

# Literature Briefs

Myron B. Laver, M.D., Editor

Literature briefs were submitted by Drs. L. Brand, L. Cooperman, B. Dalton, M. Gold, A. Goldblatt, J. Harp, L. C. Mark, H. Rackow, and G. Rockwell. Briefs appearing elsewhere in this issue are part of this column.

## Circulation

### CORONARY ARTERY BYPASS GRAFTS

Angiographic studies of 84 patients who had aortocoronary venous bypass graft operations revealed that of a total of 117 grafts, 82 grafts in 64 patients were patent and functioning well; nine grafts, although patent, functioned poorly; and 26 grafts were occluded. Selective coronary-artery angiography and left cine-ventriculography demonstrated changes when comparison was made with preoperative studies. Among those with functioning grafts (14 patients), substantial progress of disease was found in the arteries bypassed by the grafts. In contrast, those with occluded grafts had no important change in the bypassed arteries. Ventriculography showed improved contractility and decreases in end-diastolic pressures in 36 of 64 patients with patent functioning grafts; the remaining patients in this group showed little change, as did 15 patients with stenosed or occluded grafts. Five patients with occluded grafts had decreased contractility and elevated end-diastolic pressures. The statistical significance of these results is influenced by the fact that a third of the whole group was studied because of recurrence of symptoms. (Rosch, J., and others: *Aortocoronary Venous Bypass Grafts, Radiology* 102: 567-573, 1972.)

## CNS Function

### INCREASED INTRACRANIAL PRESSURE

Decerebration secondary to intracranial hemorrhage was caused in two patients by rupture of a cerebral aneurysm and in three by trauma. Decompression prior to operative evacuation of the hematoma was achieved with endotracheal intubation, hyperventilation at approximately 12 l/min (170 ml/kg/min) and administration of mannitol and dexamethasone in four of the five patients. The decerebrate

state (dilated pupils, rigidity) was successfully reversed in all five patients, and was corrected in the two patients with leaking aneurysms even before the start of operation. Postoperatively, all patients were fully conscious within 2 to 26 hours and ultimately regained normal intellectual and psychologic function. (Zervas, N. P., and Hedley-White, J.: *Successful Treatment of Cerebral Herniation in Five Patients, N. Engl. J. Med.* 286: 1075, 1972.)

## Renal Function

**RENAL TUBULAR ACIDOSIS** Renal tubular acidosis (RTA) is a syndrome of metabolic acidosis in which the extent of hydrogenion accumulation is out of proportion to the impairment of glomerular filtration and is associated with a paradoxical alkaline urine. Growth retardation is an early clinical sign. This report classifies RTA into proximal RTA and distal RTA.

The proximal form is associated with a low renal threshold for bicarbonate, together with a slight but continuous renal loss of bicarbonate amounting to 10-15 mEq/kg/day. Because of the bicarbonate loss, metabolic hyperchloremic acidemia and hypocarbia develop, while the urine remains alkaline. The treatment is 10-15 mEq/kg/day of alkali, with spontaneous recovery the rule. It occurs mainly in males. Excretion of H<sup>+</sup>, renal concentrating ability, and glomerular filtration are all normal.

The distal form is associated with a normal renal threshold for bicarbonate and is characterized by an inability to excrete H<sup>+</sup> or to concentrate urine. The normal H<sup>+</sup> intake amounts to 2-3 mEq/kg/day and in the absence of added alkali intake hyperchloremic acidemia develops, while the urine is alkaline and dilute. Polyuria and polydipsia with dehydration are secondary signs. Nephrocalcinosis often develops. The treatment is 2-3 mEq/kg/day of alkali on a permanent basis. Males and females are equally affected. (Nash, M. A., and others: *Renal Tubular Acidosis in Infants and Children. J. Pediatrics* 80: 738-748, 1972.)

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