

Literature Briefs

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Literature Briefs were submitted by Drs. G. Battit, T. Caldwell, R. Clark, B. Geffin, M. Gold, M. Laver, E. Lowenstein, H. Pontapidan, and S. Schneider. Briefs appearing elsewhere in this issue are part of this column.

CNS Function

CSF IN DIABETIC KETOACIDOSIS Cerebrospinal fluid and blood were studied in six patients with ketoacidosis before and four to nine hours after treatment, at the time neurologic improvement began. No alkali was included in the treatment regimen. Initial spinal fluid pH was normal or elevated in every patient, and decreased with treatment in four patients. Osmolality was significantly higher in cerebrospinal fluid than in blood before treatment; lactate was above normal initially and did not change with treatment. The concentrations of keto acids and glucose initially were lower in CSF than in blood and decreased with treatment at a rate consistent with slow equilibration. The authors speculate that administration of bicarbonate would aggravate the spinal-fluid acidosis that occurs with treatment, and could interfere with clinical improvement. They consider the increased osmolality before therapy to be a reason for caution against the excessively rapid correction of blood hyperosmolality. (Ohman, John L., and others: *The Cerebrospinal Fluid in Diabetic Ketoacidosis*, *NEJM* 284: 283-290, 1971.) **ABSTRACTER'S COMMENT:** Another situation in which correction of blood pH appears to be contraindicated.

Circulation

HYPOXIA AND CEREBRAL AUTOREGULATION "Cerebral autoregulation" used to describe two phenomena: 1) the capacity to maintain constant blood flow during changes in arterial perfusion pressure; 2) local adjustments in blood flow in response to tissue metabolic needs. Although losses of autoregulation in hypoxia and hypercapnia have been described, recent studies have dem-

onstrated preservation of autoregulation in response to pressure in brain areas affected by severe ischemia.

The inhalation of 6 per cent O₂-N₂ served as the hypoxic stimulus in 18 dogs. Cerebral blood flow (CBF) was measured while PaCO₂ was controlled by artificial ventilation. Cerebral arterial perfusion pressure was altered by three mechanisms: 1) pharmacologically, with intravenous Neo-synephrine; 2) by increasing intracranial pressure; 3) by hemorrhage and retransfusion of the animal's blood. The results indicated that autoregulation was not lost until PaCO₂ had been below 25 torr for 4-6 min. Autoregulation was maintained even after hypoxia had produced an increase in CBF and decreased cortical pH. This suggests that loss of autoregulation does not occur secondary to parenchymal acidosis or maximal vasodilatation. In response to increased arterial perfusion pressure autoregulation occurred after a transient increase in CBF, whereas autoregulation in response to reduced perfusion pressure appeared instantaneously, without an initial drop in CBF. (Kogure, K., and others: *Effects of Hypoxia on Cerebral Autoregulation*, *Amer. J. Physiol.* 219: 1393 (Nov.) 1970.)

Renal Function

CARBON DIOXIDE AND RENAL BLOOD FLOW This study was done in dogs under conditions of varying PaCO₂ produced by either diffusion respiration or ventilation with high concentrations of CO₂. Other investigators who have studied the effect of PaCO₂ on renal blood flow have not controlled PaCO₂ precisely in a range such as might be expected in living subjects. In general, the results have been confusing, some providing little evidence of renal vasoconstriction in hypercapnia, and others indicating that reduction in renal perfusion is part of a generalized vasoconstrictor response. The present study has attempted to resolve this problem.