Circulatory Control in Idiopathic Orthostatic Hypotension (Shy-Drager Syndrome)

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SUMMARY
Of three patients with Shy-Drager syndrome, two presented with syncope related to exercise and, in all three, exercise stress clearly defined the element of orthostatic hypotension. Autonomic cardiovascular regulatory function was absent in two, but some residual autonomic function was present in the form of heart rate response in one patient who had milder postural hypotension. Hemodynamic studies were carried out with subjects supine and tilted 45° before and after exercise, following plasma volume expansion, and during the effects of isoproterenol and atropine. Despite disordered venoconstrictor responses demonstrated during exercise, hyperventilation, and after the Valsalva maneuver, decrease in central blood volume and right atrial pressure were not excessive with tilt, and total systemic vascular resistance remained essentially unchanged as did heart rate. Cardiac index may have decreased excessively (−24%), suggesting a cardiac component in production of hypotension. Absence of sympathetic cardiac effects, both chronotropic and inotropic, may play a significant role in orthostatic hypotension by limiting heart rate and preventing shift of the Frank-Starling curve to the left. Amelioration of postural effects by plasma volume expansion was noted along with increased cardiac output, stroke volume, and pulse pressure indicating that myocardial heterometric regulation was operative. Atropine had little effect, eliminating the possibility that parasympathetic depressor effects were responsible for postural hypotension. Exercise, by decreasing systemic vascular resistance even though venous return was adequate, produced hypotension even in recumbency and aggravated postural effects. Similarly, isoproterenol caused marked hypotension in supine subjects. Exercise, by reducing systemic vascular resistance, has a deleterious effect despite increased venous return and may precipitate postural symptoms. Exercise stress is of value in the evaluation of patients with syncope or cerebral ischemia secondary to autonomic dysfunction where orthostatic hypotension is of doubtful significance at rest.

Additional Indexing Words: Venous return Vascular resistance Syncope Blood volume Venous tone

Orthostatic Hypotension in the Shy-Drager syndrome of multiple degenerative lesions of the central nervous system is a consequence of impaired autonomic vasoregulatory function.1-2 The cases described by Bradbury and Eggleston3 may represent earlier examples of this syndrome. Hypovolemia is not found, and the amount of blood pooled in the lower extremity with standing is not excessive, suggesting that arteriolar vasoconstriction is inadequate when the patient assumes an upright posture.4-5 A cardiac contribution to the syndrome has not been identified.

The three cases of Shy-Drager syndrome reported herein exhibited unusual forms of orthostatic hypotension. Supine exercise induced arterial hypotension, and orthostatic hypotension was aggravated during and following exercise even though the venous return was adequate. These features led to the examination of vascular resistance and central
blood volume as well as inotropic and chronotropic cardiac responses to tilt, exercise, plasma volume expansion, and isoproterenol.

In the course of this investigation of the pathogenesis of postural hypotension, the effect of exercise emerged as a means of evaluating patients who complain of syncope even though mild hypotension occurs during quiet standing. In addition, the contribution of abnormal cardiac regulatory function became evident in two of three cases.

**Report of Cases**

Case I (K.E.), a man aged 74 years, was seen initially by us on June 24, 1963 for part of his periodic examination. He had had a myocardial infarct in August 1952, from which he had made a good recovery. He worked regularly at his real estate business and had no symptoms during moderate physical activity. He appeared in good health at examination. There was slight bilateral ptosis, somewhat more pronounced on the left side. The heart was not enlarged. There were no abnormal cardiac sounds. The heart rate was 74/min. The blood pressure was 150/94 mm Hg. Fluoroscopic examination of the thorax revealed considerable tortuosity and broadening of the thoracic aorta. The electrocardiogram showed evidence of the old transmural anteroseptal myocardial infarction.

When seen again on November 22, 1963, the patient had noted dizziness from riding on an elevator; it had occurred when the elevator stopped after descent. Two months later, he had a momentary attack of syncope just after walking across a street. Physical examination during this period showed no change from the initial observation. There were no bruits over the carotid vessels, symptoms were not produced by carotid sinus stimulation, and the blood pressure did not change significantly with changes in position. On one occasion at this period, the blood pressure was 114/74 mm Hg in the recumbent position, 114/74 mm Hg in the sitting position, 104/74 mm Hg immediately after standing upright, and 104/70 mm Hg after standing motionless for 5 minutes. A small pulsating mass was found in the midepigastrium. Radiographic study of this mass confirmed the clinical impression of an aneurysm of the abdominal aorta. An electroencephalogram was within normal limits. He continued to have occasional episodes of falling down when walking or when voiding in the standing position. On frequent occasions, when in these same situations, the patient had premonitory sensations of syncope without actually falling.

In July 1964, a fall in systemic blood pressure from 120/80 mm Hg in the sitting position to 80/55 mm Hg in the standing position was observed. The patient was treated with methylphenidate (Ritalin), 9-alpha-fluorohydrocortisone (0.2 mg/day) and, later, potassium salts by mouth. He was fitted with garments designed to compress the venous system of the abdomen and lower extremities. There was some improvement until late in 1965, when the falling began to be more frequent. It seemed to occur almost exclusively when the patient was actually walking, especially when he was going up an incline, and hot weather seemed to render him more susceptible. He would become weak, would lean up against a building for support, and then would slide down to the ground only to regain consciousness almost immediately upon striking the ground. In a few moments, he was able to get up and move off again. There was no dyspnea or chest pain at the time of the attacks. Postmicturition syncope continued to occur even while seated. In addition, the patient noted that symptoms occurred while he was seated after ingestion of large meals. Examination at this time disclosed a systemic blood pressure of 174/100 mm Hg when recumbent and 108/60 mm Hg after standing erect a few minutes. There was slight peripheral edema which was attributed to the 9-alpha-fluorohydrocortisone (0.2 mg/day).

The response to exercise in the erect posture was also observed. The blood pressure, when recumbent, was 168/98 mm Hg. Immediately after standing erect, it was 114/78 mm Hg. The patient then made 15 trips on and off a 9-inch step. The serial observations of blood pressure thereafter were as follows: immediate 82/54; after 30 sec, 84/60; after 1 min, 100/72; after 2 min, 114/78; and after 3 min, 108/78. The exercise induced no appreciable change in the heart rate nor did the patient complain of dizziness. Neurological evaluation revealed no evidence of peripheral sensory deficit although bilateral ptosis, muscular wasting, and fasciculations were observed. Although the patient had an expressionless facies, associative movements and the gait were normal. There was no evidence of neurogenic or obstructive dysfunction of the urinary bladder. Sweating was absent over the entire body as demonstrated by the starch-iodine method.

Case II (N.S.), an 83-year-old white man, was admitted to the hospital in January 1964, complaining of increasingly frequent fainting spells and diarrhea for the last 3 weeks. The patient had been under treatment for "low blood pressure" for 1 year. Occasional syncopal episodes and frequent lightheadedness or "dizzy" spells occurred while he was standing or upon dressing.
They could be aborted by sitting or lying down when dizziness first occurred. The patient denied vertigo, aura, seizures, or postictal symptoms. He stated that, frequently during the past year, syncope and lightheadedness were precipitated by rapid walking or standing after walking, and that bending forward tended to relieve these symptoms. Others had noted that the patient would begin to stagger after walking normally. The patient generally had a good appetite but had lost 15 lbs during the previous 6 to 12 months. He denied all symptoms of congestive heart failure other than occasional dependent edema. Past history revealed only that the patient had been treated for a renal calculus in 1957.

The patient was a well-developed, thin, white man in no acute distress. The heart rate was 75 beats/min while lying down, and the blood pressure was 105/60 mm Hg. The funduscopic examination revealed moderate tortuosity of the retinal arterioles. The cervical veins were not distended. Examination of the lungs disclosed no abnormalities. The heart was enlarged, extending 2 cm to the left of the mid-clavicular line in the fifth intercostal space. An ejection murmur which extended toward the neck was heard at the cardiac base. The aortic closing sound was normal.

No abdominal masses were noted and the peripheral pulses were palpable. There was evidence of mild muscular wasting of the arms and shoulders, and fasciculation of muscles was identified. The patient had an expressionless facies and had lost associative movements. The remainder of the neurological examination, including tests of sensory function, disclosed no abnormalities. Rectal and proctoscopic examination revealed only external hemorrhoids.

Sweating, as demonstrated using the starch-iodine method, was patchy over the trunk but absent on the extremities. The electrocardiogram showed first degree atrioventricular block but was otherwise normal. Radiographic studies showed aortic ectasia and mild cardiomegaly with aneurysmal dilatation of the thoracic descending aorta. The patient also had diverticula of the duodenum and sigmoid colon.

When the diarrhea was controlled and adequate food intake was maintained, postural hypotension became less severe, but it could be aggravated by exercise while standing. The patient became asymptomatic with administration of 1 g of NaCl three times daily and 9-alpha-fluorohydrocortisone, 0.2 mg daily. This was associated with a gain in weight of 5 kg and moderate edema of the ankles. Although quiet standing produced a reduction in arterial pressure from 180/80 mm Hg supine to 160/70 mm Hg, no syncpe or symptoms of hypotension were observed or reported unless associated with walking. Symptoms returned, however, when treatment was discontinued by the patient.

Case III (W.K.), a 60-year-old white man, was admitted complaining of syncope of 2 to 3 weeks' duration, during which time nausea and vomiting were frequent. The patient stated that the first episode of syncope had occurred 3 to 4 months earlier but with only two such episodes until onset of the present illness. These episodes occurred only upon arising. Episodes were described as sudden "blackouts" without aura. Loss of consciousness could, at times, be averted if he sat down with the earliest indication of faintness. Consciousness was regained almost immediately upon falling. The patient stated that he had almost no sweating over the trunk and upper extremities but denied other neurological symptoms.

History revealed alcoholism for about 10 years prior to admission with jaundice on one occasion. In addition, he had chronic bronchitis with mild pulmonary emphysema.

At the time of hospitalization, the patient was a well-developed man with recumbent blood pressure of 136/86 mm Hg and pulse of 88/min. Upon standing, blood pressure fell to 80/60 mm Hg and pulse increased to 100/min. Following recumbent exercise, the blood pressure fell to lower levels during standing, but the pulse rate increased to 110/min. The patient had a mildly increased anteroposterior chest diameter. The heart sounds were normal. Examination of the abdomen and extremities were within normal limits. Following treatment for peptic ulcer, demonstrated by x-rays, the nausea and vomiting disappeared, and the postural hypotension was reduced in severity. Concomitant with this improvement, a 7-lb gain in weight was noted to the day of hemodynamic study.

Neurological examination revealed no cranial nerve dysfunction. The deep tendon and sensory reflexes were intact as were all sensory mechanisms. Muscular strength was normal despite fasciculations. Sweating was absent over the trunk and arms (starch-iodine method). Signs of the type associated with Parkinsonism were absent.

Results of laboratory studies, including determination of blood volume, ACTH stimulation tests, electroencephalography, glucose tolerance tests, and electrocardiography, were within normal limits. Chest x-rays revealed only mild emphysematous changes.

**Methods**

Patients were studied while under observation in the Clinical Research Center. Blood volumes were determined using $^{131}$I-tagged human serum. 

Circulation, Volume XXXVIII, November 1968
albumin and 51Cr-tagged red blood corpuscles. Hemodynamic studies were carried out at 8 a.m. with the subjects in a fasting state. Right atrial, femoral arterial, and for exercise, brachial arterial pressures were determined with P 23 Db strain-gauge pressure transducers. Cardiac output (flow) was estimated using the indocyanine-green indicator dilution method with a Waters X-300 densitometer. Heart rate was monitored by electrocardiogram. Variables were recorded on a Visicorder (model 1108) and an 8-channel FM tape recorder. Central blood volume index (volume) was calculated using the method of Hamilton and associates. Systemic vascular resistance index (resistance) was calculated by formula: Mean arterial pressure — right atrial pressure (mm Hg) \div cardiac index (L/min/M^2) = resistance units.

Central venous injection with sampling from the femoral artery, including correction for sampling volume delay, was employed except during exercise, when the brachial artery was the sampling site.

Hemodynamic responses to upright tilt before and after exercise were determined in each subject as well as the response to an intravenous infusion of isoproterenol (3 \mu g/min) and atropine administered intravenously (0.5 to 1.5 mg). To evaluate the effect of plasma volume expansion, the response to upright tilt was studied after rapid intravenous administration of 500 ml of 10% low molecular weight dextran in saline solution. In all procedures, a steady state for at least 45 sec, as indicated by blood pressure and heart rate, was achieved before determination of cardiac output.

Subsequently, the effect of the Valsalva maneuver (40-mm Hg airway pressure for 20 sec) and micturition during recumbency upon the electrocardiogram and arterial pressure following intravenous administration of atropine was observed in case I. This study was made because postmicturition syncope occurred frequently, despite urination while seated.

Studies of venous tone were carried out by the circulatory arrest method. Following insertion of a short 20-gauge needle into a vein of either the foot or forearm, circulation to and from the extremity was occluded by rapid inflation of an automatic tourniquet to 250 to 300 mm Hg. Following equilibration of the venous pressure (usually 30 sec), the response of venous tone to the Valsalva maneuver, voluntary hyperventilation, and exercise of nonoccluded extremities was inferred by changes in venous pressure of the isolated vascular segment. Studies with use of both foot and forearm veins were carried out in each subject. The arm was not occluded for more than 3 min and circulation was re-established for at least 15 min between procedures. The hydraulic system utilized (20-gauge needle and rigid tubing) has a frequency response of 185 cycles/sec. The recording galvanometer is critically damped at 40 cycles/sec.

Results

Hemodynamic data are graphically depicted in figures 1 and 2. The alteration of variables in response to the experimental maneuvers are expressed as the mean percentage change related to supine resting
values (control) in the text. Anemia was not present, and blood volume was within normal limits in all subjects at the time of hemodynamic studies.

**Effect of Passive Tilt**

With subjects supine and resting, control flow values were normal in cases I and III and at the lower limits of normal in case II. Values for mean arterial pressure were mildly elevated in all three cases, and consequently resistance was well above normal in case II. Although central blood volume was greater than expected, right atrial pressure and heart rate were within normal limits. Upright tilt to 45° was followed by nearly proportional reduction in arterial pressure (27%) and flow (24%). Resistance was unchanged. Right atrial pressure decreased 50% and volume decreased 12%. Rate was unaffected by tilt in cases I and II; patient III exhibited an increase in rate and showed a less striking fall in pressure.

**Tilt After Exercise**

With supine exercise (alternate straight-leg-raising for 5 min), arterial pressure decreased 16%, and flow rose 24% as resistance decreased 38%. The heart rate increased significantly in case III only. Although right atrial pressure rose, central blood volume was unchanged. Immediately following exercise, patients were passively tilted upright (45°). Heart rate increased further in case III, but remained stable in the others. The response of right atrial pressure and volume on tilt was similar to that at rest. Resistance was not altered during tilt, but flow decreased to the same levels observed during resting tilt in cases I and II, so that a marked decrease in arterial pressure was noted. Case III differed again in that the decrease in arterial pressure was not so severe.

**Effects of Pharmacological Agents**

Intravenous administration of isoproterenol to supine patients resulted in marked hypotension; precluding upright tilt. Although venous pressure was increased, little change in volume was noted. Resistance decreased proportionally more than the flow rose. The heart rate increased modestly in cases I and II, but reached 120 beats/min in case III. Atropine (0.5 to 1.5 mg) by vein had little effect on the variables studied: rate, +12%; flow, −12%; volume, +2%; resistance, −6%; and arterial pressure, −15%, respectively. Venous pressure increased from 3.5 to 5 mm Hg (+50%). Atropine increased the rate in case III, but changes in arterial pressure, resistance, flow, right atrial pressure, and volume were minimal. The effects of upright tilt closely resembled the response to tilt before atropine.

**Effects of Plasma Volume Expansion**

From the change in hematocrit, infusion of low molecular weight dextran produced an increase of plasma volume of approximately 1 L. Venous pressure increased 350%, and volume, 21%. Flow increased and resistance decreased with little change in arterial pressure in cases I and II and a slight increase in case III. Heart rate was unaltered. A 45° upright tilt following dextran infusion produced minimal alteration in rate and arterial pressure. Flow and resistance remained near the supine values as volume and venous pressure (during tilt) were substantially higher than while the patients were supine prior to volume expansion.

**Venous Tone Responses**

Venous tone in arm and leg, as tested by the circulatory arrest method, did not change with the Valsalva maneuver or during voluntary hyperventilation. With exercise of the contralateral leg, pressure in the isolated venous system rose from 35 to 55 mm Hg (average values) and remained elevated despite cessation of exercise in all subjects (fig. 3). However, when the isolated venous system of the arm was used, no response to exercise of the leg was recorded.

**Effect of Valsalva Maneuver and Micturition**

With subjects I and II recumbent, the Valsalva maneuver produced the typical changes described in subjects without autonomic
IDIOPATHIC ORTHOSTATIC HYPOTENSION

function. In subject I, the effects of the Valsalva and micturition were compared after intravenous atropine (1.5 mg). With Valsalva, arterial pressure (200/108), rose during the initial period of increased intrathoracic pressures (215/144) and after release of intrathoracic pressure, it decreased to 150/96. With micturition, arterial pressure decreased slowly over a 30-sec period from 200/108 to as low as 113/72 and then gradually returned to control (20 sec). Mean arterial and pulse pressures seemed to be much more affected by micturition than by the Valsalva maneuver, and no change in heart rate or rhythm occurred in either case. The patient admitted to "bearing down" mildly at onset of micturition, but otherwise respiratory movements did not seem excessive.

Discussion

In the pathophysiology of orthostatic hypotension, three interrelated factors should be considered: (1) arteriolar resistance; (2) venous return; and (3) cardiac function, which includes heart rate and contractility. In addition to postural factors, the complex effects of exercise play an important role, and hypotension with exercise has been noted by others.3, 10

Normal Response

Normal subjects have several adaptive mechanisms which defend arterial pressure as the upright position is assumed. Flow diminishes since venous blood shifts to the lower extremities, decreasing central blood volume by approximately 25%.11 The consequent reduction in arterial pressure results in baroreceptor stimulation and reflex increase in rate, contractility, and arteriolar constriction.12, 13 The response of a normal individual to respiratory maneuvers such as Valsalva and micturition reflect these adaptive mechanisms as well as changes in venous tone.

Resistance

The primary disorder of regulation of resistance in this syndrome was demonstrated by the observation that orthostatic hypotension was much more severe when the vascular resistance was decreased by exercise. Presumably, failure of vasoconstriction in vascular beds of visceral and other nonexercising parts allowed reduction in mean resistance,13 thereby requiring greater flow to maintain arterial pressure. The observation that syncope frequently followed exertion in cases I and II is explainable. In case I, syncope could have been precipitated by food ingestion which may decrease splanchnic bed resistance14 or, at other times, by high, effective environmental temperatures, causing vasodilatation of the skin vessels,15 again opposed by vasoconstriction in other vascular beds.

The reduction of arterial pressure, which occurred in all three subjects with supine exercise, also occurred after beta-adrenergic stimulation by isoproterenol infusions. Although cardiac output increased, in part related to decreased after-load, the reduction in vascular resistance was disproportionately

Figure 3

Effect of contralateral leg exercise on the venous pressure of foot with arrested circulation. Persistent elevation of pressure is shown after cessation of exercise (indicated by altered respiratory rate). Right atrial pressure rose only during period of active exercise. Hyperentilation had no apparent effect on venous tone.
greater. These responses resembled those seen in normal subjects with depletion of catecholamine stores.3

The possibility that parasympathetic vasodepressor effects were responsible for the postural hypotension seems excluded since vascular resistance did not change with upright tilt and atropine had little effect. In case III, hemodynamic data and responses to tilt differed little after atropine from resting tilt studies despite an increased heart rate.

**Venous Return**

Although the various maneuvers did not produce patterns of venous return which differed from normal, hypotension did not evoke appropriate adaptive responses in venous tone. It seems clear that reduction of arterial pressure in these subjects may occur as vascular resistance decreases, even though venous volume is maintained and venous return is sufficient for a rise in flow appropriate to the level of exercise. Many factors enhancing venous return were operative with supine exercise. These are: (1) reduction in arteriolar resistance; (2) rhythmic muscular contractions; and (3) the abdominothoracic respiratory pump. During isoproterenol administration, rhythmic muscular contractions, important in muscular exercise, were absent. However, the venoconstrictor effect of isoproterenol appeared to operate since an increase in central blood volume was noted, as with supine exercise, in all three subjects. The decrease in RA pressure in cases II and III, despite an increase in central blood volume, has been observed by others.17

With exercise in the upright position, excessive reduction of venous return would not be expected since the mechanical effects of muscular contraction and respiration would still enhance venous return. Although laboratory studies were not carried out during exercise when upright, the hypotension which occurred in this circumstance could be demonstrated clinically in cases I and II, but not in case III. Postural venous pooling was not excessive. The mean decrease in flow (24%) was greater than reported by Lee and associates18 for normal subjects of comparable age (10%), but was not excessive compared to other studies of normal subjects.19

As stated above, the effect of the upright position on venous pooling has been studied in normal subjects and in patients with postural hypotension.4,5 It appears that the volume of blood pooled in the lower extremities does not exceed normal. The reduction in central blood volume in these subjects was not greater than one might expect in a normal subject despite the fact that the responses of venous tone were subnormal or absent. Samueloff and associates5 showed that venoconstriction is not an essential feature of the adaptation to the upright position in normal subjects. The absence of venoconstriction when hypotension exists, however, is distinctly abnormal.20

**Cardiac Factors**

Two of these subjects exhibited subnormal chronotropic cardiac response. With postural reduction in arterial pressure in normal subjects, increased heart rate and contractility represent important adaptive mechanisms. Warner and Toronto21 studied the effect of exercise in dogs with controlled heart rates and reported that cardiac output increases proportionally with increased metabolic demands by increasing stroke volume, thereby maintaining blood pressure. They inferred that some type of feedback control of cardiac output is mediated through autonomic mechanisms. Whatever the normal control mechanism, the blood pressure was not maintained in these patients during a relatively brief period of exercise. Recumbent exercise resulted in decreased arterial pressure because the decrease in vascular resistance exceeded the increase in cardiac output. Inadequate inotropic sympathetic effect is implicated in these subjects as the Frank-Starling curve was not shifted to the left.22 In patient III, who exhibited a chronotropic and possibly an inotropic cardiac response, the depression of arterial pressure was less severe with tilt.

Bruce and associates23 reported mild exertional hypotension in patients with heart
disease due to inadequate cardiac output in relation to the decreased vascular resistance. This occurred despite increased heart rate and was considered indicative of impaired cardiac reserve. Although the patients with Shy-Drager syndrome also had limitation of cardiac output, this seems more related to absence of autonomic effects than to cardiac disease. The increased stroke volume, cardiac output, and pulse pressure which occurred with plasma volume expansion are consistent with normal heterometric responses in the absence of autonomic influences.24

**Methodology of Measurements of Central Blood Volume**

Exercise studies were designed to resemble the activity which induced syncope and required exercise of both legs. Brachial arterial sampling was employed where femoral arterial sampling might be hazardous. It has been reported previously that sampling from brachial artery could induce time delay and skewing of indicator-dilution curves which might preclude accurate estimation of central blood volume.25 The delayed arterial transit of indicator encountered in some normal subjects was thought to be related to brachial arterial constriction. The rather fixed arterial resistance in our cases suggested, however, that skewing of curves would be minimal. Furthermore, calculated central blood volume from femoral arterial and brachial arterial indicator-dilution curves in the control state supported this conclusion and permitted the use of brachial arterial sampling during straight-leg-raising when femoral arterial sampling was not feasible.

**Response to Respiratory Maneuvers and Micturition**

The supine postmicturition hypotension observed in case I would account for syncope when urination was performed while seated and differs from the explanations published by others, which include cardio-inhibitory and vasodepressor effects leading to arrhythmia and hypotension. These are related to the Valsalva maneuver or depressor reflexes emanating from the distended bladder.26-28 The study carried out in case I indicated that, with micturition, a true Valsalva maneuver was not performed because there was no initial arterial pressure rise. The mild "bearing down" to which the patient admitted was apparently an increase in intra-abdominal pressure. Although the following explanation is conjectural, it seems reasonable to point out that, when intra-abdominal pressure greatly exceeds intrathoracic pressure, local collapse of the vena cava, compromising venous return, may be expected. The resultant decrease in cardiac output and stroke volume was reflected in diminished mean arterial and pulse pressures.29,30 Since the hypotension was not accompanied by any alteration in heart rate or rhythm and the patient had received 1.5 mg of atropine, it is unlikely that vasodepressor effects of parasympathetic origin were responsible.

The blood pressure response to a vigorous Valsalva maneuver (airway pressure of 40 mm Hg for 20 sec) was much less striking than the effects of micturition and also indicated the absence of autonomic effects. This difference may have been related to the application of intrathoracic pressure (glottis closed) as opposed to intra-abdominal pressure (glottis open). With the Valsalva maneuver, the transmission of increased intrathoracic pressure to the arteries tends to support arterial pressure, and compression of the pulmonary vasculature makes maximum use of this blood reservoir for left ventricular output. Pulmonary volume is quickly restored with inspiration, however. With micturition, increased intra-abdominal pressure may have a more profound effect on venous return since it can be maintained longer without discomfort and prevents maximum utilization of the pulmonary blood reservoir.

These patients displayed ample clinical evidence of autonomic dysfunction. Abnormalities in sweating in cases II and III and anhydrosis in case I, postural hypotension, and a nearly fixed heart are characteristic of this disorder. Skeletal muscle wasting and fasciculations were prominent findings, indicative of motor nuclear degeneration, and are
often seen in the Shy-Drager syndrome. \(^1\) \(^2\) \(^3\) \(^4\) The chief symptom in these subjects is syncope due to arterial hypotension and cerebral ischemia as the result of lack of vasoregulatory function. The patients presented here are particularly interesting because the clinical history suggested that syncope and near syncope were frequently associated with exercise, particularly in case I. Syncope was related to quiet standing only when mild dehydration was present (cases II and III). It would seem that patients suspected of syncope due to altered autonomic function should be given exercise stress to produce sufficient reduction of the blood pressure before other causes are thought operative. The occurrence of syncope with mild hypovolemia may also point to unrecognized autonomic dysfunction. Similarly, the consistent postmicturition syncope in case I also provided evidence of a vasoregulatory disorder.

References


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... it must be emphasized most strongly that it would be a grievous mistake to think that all practical problems in medicine could be solved more expeditiously by centrally managed planning. Numerous examples have been given of instances in which practical progress was frustrated until basic observations made without reference to the particular applied research goal, had been made, after which the practical goal became relatively easy of achievement. The genius of science, apart from the enormous power of the method, lies in the intelligence, the training, and the enthusiasm of its practitioners. There is probably no area of human endeavor in which the "free-enterprise system" has been more successful than in science.—Maurice B. Vischer: Applied Science and Medical Progress. In: Applied Technological and Medical Progress: A Report to the Committee on Science and Astronautics, U. S. House of Representatives, By the National Academy of Sciences, 1967, pp. 185-206.
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Circulation. 1968;38:870-879
doi: 10.1161/01.CIR.38.5.870

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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