"The transmission of disease by means of transfusion must be considered as a type of transfusion reaction. . . . Every attempt to rule out the presence of syphilis, malaria, and marked degrees of allergy in the donor, must be made. Severe hemolytic reactions are practically always caused by errors in the technic of grouping or cross matching. . . . Fever reactions with or without chills are fairly common. The importance of strict cleanliness and of freshly distilled water has already been stressed. Allergic reactions occur in 3 to 5 per cent. of all transfusions. Anaphylactic shock may prove fatal, and several deaths have been reported following the use of the same donor on repeated occasions. It is wise to use fasting donors to prevent the transfer of certain proteins to which the recipient may be sensitive. It is dangerous to use autotransfusions, such as from a ruptured liver or spleen, and this is particularly true in the case of the spleen. Defibrinated blood is very toxic and should not be used."

J. C. M. C.

BULL, DAVID, AND DREW, CHARLES R.: *Preservation of Blood.* Ann. Surg. 112: 498–501 (Oct.) 1940.

A summary of recent laboratory studies on preserved blood.

1. Red cell count remained unchanged for 30 days with heparin or anticoagulant; and 15 days with 0.3 per cent. sodium citrate.

2. Red cell diameter decreased after 35 days.

3. Survival of red cells after transfusion: studied by means of specific M and N factors which showed that cells of fresh blood survived 95 days in recipient; 3 day old blood, 80 days; 10 day old, 60 days; 14 day old, 20 days.

4. White cell count dropped 50 per cent. in 24 hours.

5. Thrombocytes fell rapidly.

6. R.B.C. Fragility Tests gave poor end points but it was apparent that the cells were less resistant on the 10th day than on the first.

7. Prothrombin showed a prompt initial fall to 50 (by the Quick Test) and then remained almost constant at this level over a long period.

8. Electrolyte — potassium diffuses rapidly from R.B.C. into serum for first few days and then more slowly to equilibrium. This diffusion is increased (1) by shaking, particularly of old blood (transportation should therefore be done while fresh), (2) by increasing the area of interspace between cells and supernatent serum, and (3) by ammonia concentration. This latter rises rapidly, within the first few minutes as the blood is exposed to air and then remains constant until after the 4th day. It is believed to increase cell permeability and thereby diffusion of potassium. If the blood is drawn bellow CO_2 ammonia nitrogen concentration is left low and diffusion of sodium and potassium is retarded.

Tolerance for potassium is increased when given slowly and decreased when given rapidly. Therefore, in shock and hemorrhage where large amounts of blood must be given rapidly, fresh blood should be used, especially if excretion is poor, or serum potassium already high.

Cadaver blood is not suitable for preservation because of much higher concentration of ammonia nitrogen and consequent rapid diffusion of Na and K ions.

Placental blood behaves much as does adult blood and appears to be a suitable source.

The outstanding changes of clinical interest taking place in stored blood are the loss of white cells and platelets, increase in plasma potassium and decrease in prothrombin. For most purposes it should give results comparable to fresh blood; for infection, prothrom-

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.