Literature Briefs

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Literature Briefs were submitted by Drs. C. M. Ballinger, N. A. Bergman, R. Boettner, A. Boutros, W. Boyd, D. R. Buechel, H. Cascorbi, R. B. Clark, M. Gold, J. Jacoby, L. M. Kitahata, W. Mannheimer, F. C. McPartland, D. Morrow, E. S. Munson, A. S. Paterson, J. W. Pender, H. Roe, L. J. Saidman, and A. D. Sessler. Briefs appearing elsewhere in this issue are part of this column.

Circulation

CARDIAC PERFORATION Perforation of the heart may be caused by cardiac catheters, transvenous pacemakers, or free-floating foreign bodies. There may be no symptoms from perforation, or there may be progressive pericardial tamponade with increasing venous pressure, enlarged heart shadow and muffled heart sounds. The pericardial sac may accommodate 200 to 1,500 ml of fluid. In many instances no treatment is required. If symptoms develop, needle aspiration or surgical exploration will be required. In 12,000 cardiac catheterizations, there were 96 known perforations. (*Lawton, R. L., Rossi, N. P., and Funk, D. C.: Intracardiac Perforation, Arch. Surg. 98: 213 (Feb.) 1969.*)

PULMONARY EMBOLECTOMY Records of 101 necropsies of patients who died of major pulmonary emboli were reviewed. Twenty-one died suddenly without symptoms being recorded. Twenty-six seriously ill from other diseases suffered gradual decline without a sudden change in status to identify an embolic catastrophe. Twenty-eight patients with symptoms survived more than an hour. Of these, seven had carcinomatosis, four were comatose, six had severe intra-abdominal disease prohibiting surgery, and in five the disease was unsuspected. Thus, only six of the 101 patients were considered subjects for embolectomy. Two ways to improve the potential salvage rate from this disease are: improve accuracy of diagnosis, and shorten time between symptoms and angiography. This, then, means that a pulmonary embolectomy team should be available 24 hours a day. (*Gifford, R. W., and others: Limitations in the Feasibility of Pulmonary Embolectomy, Circulation 39: 523 (April) 1969.*)

CEREBRAL BLOOD FLOW Regional cerebral blood flow (rCBF) was measured before and after implantation of a cardiac pacemaker in each of seven patients with complete atrioventricular block and Adams-Stokes syndrome. Pre-pacemaker total CBF/100 g/min averaged 40 ml compared with a control-group value of 51 ml. Mean rCBF in hemispheric gray matter was 60 ml (control 82 ml). Pacing increased heart rate from 39 to 72, cardiac output from 3.6 to 5 l/min, total CBF from 40 to 44 ml/100 g/min, and rCBF (gray matter) from 60 to 66 ml/100 g/min. In addition, the mean spectral frequency of the EEG increased. Changes in EEG and cerebral blood flow paralleled the clinical improvement which accompanied the pacemaker therapy. (*Sulg, I. A., and others: The Effect of Intracardial Pacemaker Therapy on Cerebral Blood Flow and Electroencephalogram in Patients with Complete Atrioventricular Block, Circulation 39: 487 (April) 1969.*)

CO_2 IN CEREBRAL ISCHEMIA The effects of inhalation of 5 per cent CO_2 on blood flow to ischemic areas of the brain were studied in 14 days. The middle cerebral artery on one side was ligated and blood flow to the normal and ischemic cortical areas was measured with fluorescein and infrared angiography and with a heated thermistor flowmeter. The expected maximal vasodilation of the ischemic areas in response to local hypoxia, acidosis and hypocarbia did not occur. Although inhalation of 5 per cent CO_2 decreased

pressures in the non-occluded middle cerebral artery, it caused an even greater decrease in pressure in the distal end of the occluded artery and *increased* flow to the ischemic area. Systemic l-norepinepherine increased both pressure and flow in the ischemic areas; the pressure remained elevated, but the flow returned to normal after ten minutes. This indicates that the vessels still had the capacity either to respond to local vasoconstrictor activity or to autoregulate flow in response to increased systemic pressure. No evidence of intercerebral stealing of blood from ischemic areas by increased flow to the nonischemic areas in response to elevated P_{CO_2} was found. (*Kogure, K., and others: Effects of Changes in Carbon Dioxide Pressure and Arterial Pressure on Blood Flow in Ischemic Regions of the Brain in Dogs, Circ. Res. 24: 557 (April) 1969.*) ABSTRACTER'S COMMENT: The conclusion that hypercarbia improves circulation to the ischemic canine brain is directly opposite of that of Soloway (ANESTHESIOLOGY 29: 975, 1968), who found that hyperventilation to a P_{CO_2} of 25 mm Hg markedly decreased the size of infarcts produced by tying a middle cerebral artery and the ipsilateral internal carotid artery between the posterior communicating and anterior cerebral arteries. Obviously, the question of the beneficial vs. harmful effects of increased or decreased CO_2 on the cerebral circulation remains unanswered.

VENOUS CATHETERIZATION Complications recorded from the use of indwelling venous catheters include phlebitis, thrombosis, embolism, and sepsis. Long-term tolerance to venous catheters without complications was achieved in 23 patients. The recommended procedure includes selection of a large vein with large-volume blood flow, such as the jugular or subclavian, for insertion of the catheter. The skin was shaved, defatted with ether or acetone, and scrubbed with two per cent iodine tincture. Sterile towels and gloves were used and the catheter was fixed to the skin with a silk suture. Neosporin ointment was applied to the exit site, and the wound was covered with a sterile dressing. Skin prep was repeated every three days. Catheters remained in the vein an average of 24 days, and in all cases bacterial cultures were negative. (*Wilmore, D. W., and Dudrick, S. J.: Safe Long-term Venous Catheterization, Arch. Surg. 98: 256 (Feb.) 1969.*)

SPLANCHNIC POOLING IN SHOCK The role of pooling of blood in the splanchnic vascular bed in the development of irreversible hemorrhagic shock is controversial. To study the problem, irreversible hemorrhagic shock was induced in splenectomized dogs with the use of prolonged hemorrhagic hypotension, followed by reinfusion of blood. Changes in abdominal and thoracic blood contents were measured by injection of 51-chromium-labeled erythrocytes, and monitored over the shielded abdomen in nine dogs and over the shielded thorax in four other dogs with scintillation crystal detectors. Average abdominal counts immediately after reinfusion were at the prehemorrhagic levels. Counting rate then increased gradually to 113 per cent of control, whereas cardiac output and arterial pressure declined. After reinfusion, thoracic counts returned to control and then decreased to 88 per cent of control. The correlation between the post-reinfusion decrease in cardiac output and the increase in abdominal counts was highly significant. In several animals, however, the decreases in cardiac output were not in phase with the changes in counting rate. It appears, therefore, that some degree of splanchnic pooling of blood is usual in irreversible shock, and that this is sometimes, but not always, associated with decreased cardiac output. (*Hirshfeld, J. W., Jr., and Fell, C.: Changes in Regional Blood Content during Hemorrhagic Shock in Dogs, Amer. J. Physiol. 216: 380 (Feb.) 1969.*)

HYPOVOLEMIC SHOCK Anesthetized dogs were bled sequentially to remove 75 per cent of the erythrocyte mass. Sufficient volume of lactated Ringer's solution was administered to maintain arterial and venous pressures and urinary output at normal levels. The initial blood removal (36 per cent) was replaced with five times its volume of lactated Ringer's solution. The second blood removal (equivalent to 24 per cent of the original erythrocyte mass) was replaced with eight times its vol-