bin deficiency and shock, it would be inferior.

R. B.

WIGGERS, CARL J.: *The Mechanism and Nature of Ventricular Fibrillation.* Am. Heart J. 20: 399 (Oct.) 1940.

Ventricular fibrillation was first described by Ludwig and Hoffa in 1849. The author has a series of 400 records of ventricular fibrillation in dogs with with only one recovery. The fibrillation process is an evolution of changes from the moment of inception until it ceases completely, within thirty to forty-five seconds. The phenomenon is caused by a reentry of circulation wave fronts which involve smaller and smaller blocks of myocardium, each of which develops an independent excitation. Anoxia results in slowed conduction and a decrease in vigor of contractions. The results of these changes cause in succession what the author calls the undulatory, convulsive, tremulous and atonic stages of the evolution of ventricular fibrillation. Two paragraphs are devoted to a theory which best explains the phenomenon.

C. P. W.

WIGGERS, CARL J.: *The Physiologic Basis for Cardiac Resuscitation from Ventricular Fibrillation.* (Method for Serial Defribillation.) Am. Heart J. 20: 413 (Oct.) 1940.

A number of procedures are reviewed for the restoration of normal beats, and the physiological conditions which seem to determine success or failure are analyzed. The author has devised and described a more successful form of countershock which is called serial defibrillation. By the use of this method, since September 1939, he has been able to revive 327 fibrillating hearts out of 328 attempts. A 15 Kg. dog was revived 41 different times. It is essential that the thorax be open and that the heart be immediately stimulated when ventricular fibrillation occurs in order to attain success. The difficulties encountered in reviving human hearts are analyzed. It is concluded that revival of the human heart, although not impossible, for the present is impracticable. It is suggested that the discovery, through research, of means of rendering the ventricles less sensitive to agents which cause fibrillation, is the most likely solution to the problem.

C. P. W.

FOLLIS, R. N., JR.: *The Coronary Arteries in Relation to Sudden Death During Anesthesia.* Bull. Johns Hopkins Hospital 67: 211–216 (Sept.) 1940.

"Sudden death during anesthesia for surgical procedures has fortunately been a rare event. Where it has occurred, the surgeon, anesthetist, and pathologist frequently have been unable to determine the exact cause of exitus. Recently at the Johns Hopkins Hospital there have been three cases of sudden death on the operating table when nitrous oxide, oxygen and ether were being administered. Because of the positive findings at autopsy, it was felt worthwhile to record them and draw attention to the extremely important role the state of the coronary arterial system may play in leading to sudden death during anesthesia.

Case I.—A 45 year old colored male complained of pain in the pit of the stomach for four days. B.P. 115/70, P. 100, T. 100.2. It was thought he had acute cholecystitis. Premedication consisted of morphine 8 mg. and atropine 0.6 mg. thirty-five minutes before induction. Nitrous oxide and oxygen was used for induction, followed by drop ether. At this point, the patient took two or three smothered breaths and stopped breathing before any incision had been made. The heart continued beating for two minutes.