proximately 60% of the patients whose fat intake exceeded NCEP Step 1 specifications were able to reduce fat intake to meet Step 1 goals after a single session with a dietitian. Of 92 patients with more than 30% fat intake at baseline, 34 (37%) had lowered their fat intake to less than 25% at first follow-up; 22 (24%) lowered their fat intake to 25-30%. Fat intake remained at more than 30% for 36 (39%) subjects.

The physician and nutritionist can provide positive feedback during follow-up visits and enhance long-term adherence to diet. For many patients, a substantial reduction in blood cholesterol level can be achieved.

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REFERENCES

1. Gorder DD, Dolecek TA, Coleman GG, et al. Dietary intake in the Multiple Risk Factor Intervention Trial (MRFIT): nutrient and food group changes over 6 years. J Am Diet Assoc 1986; **86**:744–751.
2. Report of the National Cholesterol Education Program expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. Arch Intern Med 1989; **148**:36–69.

COST-EFFECTIVE EVALUATION OF ORTHOSTATIC HYPOTENSION

Because of the multiple etiologic factors in orthostatic hypotension, it is necessary that the approach to diagnosis be cost effective. Our long-term experience with the hemodynamic evaluation of patients with orthostatic hypotension[1–5] has led to the development of such an approach (*Table 1*). Screening graded tilt testing, blood volume determination, and noninvasive hemodynamic evaluation form the basic minimum requirement of the work-up.

Noninvasive hemodynamic evaluation includes determination of cardiac output and cardiopulmonary volume in the supine position and during head-up tilt to 60°, or as tolerated by the patient, with subsequent calculation of changes in total peripheral resistance. Plasma catecholamines are measured with the patient in the supine position and during head-up tilt. Specific tests can then be planned according to the suspected diagnosis.

In patients with suspected autonomic insufficiency or in whom a diagnosis is not confirmed, intra-arterial blood pressure recording with autonomic reflex testing[1-3] is performed. This includes the Valsalva maneuver, cold pressor test, and pharmacologic baroreflex testing (phenylephrine IV and/or amylnitrite inhalation). In cases of suspected pacemaker syndrome, the tilt test is repeated with the pacemaker off (if safe and feasible) or with the pacemaker turned down as low as 30-40 bpm, in order to test the response of the underlying cardiac rhythm. In cases of suspected vasovagal syncope, the tilt test is repeated after administration of sublingual atropine or transdermal scopolamine. When hypovolemia is documented, the tests are repeated after expansion of blood volume, acutely with human serum albumin or long term with alpha fluorohydrocortisone and a high-salt diet. In patients with excessive upright peripheral venous pooling, the tilt test is repeated after lower limb compression with a support hose.

TABLE 1
EVALUATION OF ORTHOSTATIC HYPOTENSION

Screening tests
1. Graded tilt test
Hemodynamic evaluation
1. Blood volume determination
2. Noninvasive hemodynamics
3. Autonomic reflex testing
Specific tests
1. Repeat tilt test with lower limb compression in patients with excess venous pooling
2. Repeat tilt test following blood volume expansion in patients with hypovolemia syndrome
3. Repeat tilt test; postsublingual atropine or transdermal scopolamine in patients with vasovagal syncope
4. Repeat tilt test at a low pacing rate (or deactivate pacemaker) in patients diagnosed as pacemaker syndrome

LOCALIZING THE LESION IN AUTONOMIC DYSFUNCTION

The hallmark of autonomic insufficiency is an abnormal Valsalva test result. The normal Valsalva response has four components. Phase I is characterized by an initial rise in blood pressure associated with deep inspiration. Phase II represents the increase in intrathoracic pressure, resulting in a reduction of venous return and consequent marked diminution of pulse pressure. During phase II, severe vasoconstriction occurs in a normal person. Phase III represents the restart of normal breathing with the initial filling of the pulmonary circulation leaving the systemic circulation empty; thus, the arterial pressure falls dramatically. During phase IV, cardiac output is distributed again to the systemic circulation, which is markedly vasoconstricted, and therefore the end result is an overshoot of systemic blood pressure, both systolic and diastolic.

Heart rate changes during these phases have been well described. The blood pressure decrease during phase

II is associated with tachycardia, while phase IV is associated with bradycardia due to modulatory effects of the arterial baroreceptors. In a patient with autonomic insufficiency, both phase II and phase IV are abnormal; in Phase II, the blood pressure continues to fall without reaching a plateau, and in phase IV there is no overshoot of blood pressure. During these two phases, heart rate does not change significantly. An attempt was made to quantitate these abnormalities by calculating the constriction and acceleration indexes; however, use of these indexes was not widely adopted.

Once autonomic insufficiency has been diagnosed, the next task is usually to localize the site of the abnormality along the baroreflex arc. Several tests have been devised for this purpose.

Cold pressor test

In this test, the patient is asked to immerse the hand up to the wrist in ice-cold water for one minute. Somatic pain impulses are transferred through the spinothalamic tract to the hypothalamus. Efferent sympathetic impulses are directed to the heart and peripheral arterioles, producing tachycardia and an increase in total peripheral resistance (and blood pressure). A lesion in the efferent pathway of the baroreflex arc will prevent this response.

Hyperventilation

Hyperventilation for 15 seconds results in hypocarbia and vasoconstriction of the brain stem vessels. The normal vasomotor centers sense this anoxia and induce peripheral vasodilation. A lesion in the vasomotor centers results in no change in blood pressure.

Baroreceptor sensitivity testing

These receptors are located in the aortic arch and the carotid sinus. The pharmacologic approach to test sensitivity of these receptors consists of using either a phenylephrine IV bolus or amylnitrite inhalation. A bolus of phenylephrine (25 μg) increases the blood pressure and results in a reflex slowing of the heart rate if the baroreceptors are sensitive. Amylnitrite inhalation, on the other hand, produces a reduction in blood pressure and a reflex increase in heart rate. The sensitivity of the baroreceptors is quantitated by correlating the individual systolic blood pressure levels with their subsequent R-R intervals, obtained from a simultaneous ECG tracing.

Baroreceptor sensitivity was found to be blunted in a variety of conditions, notably old age, hypertension, uremia, and congestive heart failure.

Other tests

Other tests include the mental arithmetic and the reflex sweat tests. Efferent cardiac vagal fibers have been tested by examining the response of heart rate to atropine injection (0.03 mg/kg). Changes in heart rate in patients with idiopathic orthostatic hypotension are usually diminished compared to controls. Moreover, a nonpharmacologic approach for evaluation of vagal control of heart rate depends on the changes in R-R intervals (variations in heart periods or VHP), concomitant with respiration.[6]

Finally, extra-adrenal stores of norepinephrine have been tested by injecting tyramine intravenously in three consecutive doses of 1, 2, and 3 mg.[7] Because of the occasional extensive rise of blood pressure in some patients, 10 minutes are allowed between the individual injections after the blood pressure has returned to control level after each injection. This test allowed the differentiation of two forms of autonomic insufficiency: the central type and the peripheral type. Central autonomic insufficiency is characterized by abnormalities of long tracts (pyramidal, extrapyramidal, or cerebellar), as well as marked increase in blood pressure and plasma norepinephrine in response to tyramine (sensitization hypersensitivity). Peripheral autonomic insufficiency shows absent or blunted responses of both blood pressure and plasma norepinephrine to tyramine injection.

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REFERENCES

1. Tarazi RC, Gifford RW Jr. Systemic arterial pressure. [In] Sodeman WA Jr; Sodeman WA, eds. Pathologic Physiology: Mechanisms of Disease. Philadelphia, WB Saunders, 5th ed, 1974, pp 177–205.
2. Tarazi RC, Fouad FM. Circulatory dynamics in progressive autonomic failure. [In] Bannister R, ed. Autonomic Failure: A Textbook of Clinical Disorders of the Autonomic Nervous System. Oxford, Oxford University Press, 1983, pp 960–1114.
3. Fouad FM, Tarazi RC, Bravo EL. Orthostatic hypotension: clinical experience with diagnostic tests. Cleve Clin Q 1984; **52**:561–568.
4. Fouad FM, Maloney JD. Orthostatic hypotension: circulatory dynamics and clinical spectrum. [In] Furlan AJ, ed. The Heart and Stroke: Exploring Mutual Cerebrovascular and Cardiovascular Issues. New York, Springer-Verlag, 1987, pp 235–248.
5. Abi-Samra FM, Fouad FM, Sweeney PJ, Maloney JD. Syncope: a practical diagnostic approach. [In] Furlan AJ, ed. The Heart and Stroke: Exploring Mutual Cerebrovascular and Cardiovascular Issues. New York, Springer-Verlag, 1987, pp 249–283.
6. Fouad FM, Tarazi RC, Ferrario CM, Fighaly S, Alicandri C. Assessment of parasympathetic control of heart rate by a noninvasive method. Am J Physiol 1984; **246**:H838–H842.
7. Ibrahim MM, Tarazi RC, Shafer WH, Bravo EL, Dustan HP. Unusual tyramine responsiveness in idiopathic orthostatic hypotension. Med J Cairo Univ 1979; **47**:49–55.