Physiological Pharmacology—A Comprehensive Treatise. Vol. III, The Nervous System, Part C. EDITED BY WALTER S. ROOT AND FREDERICK G. HOFMANN. Pp. 519, with illustrations and tables. Cloth. $21.00. Academic Press, Inc., New York & London, 1967.

Physiological Pharmacology—A Comprehensive Treatise. Vol. IV, The Nervous System, Part D. (Same author and publisher as above.) Pp. 535, with illustrations and tables. Cloth. $23.00.

These two volumes complete the section on the nervous system in this series. Both are devoted to the autonomic nervous system and give exhaustive treatment of drugs and the physiologic changes associated with these. Bibliographies are comprehensive.

Wright's Veterinary Anaesthesia & Analgesia Sixth Edition. BY L. W. HALL. Pp. 480, with illustrations and tables. Cloth. $11.00. The Williams and Wilkins Co., Baltimore, Md., 1966.

While this revision is devised for the veterinarian who wishes to specialize in anesthesia, the basic applications of anesthetics for animals will be a ready reference for laboratory workers, including anesthesiologists.

The Human Heart: The Layman's Guide to Heart Disease. BY BRENDAN PHIBBS. Pp. 253, with illustrations and tables. Cloth. $4.95. The C. V. Mosby Co., St. Louis, 1967.

A simple presentation designed to help the physician instruct his cardiac patient in the pathology, etc., of his disease.

Clinical Pathology of the Serum Electrolytes. BY F. WILLIAM SUNDERMAN AND F. WILLIAM SUNDERMAN, JR. Pp. 446, with illustrations and tables. Cloth. $24.50. Charles C Thomas, Springfield, Ill., 1966.

General reference work.

Surgery

CIRRHOSIS Hepatic cirrhosis may produce significant alterations in pulmonary and cardiovascular function. Hyperventilation with resultant respiratory alkalosis is a common finding, as is arterial oxygen desaturation. The degree of the former roughly parallels the severity of the liver disease, whereas the degree of the latter does not correlate well with the severity of the cirrhosis. Pulmonary function studies show an increase in physiologic deadspace despite an increase in minute volume. Alveolar-arterial oxygen tension differences indicate significant shunting. Other blood studies show a low plasma bicarbonate, increased *p*H, and a slight decrease in base excess. The mechanism of the hyperventilation is not clear. It probably is related to increased blood levels of ammonia and various amines, although there is poor correlation between the blood ammonia level and the degree of hyperventilation. The decrease in buffer base may sensitize the chemoreceptors to hydrogen ions, thereby increasing the respiratory drive. The decrease in arterial oxygen saturation is partially due to some shunting of blood directly to the pulmonary veins from periesophageal and mediastinal veins as a consequence of the portal hypertension. Pulmonary arteriovenous shunting probably is an even more important factor. Peripheral vasodilation and increased peripheral blood flow commonly occur in cirrhosis and tend to cause an increase in cardiac output. Total blood volume is increased, largely due to increased plasma volume. A sustained increase in blood volume and cardiac output may lead to cardiac hypertrophy. Degenerative changes in the heart muscle also occur. (*Bashour, F. A., and others: Circulatory and Respiratory Changes in Patients with Laennec's Cirrhosis of the Liver, Amer. Heart J. 74: 569 (Oct.) 1967.*)

ABSTRACTOR'S COMMENT: The pulmonary and circulatory dysfunctions described above are in addition to the more well-known problems of the cirrhotic (*i.e.*, ascites, hypoproteinemia, coagulation problems, poor salt tolerance, poor drug detoxification, etc.), making him an even poorer anesthetic risk than previously thought.