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.

Case II.—A 35 year old colored male complained of pain associated with indiscretions in eating. B.P. 122/82. Preoperative medication consisted of 0.4 Gm. nembutal and 0.6 mg. of atropine. Anesthesia was induced and carried out with nitrous oxide and oxygen together with coeliac block. The pyloric end of the stomach was resected and a gastro-enterostomy was about to be performed when respirations ceased. It is not known whether pulse or respirations stopped first.

Case III.—A 43 year old colored male complained of cramping abdominal pains, etc. He stated that for two years he noted pains radiating down the left side of the neck and arm with some precordial oppression. It was found that he had a positive Wassermann. B.P. 160/108. He was operated for an acute appendicitis. He was given morphine 10 mg. and atropine 0.6 mg. Anesthesia was induced with nitrous oxide and oxygen. Afterwards, ether was employed. Twenty minutes after commencement of operation, respirations were three a minute; pulse 88. It was not known whether the heart or respirations stopped first.

In each of the first two cases, the orifice of the right coronary artery was very small in proportion to the main right coronary artery in its proximal portion. Another important finding was the great difference in size between the left and right main coronary arteries. In both cases the former vessel was twice the size of the latter. In the third case there was extreme coronary obstruction caused by arteriosclerosis as well as stenosis of the right coronary artery by syphilis.

It seems logical to assume in these three cases these changes were responsible for death due to heart failure. It seems not unlikely that there was anoxemia of the myocardium brought on by diminution in oxygen saturation of the blood because of the nitrous oxide, together with the probable insufficient coronary flow at the time. This would lead to cardiac insufficiency which would reflect itself on the respiratory center and in this manner the respirations would cease.

R. B. S.

QUERIES AND MINOR NOTES: *Local Anesthesia in Infected Area.* J. A. M. A. **115**: 1393 (Oct. 19) 1940.

Question.—"Is it good practice to inject a local anesthetic directly into a finger infection for surgery? Is nerve block considered safe in treating finger infections?"

Answer.—"It is generally regarded as poor practice to inject a local anesthetic directly into an infected area, particularly a finger, in preparation for a surgical therapy. General anesthesia is preferable. However, there is some weight of authority on the other side of this question. . . . A nerve block is less hazardous than infiltration of the inflamed area but is more hazardous than a general anesthesia provided the latter is given by an expert. It would seem that the safest rule is never to inject local anesthesia into an inflamed area, and to use nerve blocks only when general anesthesia is dangerous or unavailable."

J. C. M. C.

WHITE, J. C.: *Technique of Paravertebral Alcohol Injection. Methods and Safeguards in its Use in Treatment of Angina Pectoris.* S. G. & O. **71**: 334 (Sept.) 1940.

As a result of observations on 63 patients who have received 85 paravertebral injections for intractable cardio-aortic pain since 1924, and follow-up on patients after upper thoracic sympathectomy, the author is convinced that, "pain arising from all varieties of coronary and valvular disease, as well as from syphilis of the aortic arch,