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Cerebral Hemodynamic and Pathophysiological Alterations in Air Embolism. Javad Hekmatpanah, M.D., Professor of Neurosurgery, Pritzker School of Medicine, University of Chicago.

Cerebral air embolism was produced in 13 monkeys. Increments of 1-4 ml of air were injected in the internal carotid artery while blood and air circulation in the cerebral capillaries was observed and photographed through a microscope. Simultaneously, a quantitative measurement of cerebral blood flow was obtained with Xenon-133 and vital signs were monitored. Blood-brain barrier and histologic alterations in the brain were studied with vital dye and electron microscopy. One ml of air caused slowing of EEG. Only 2 ml of air caused marked EEG changes, bradycardia, irregular respiration, and varied degrees of hypotension. The air bubbles blocked numerous capillaries for periods of up to 30 min. and in some animals up to 60 min. Cerebral blood flow decreased as measured with Xenon-133. The air bubbles moved gradually with pulsations dispersing air into smaller vessels. Some vessels remained completely blocked. These alterations were more severe when a total of 4 ml of air were injected; EEG became almost flat and respiration stopped. Although artificial respiration was given and a few animals could be weaned off the respirator later, all animals died within 24 hours. Histological examinations revealed marked disturbance of blood-brain barrier in cortex and deep areas of the brain. Perivascular spaces increased, swelling of astrocytic processes occurred, cell membranes were torn and distorted, cellular cyttoplasm was ruptured. These findings might elucidate some of the neurological side effects seen in some cases of cardiovascular or carotid artery surgery.

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branes, namely those of endoplasmic reticulum and mitochondria. Chronic feeding of low levels of CPFA leads to inhibition of glucose-6-phosphatase activity and the formation of peculiar membrane profiles in the rough surfaced endoplasmic reticulum. Smooth surfaced endoplasmic reticulum on the other hand does appear to be affected as evidenced by the fact that the activity of the drug metabolizing enzyme system responsible for o-dealkylation of phenacetin remains at normal levels in face of long term feeding of CPFA. Chronic feeding of CPFA also significantly increased the binding of C134-afatoxin to nuclear DNA, RNA and protein of liver cells. In an effort to determine whether alterations of cell permeability were involved, studies with quinacrine hydrochloride as a fluorescent probe were undertaken; these indicate that CPFA renders rat liver cells more permeable following as short a period as 2 weeks of feeding 500 ppm of CPFA. This finding is further supported by the fact that CPFA inhibits Na+ - K+ activated ATP-ase activity of the plasma membrane of liver cells (Na+ - K+ ATP-ase, without ouabain) activity of plasma membrane of hepatocytes from CPFA fed rats 12.0 nm PO3/g protein/hr as compared to 20.9 nm PO3/g protein/hr for those from normal hepatocytes, and cause an increased Na+ and Ca2+ ion concentration and concomitant decrease in K+ and Mg2+ ions in liver tissue.

The cocarcinogenic effect of CPFA appears to be due to the additive effects of its ability to increase the permeability of liver cells and its stimulation of liver cell mitoses.

Colloid Osmotic Pressure and the Development of Pulmonary Edema. Christopher K. Zarins, M.D. and Richard W. Virgilio, M.D. Department of Surgery, The University of Chicago, Chicago, Illinois and Trauma Research Unit, Naval Regional Medical Center, San Diego, California. Sponsored by David B. Skinner, M.D.

Increased pulmonary capillary wedge pressure (PCWP) causes increased filtration force across the pulmonary capillary and may result in pulmonary edema. Decreased plasma colloid osmotic pressure (COP) also increases filtration force and it has been suggested that low COP or a decrease in the gradient between COP and PCWP can cause pulmonary edema. Forty-three patients were resuscitated with large volumes of colloid or crystalloid solution resulting in a wide variation in COP (8 to 28 mm Hg) and PCWP (0 to 30 mm Hg). The gradient COP-PCWP ranged from -11 to +23 mm Hg. All patients gained weight. Patients with the lowest COP gained most weight (+10.2 ± 1.2 Kg) and had marked peripheral edema but none had clinical or radiographic evidence of pulmonary edema. Pulmonary edema developed in two patients resuscitated with colloid solution. Both had normal COP and increased PCWP. Regressions of intrapulmonary shunt (Qs/Qt) on COP (y=21.37-0.291X) and COP-PCWP (y=18.01-0.275X) revealed neither COP nor COP-PCWP to be a predictor of pulmonary dysfunction as determined by Qs/Qt (p<1).

Plasmapheresis with maintenance of a normal PCWP in five baboons resulted in marked reduction in COP (to 5 mm Hg) and COP-PCWP (−1 mm Hg), weight gain (+3.5 Kg), asxies and marked peripheral edema. No pulmonary edema developed and intrapulmonary shunt and dynamic compliance remained normal. Pulmonary lymph flow increased seven-fold, pulmonary interstitial shunt was not detected (to 5 mm Hg), pulmonary lymph albumin flow increased two-fold and calculated pulmonary interstitial pressure increased by seven mm Hg.

Decreased COP or COP-PCWP gradient does not result in pulmonary edema if pulmonary capillary hydrostatic pressure is not elevated despite the development of edema elsewhere. When plasma oncotic pressure is low, the lung appears to be protected from edema by increased lymph flow, increased lymph albumin clearance to lower interstitial COP and increased pulmonary interstitial pressure.

Gastric Ulcers Treated by Vagotomy and Pyloroplasty. James R. Hines, M.D., Professor of Surgery; Robert E. Geurink, M.D., Assistant Professor of Surgery; Gerald T. Ujiki, M.D., Assistant Professor of Surgery; and Robert T. Gordon, M.D., Instructor in Surgery, all from Department of Surgery, Northwestern University Medical School.

Thirty-eight patients with a variety of gastric ulcers were treated by vagotomy and double pyloroplasty and were followed from six months to six years. The average follow-up was 3½ years. The primary reason for operation was pain in 13, bleeding in 18, perforation in 3 and obstruction in 4. Thirteen of the 18 bleeders were active, massive bleeders that required oversewing. Single ulcers were biopsied by endoscopy and again at operation. Twenty-five patients had single proximal ulcers, five had two or more ulcers and eight had prepyloric or channel ulcers. Eleven of the 38 patients had duodenal ulcers, four active and seven inactive. Truncal vagotomy and a wide double (buck wall) pyloroplasty was used in each patient. We feel that a large outlet is essential in the treatment of patients with gastric ulcers.

One patient (a Ca quadraplegic with multiple stress ulcers) (Continued on next page)