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