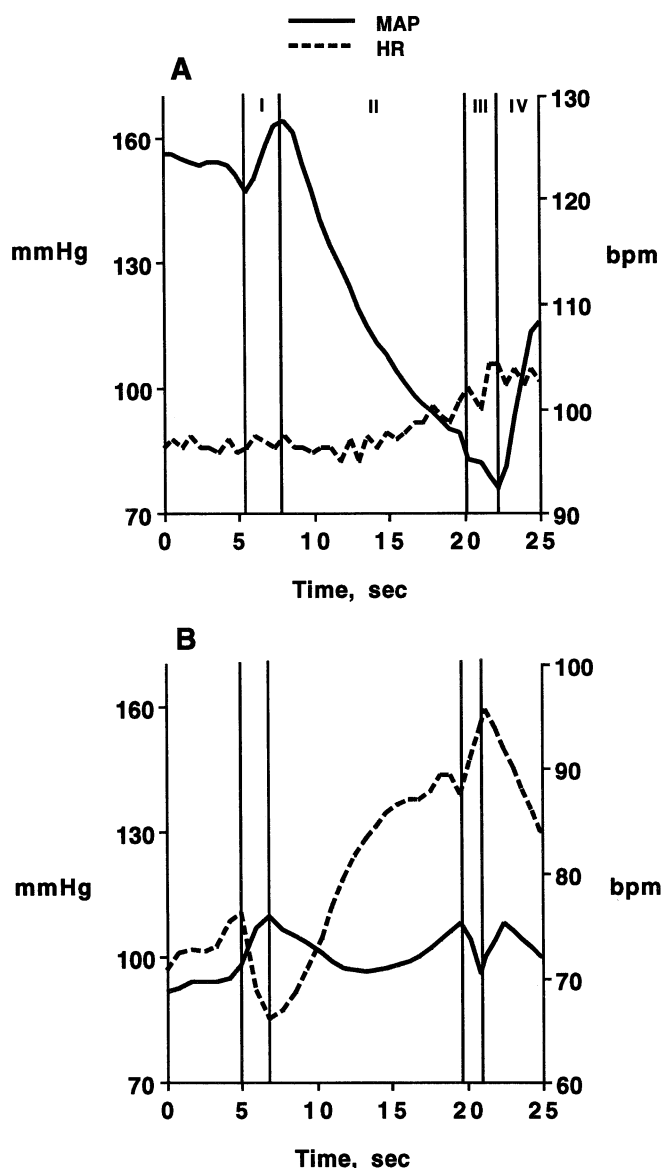
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Fig. 2. Heart rate (dashed line) and mean arterial pressure (solid line) responses to a 15-s Valsalva maneuver at an expiratory pressure of 30 mm Hg in the IDDM patient (Panel A) and in 8 subjects with normal baroreflex function (Panel B). Data from Panel B were modified from Luster et al. (15).
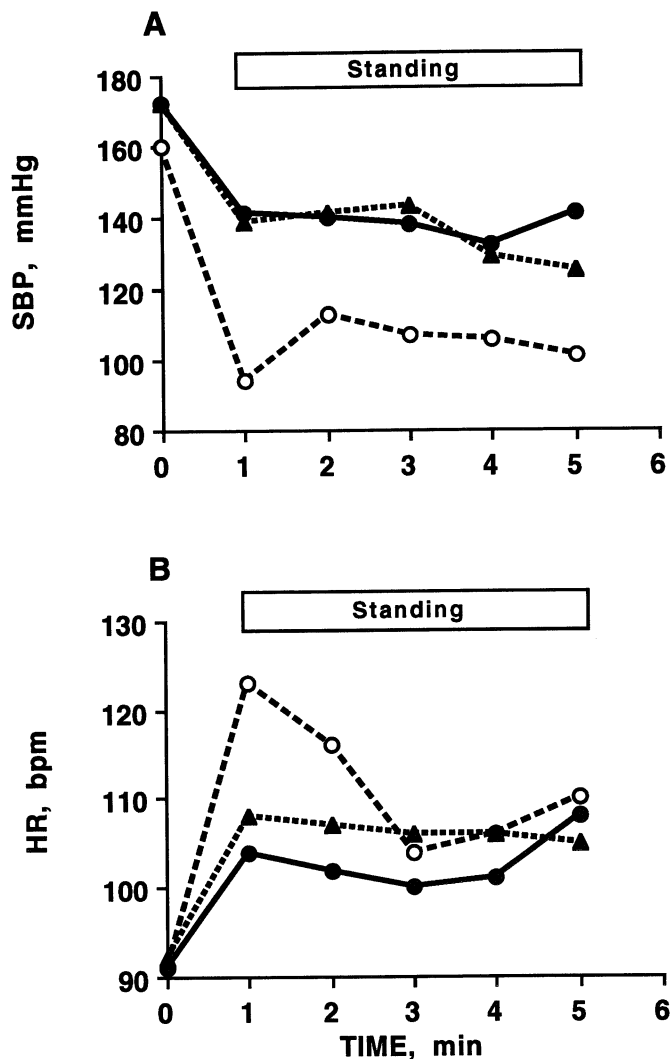

Fig. 3. Responses of systolic blood pressure (SBP, top panel) and heart rate (HR, lower panel) during a 5-min passive standing without G-suit (open circles and broken line), with G-suit not inflated (closed circles and solid line), and with inflated G-suit (closed triangles and broken line). Reference value at time "0" represents average SBP or HR during the final 5 min of a 30-min baseline period in the supine position.

of physiologic measures associated with blood pressure regulation to compare the patient's cardiovascular condition with functions in a group of 24 male subjects (con-

trols) matched for age (36 ± 1 yr), height (176 ± 1 cm), and weight (79.8 ± 3.5) and with normal orthostatic competence (9). We substantiated that our IDDM patient had mild hypovolemia (~2600 ml plasma volume) coincident with furosemide treatment compared to the controls

TABLE I. CHANGES IN HEART RATE (ΔHR) AND MEAN ARTERIAL PRESSURE (ΔMAP) DURING THE FOUR PHASES OF THE VALSALVA MANEUVER (VM).

VM Phase		IDDM Patient	Controls (N = 8)
Phase I	ΔMAP (mm Hg)	14.3	19.8 ± 3.3
Early Phase II	ΔHR/ΔMAP (bpm/mm Hg)	−0.06	−1.12 ± 0.5
Late Phase II	ΔMAP (mm Hg)	0.00	15.6 ± 4.3
Phase III	ΔMAP (mm Hg)	−18.9	−16.4 ± 2.6
Phase IV	ΔHR/ΔMAP (bpm/mm Hg)	0.00	−1.83 ± 0.7

TABLE II. VASOPRESSOR HORMONE RESPONSES OF IDDM PATIENT TO STANDING WITHOUT THE G-SUIT (NO SUIT) AND WEARING THE G-SUIT PRESSURIZED TO 50 MM HG (WITH SUIT).

Hormone	Supine	Stand (no suit)	Stand (with suit)
NE, pg·ml^{-1}	174	514	535
PRA, ng All·ml^{-1}·h^{-1}	1.7	1.4	1.7
AVP, pg·ml^{-1}	1.1	1.9	2.1
ANP, pg·ml^{-1}	32	25	20

NE = norepinephrine; PRA = plasma renin activity; AVP = plasma vasopressin; ANP = atrial natriuretic peptide.

(2980 ± 75 ml plasma volume). The IDDM patient suffered from chronic supine hypertension (175/95 mm Hg) and tachycardia (97 bpm) compared to the controls (113/70 ± 3/2 mm Hg and 65 ± 3 bpm, respectively). Severe dysautonomia was evident by minimal baseline vagal tone and no carotid-cardiac baroreflex response compared to controls with 67.2 ± 7.1 ms baseline R-R standard deviation and 4.4 ± 0.8 ms·mm Hg^{-1} baroreflex gain. The primary finding of this study was that the G-suit successfully compensated for these compromised mechanisms of blood pressure regulation in our IDDM patient by ameliorating orthostatic hypotension during passive standing without complicating the patient's supine hypertension. The success of the G-suit alone without pressure application may reflect the simple effectiveness of wearing tight-fitting garments in patients with conditions similar to those of our patient.

Our patient demonstrated complete carotid-cardiac baroreflex dysfunction compared to normal subjects before and after 30 d of bedrest confinement (ref. 5; Fig. 1). We are unaware of any previous studies to demonstrate this finding. Previous investigations using both human and animal models have demonstrated that impaired responsiveness of the vagally-mediated carotid-cardiac baroreflex response reduced the ability of subjects to adjust to transient reductions of blood pressure during standing and was associated with orthostatic incompetence (6,7,10,11,15). The findings from our IDDM patient were consistent with these data and extended this relationship to indicate that positive pressure applied to the lower extremities can compensate for this dysfunction.

After a transient increase in heart rate during the initial 2 min of standing without the G-suit, orthostatic hypotension and incompetence in the IDDM patient were associated with an inability to maintain elevated heart rate (Fig. 3). This observation was consistent with previous findings that the degree of impairment of baroreflex function correlated directly with a greater reduction in systolic blood pressure and decreased tachycardia while standing following prolonged bed rest confinement (6), in dogs with sinoaortic baroreceptor denervation (10), and patients with spinal injury (11). Therefore, the inability of baroreceptor-mediated reflexes to maintain appropriate tachycardia in a hypovolemic state during standing may represent a primary mechanism underlying orthostatic incompetence in IDDM patients with supine hypertension.

We used heart rate and blood pressure responses during a Valsalva maneuver with controlled expiratory pressure as a measure of integrated arterial baroreflex function in the IDDM patient and compared these responses with Valsalva responses in a group of 8 subjects (controls) with normal orthostatic competence (ref. 15; Table I and Fig. 2). The small rise in MAP during phase I is considered to be mechanical in nature and associated with vascular volume (24). Consistent with this notion, our hypovolemic IDDM patient demonstrated a 38% smaller rise in MAP during phase I than the normovolemic subjects with normal orthostatic stability. The ratio of unit change in heart rate to unit change in arterial pressure (ΔHR/ΔMAP) during early phase II and phase IV represents an index of nonspecific integrated baroreflex control of heart rate because pressure reductions

are likely to influence heart rate through interaction of cardiopulmonary, aortic, and carotid baroreceptor stimulation (13,21,23,24,26). Despite the large stimulus to arterial baroreceptors provided by a dramatic reduction of 75 mm Hg in mean arterial pressure during phase II in the IDDM patient (Fig. 2A) compared to only a 15-mm Hg reduction in arterial pressure in the controls (Fig. 2B), the IDDM patient demonstrated essentially no reflex heart rate response. The lack of heart rate response during phases II and IV in the IDDM patient compared to normal responses corroborated the complete impairment of the carotid-cardiac baroreflex response determined by the neck pressure cuff test (Fig. 1). These data suggest that a Valsalva maneuver test employing controlled expiratory pressure can be useful as a simple diagnostic technique for assessment of volemic status and autonomic function associated with orthostatic incompetence and application of effective G-suit countermeasures.

Our interpretation that impaired cardiac baroreflex function represented some pathophysiology of diabetes mellitus was based on the assumption that these reflex responses were not influenced by pharmacological treatments with a diuretic (furosemide), a mineralcorticoid (fludrocortisone), and a calcium channel blocker (verapamil). Although fludrocortisone could have influenced cardiac baroreflex responses, this drug was withheld from our patient for 3 d prior to testing in an effort to eliminate its potential effects. However, use of furosemide and verapamil were continued up to the day prior to testing for the treatment of our patient's supine hypertension. The effect of reduced vascular volume by furosemide does not influence cardiac baroreflex responses in humans measured by Valsalva maneuver (16) or direct carotid baroreceptor stimulation (27). Although our subject was not clinically normal, there is no evidence to suggest that her baroreflex responses should be influenced by volume contraction any differently than normal subjects. Verapamil reduces heart rate by its inhibitory effect on sinoatrial automaticity or atrioventricular conduction (1). However, direct chronotropic effects are questionable since verapamil, with a relatively short half-life (4 h), was withheld from our patient on the day of testing and our patient had an unusually high baseline heart rate (97 bpm). Verapamil may also cause reflex tachycardia through its vasodilatory action (1). Therefore, we cannot dismiss the possibility that pharmacological treatment of our patient complicated our interpretation of baroreflex dysfunction associated with diabetes mellitus alone. In any case, we determined that specific deficiencies in blood pressure regulation that were associated with diabetes mellitus were countered by appropriate application of abdominal and lower body pressure.

The IDDM patient did not demonstrate a late phase II elevation in arterial pressure compared to that normally observed in orthostatically stable subjects (Table I and Fig. 2). The typical late phase II increase in arterial pressure is attributed to increased autonomically-mediated reflex peripheral vasoconstrictor mechanisms (13). Our result suggests that the IDDM patient had virtually no capacity for increasing peripheral vascular resistance as a compensatory mechanism against an orthostatic challenge. This finding is consistent with previous data that patients with

idiopathic orthostatic hypotension did not increase systemic peripheral resistance upon standing or tilting compared to control subjects (3,12). However, the underlying mechanism for absent vasoconstriction was unclear. Despite the observation that hypovolemia causes enhanced peripheral vasoconstriction (27), lower blood volume did not elicit a vasoconstrictor response in our IDDM patient. Some investigators have reported depressed responses in plasma norepinephrine in IDDM patients during standing, an indirect index of depressed sympathetic nerve discharge (2,12) and that infusion of norepinephrine provides orthostatic protection (3). However, it is unlikely that a hypoadrenergic function was the mechanism of vasoconstrictor failure in our IDDM patient since she demonstrated a normal 3-fold elevation in plasma norepinephrine during standing compared to individuals with orthostatic stability who exhibit similar elevations (9). The inhibition of other circulating vasoactive neuroendocrine secretions may have contributed to the depressed vasoconstriction observed in our patient since renin-angiotensin and vasopressin showed little response to standing compared to elevations of 4-fold and 8-fold, respectively, in individuals with orthostatic stability (9). We cannot dismiss the possibility that the absence of a vasoconstrictive response in our IDDM patient was a consequence of verapamil treatment which reduces systemic vascular resistance by dilating peripheral arterioles (1). Thus, our data suggest that in addition to hypovolemia and impaired baroreflex control of heart rate, the capability of our IDDM patient to regulate blood pressure during orthostatism was compromised by the absence of a peripheral vasoconstrictor response capacity.

The use of G-suit technology for use in pathologic orthostatic hypotension is not new since other investigators have reported such applications (4,18,22). However, the effects of counterpressure garments on orthostatic tolerance in patients with idiopathic orthostatic hypotension have produced conflicting results. While data from some studies indicated that application of a G-suit provided significant protection in the rehabilitation of patients with severe postural hypotension (3,4,18,22), other studies failed to corroborate a protective effect (3,25). In one experiment, while blood pressure was maintained in an IDDM patient for 4 min after applying external pressure with a lower body gravity suit, hypotension developed to a similar degree to that without the suit after 5 min of standing (25). A major limitation to the interpretations of the effectiveness of applications of the G-suit from previous studies is that investigators failed to identify the underlying volemic states and autonomic dysfunctions associated with orthostatic pathology and pharmacological treatments. It is possible that G-suit effectiveness may be limited to specific abnormalities and may not be effective with other abnormalities. We believe our data are unique in that they are the first to our knowledge to identify specific physiological deficiencies of blood pressure regulation in a patient with IDDM; identification of these autonomic dysfunctions was important to our assessment of the effectiveness of the G-suit application in our patient independent of whether they were caused by the disease or secondary to drug therapy. Unexpectantly, our results also demonstrated the effectiveness of an anti-G suit without pressure application in compensating for these pa-

thologies in our patient. This observation suggests that simple tight-fitting garments designed to apply pressure to the legs and abdomen without the requirement of air pressure sources can be equally effective in providing protection against orthostatic instability in these patients. Our results suggest that application of anti-G suit technology could prove effective in ameliorating orthostatic incompetance in individuals with combinations of moderate hypovolemia and complete impairment of carotid-cardiac baroreflex responsiveness and peripheral vasoconstriction without adversely affecting their supine hypertension. This finding is not only important to patients with idiopathic orthostatic hypotension, but could prove effective in astronauts returning from spaceflight who share in common the development of hypovolemia and carotid-cardiac baroreflex impairment (8).

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