BRIEFS FROM THE LITERATURE

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Briefs were submitted by Drs. Peter P. Bosomworth, M. T. Clarke, Martin Helrich, J. J. Jacoby, F. C. McPartland, S. J. Martin, R. E. Ponath, William Rabenn, R. W. Ridley, and H. S. Rottenstein. Briefs appearing elsewhere in this issue are a part of this column.

CIRCULATION AND ANESTHESIA Tissue survival is most likely in the presence of drugs which interfere minimally with the responses of the fine circulation to their normal stimuli (including catecholamines). Ether, halothane, cyclopropane, and thiopental share some similarities in their effect on circulation and yet have many different effects. The significance of present, superficial observations must be conjectural. Deeper understanding awaits better methods and measurements. Maintenance of near normal levels of myocardial contractility during cyclopropane anesthesia depends on the high level of sympathetic tone which the drug elicits. Ether seems to "sensitize" the carotid sinus mechanism and to act in the central nervous system in some manner which provokes a rather general increase in sympathetic nervous activity. Sympathetic nervous activity is relatively weak during halothane anesthesia and the effectiveness of whatever activity does exist is reduced by peripheral actions of the anesthetic. The salient hemodynamic feature of thiopental anesthesia is that arterial pressure is well maintained while cardiac output and tissue blood flow are reduced. This implies increased vascular resistance. (*Price, H. L.: Circulatory Actions of General Anesthetic Agents and Homeostatic Roles of Epinephrine and Norepinephrine in Man, Clin. Pharmacol. Ther. 2: 163 (Mar.–Apr.) 1961.*)

BLOOD VOLUME The major determining factor in the regulation of blood volume appears to be the osmotic pressure of the extracellular fluid. The regulation of the extracellular fluid volume seems to depend on sensory information from volume receptors in the low and high pressure sides of the circulation. The extracellular fluid volume is corrected by changes in the circulating blood volume which in turn results from changes in urinary water and electrolyte output. The low pressure receptors control renal water output by varying the level of secreted antidiuretic hormone. The high pressure system receptors seem to regulate the sodium output by varying the production of aldosterone. Uncertainty exists as to the role of additional regulation of urinary sodium excretion by a natriuretic hormone secreted in the midbrain and/or renal hemodynamic control of the tubular concentrating mechanism. (*Pearce, J. W.: A Current Concept of Regulation of Blood Volume, Brit. Heart J. 23: 66 (Jan.) 1961.*)

IRREVERSIBLE SHOCK A series of 105 patients who died in a state of irreversible shock after surgical operation was studied. In 99 patients (94.3 per cent) a definite etiologic factor could be found either on a clinical or on a pathologic basis. Hemorrhage was the underlying cause in 32 patients, a site of continuing hemorrhage being found at postmortem examination. It is suggested that failure to remove an underlying initiating cause is the chief factor in the lack of response to treatment in "irreversible shock" following surgical operation, and that a significant number of such deaths may be prevented by surgical intervention at the optimal time. (*Davis, H., and others: "Irreversible" Shock Following Surgical Operation in Man, West. J. Surg. 69: 1 (Jan.–Feb.) 1961.*)

SHOCK Two hours of hemorrhagic shock so weakens the anti-bacterial defense that gram negative bacteria, as well as gram posi-