Literature Briefs

Myron B. Laver, M.D., Editor

Literature Briefs were submitted by Drs. A. Boutros, T. Caldwell, G. Battit, M. Gold, M. Laver, and J. Pender. Briefs appearing elsewhere in this issue are part of this column.

Circulation

HEMODYNAMICS IN "BORDERLINE" HYPERTENSIVES Hemodynamic measurements were made in 77 "borderline" hypertensives and in 82 normal, healthy volunteers during changes in position, mild exercise, volume expansion with dextran, and autonomic blockade with propranolol and atropine. Borderline hypertension was defined as one diastolic blood pressure recording above 90 mm Hg within the year prior to study. No patient had congestive cardiac failure or funduscopic changes. Urine, intravenous pyelograms, and glucose tolerance tests were normal. Men 18 to 54 years old were studied; controls did not differ significantly in age from the borderline hypertensives. Cardiac output was measured by dye-dilution techniques and blood pressures were measured with intra-arterial cannulae. The peripheral resistance index was defined as mean blood pressure divided by cardiac index.

At rest, borderline hypertensives differed from controls in cardiac index (3.79 ± 0.10 vs. 3.31 ± 0.08 l/min/m^2), mean arterial blood pressure (100 ± 1.5 vs. 83 ± 1.1 mm Hg) and heart rate (76 ± 0.67 vs. 67 ± 1.1 beats/min), $P < 0.001$. At rest and recumbent, values for the peripheral resistance indices were the same in the two groups.

When the subjects changed to the sitting position, borderline hypertensives continued to have elevated arterial blood pressures (114.9 $\pm$ 1.7 vs. 95.3 ± 2.2 mm Hg, $P < 0.001$) but also developed significant increases in peripheral resistance index (38.1 ± 2.1 vs. 32.7 ± 1.2, $P < 0.05$). Cardiac indices were similar to those in control group.

During mild exercise, borderline hypertensives had lower cardiac indices than normals (5.3 ± 0.2 vs. 5.6 ± 0.21/min/m^2), significantly higher mean arterial pressures (124.9 $\pm$ 2.0 vs. 106 ± 2.1 mm Hg), and significantly higher peripheral resistance indices (24.8 ± 1.0 vs. 19.8 ± 0.7), $P < 0.001$.

After intravascular volume expansion with 5 per cent dextran (13 ml/kg), patients with borderline hypertension decreased their peripheral resistance indices less than did normal controls ($P < 0.05$).

Administration of propranolol, 0.2 mg/kg, with the subjects supine had little effect on arterial blood pressure, but did result in significant elevation of the peripheral resistance indices in borderline hypertensives, from 27.7 $\pm$ 1.1 to 37.4 ± 1.4 units ($P < 0.05$).

Atropine (0.04 mg/kg iv) given to controls and 18 patients, all of whom had received propranolol, caused significant decreases in the cardiac output in the borderline hypertensives (from 3.6 to 3.0 l/min/m^2, $P < 0.001$) but not in the controls. Decreases in output occurred simultaneously with significant increases in the peripheral resistance indices from 27.9 ± 1.8 to 34.0 ± 1.4 ($P < 0.01$). No increase in peripheral resistance appeared in the controls.

The authors conclude that in the borderline hypertensive patient at rest in the supine position cardiac output is higher than normal with normal peripheral resistance. Yet, with stimuli such as exercise or volume expansion the patient with borderline hypertension fails to lower his peripheral resistance to the same extent as the normal subject; with stimuli that normally increase peripheral resistance (sitting, treatment with propranolol), the borderline hypertensive will have a greater-than-normal increase in peripheral resistance. Moreover, with "chemical denervation" of the heart (propranolol and atropine) the borderline hypertensive has a marked increase in peripheral

resistance, a slight increase in blood pressure, and a considerable decrease in cardiac output. The authors suggest that in the borderline hypertensive cardiac output and peripheral resistance are adjusted to maintain a slight increase in blood pressure. There was no evidence of a hyperactive pressure response to any of various stimuli. (*Julius, S., and others: Relationship between Cardiac Output and Peripheral Resistance in Borderline Hypertension, Circulation 43: 382 (Mar.) 1971.*) EDITOR'S COMMENT: The anesthesiologist frequently treats patients with labile or borderline hypertension. The changes in cardiac output and peripheral resistance described for challenges to circulatory homeostasis such as volume expansion, change in position, or administration of atropine do not follow the pattern described for the normal adult volunteer. It is time for the anesthesiologist to begin the definition of patient physiology in terms of pre-existing disease. Normal responses provide a good baseline but are not always certain to keep us out of trouble.

Coagulation

CLOTTING OF PLASTIC CATHETERS Autopsy dissections in 55 patients who had indwelling subclavian catheters are reported. In 31 of these patients, cinefluoroscopic studies were carried out prior to dissection of the subclavian vein. The dissections revealed that circumferential fibrin sleeves had developed around each catheter studied. The catheters, made of either Teflon or polyethylene, had been *in situ* for various periods ranging from one to 76 days. One group of patients had subclavian catheters made of polyethylene coated with colloidal graphite, benzalkonium chloride, and heparin. The fibrin sleeves were still present, but smaller in length and thickness than those on the uncoated catheters. The authors suggest that special treatment of catheters can reduce thrombogenicity and that further studies should be conducted to minimize or eliminate the thrombogenic characteristic of indwelling venous catheters. (*Hoshal, V. L., Jr., and others: Fibrin Sleeve Formation on Indwelling Subclavian Central Venous Catheters, Arch. Surg. 102: 353–358, 1971.*) EDITOR'S COMMENT: This complication, associated with a technique that is gaining wide acceptance, has been described as "the pathology of progress." Documentation of this nature should stimulate improvement of materials allowed to reside within blood vessels for prolonged periods.

Endocrine Function

MYXEDEMA AND IMPAIRED CONSCIOUSNESS Impaired consciousness in myxedema may result from mechanisms other than simple depression of the metabolic state. An evaluation of nearly 100 cases reported from 1953 to 1963 indicates that four other factors may contribute to the depression of consciousness: hypoxia, hypercarbia, hyponatremia, and hypopituitarism.

Cerebral hypoxia may result from low cardiac output or severe congestive heart failure, both of which are common in myxedema and may contribute to depression of the level of consciousness. Myocardial infarction and pericardial effusion resulting in tamponade are rare causes of stupor or coma, but their sudden onset should raise the suspicion of a cerebral vascular accident.

Hypercarbia in myxedematous patients in coma has been reported, but present data are inadequate to establish its incidence. Impaired function of the respiratory center or of the respiratory muscles may account for the reduced minute ventilation and maximal breathing capacities, while obesity may increase the work of breathing. Death resulting from administration of morphine has been reported; respiratory arrest has been known to occur after small quantities of thiopental, and coma has followed barbiturate administration.

Hyponatremia is often present, and apparently results from water retention in spite of low serum osmolarity. Convulsions followed by postictal coma may occur. Therapy includes fluid restriction or the use of hypertonic saline solution, in addition to thyroid hormone replacement.

Hypopituitarism with secondary myxedema may cause hypoglycemia, coma, cardiovascular depression, and hypothermia. Until pituitary function can be evaluated, therapy with glucocorticoids is necessary.

The treatment of coma in the myxedematous patient includes therapy with thyroid hormones, but present experience is insufficient