

In previously published work¹⁵ we reported that we have indisputably demonstrated that the lymph vessels of the dental pulp can be injected, not only in a retrograde direction by way of the gingiva, as Schweitzer did, but also by forcing the injection mass through the tooth directly into the exposed pulp tissue. The first investigation was limited to a study of the lymph supply of the pulp. The purpose of the present work was to follow the course of the outflowing vessels, to examine the extent of their communications with neighboring tissues, and to study their course in the mandibular and infra-orbital canals: in short, to fill out some gaps left in Schweitzer's results.

ABSTRACT OF DISCUSSION

DR. EUGENE S. TALBOT, Chicago: Twelve years ago Dr. Latham and I did some work on dental pulp, but not in relation to the lymph channels. We were astonished at that time because of the large number of abscesses that form in the dental pulp. It is almost impossible to believe that abscesses form in the dental pulp without causing any pain, and later heal up, which naturally would show that there must be lymphatics connected with the dental pulp. It was not until Dr. Rosenow showed how the streptococcus travels through the blood stream into different parts of the body that it was fully explained how these germs pass through the ends of the roots into the dental pulp and form abscesses. We have on record one case, which was a wonderful illustration of the abscess theory. This pulp shows an abscess forming, another one healing and one practically ripe with the pus inside of it, showing the different changes that had taken place from the time of infection to the healing of the abscess in the dental pulp. If one makes a study of the dental pulp with a view to looking into this abscess formation, he will find a great many scars in the dental pulp where abscesses have formed and healed.

DR. EDWARD H. HATTON, Chicago: These pathologic changes which are found about the gingivae and in the regions adjacent to these pus pockets can be explained in no other way than by the presence of lymphatics in the periodontal membrane leading down along the teeth and into the pulp cavity.

DR. HERBERT A. POTTS, Chicago: Aside from the histologic and anatomic interest of these studies, the demonstration explains many of our brilliant successes and many of our failures in the induction of local anesthesia. Two years ago Dr. Noyes told us the difficulty by which these injections were attended. I am sure that most of my success is due to the technic of the injection itself. Only by slow, steady injection do you obtain results.

DR. FREDERICK B. NOYES, Chicago: I think you will find that this work has a bearing not only on local anesthesia but on the whole realm of dental pathology, especially the destructive inflammations which have their origin in the gingival spaces.

15. Noyes, F. B., and Dewey, K. W.: A Study of the Lymphatic Vessels of the Dental pulp, *Dental Cosmos*, 1917, **59**, 436.

Reeducation of French War Cripples.—It was recently estimated that France had 300,000 disabled soldiers, with a monthly increase of between 6,000 and 7,000. They are cared for in national centers of physiotherapy, of which there is one for each of France's twenty military regions. To these centers are attached schools or workshops which the cripples may attend for purposes of reeducation. Independent of the centers, there are also civil schools for vocational reeducation, supported by both public and private funds; and finally there is the National Institute near Paris under the joint direction of the Ministries of War and of the Interior, with a well equipped military hospital, together with workshops and dormitories. Almost universally the "boarding-in" system prevails, that is, the soldier lives at the school and is under its direct discipline during his period of training.

FURTHER EXPERIMENTAL STUDY OF SURGICAL SHOCK *

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A review of the results obtained in the extensive amount of work that has been done on the problem of shock, both experimentally and clinically, makes the point I have repeatedly emphasized particularly clear, namely, that the condition the surgeon calls shock may be due to a variety of causes.¹ The variation in the primary etiologic factor in a condition in which identical clinical symptoms occur is probably the cause of the more or less contradictory conclusions arrived at by investigators, both experimentally and clinically.

The value of experimental work on surgical shock is certainly decreased by the fact that it is impossible to reproduce the experimental environment, and the condition itself, in a manner identical to its clinical manifestation. From the results of a large series of experiments I am forced to conclude that the experimental investigator approaches, in only a few specific types, the condition that the surgeon studies clinically.

The future work on the subject termed shock will, in all probability, not concern itself with the condition as an entity, but with the different possible etiologic factors that could produce the clinical picture the surgeon has in mind. Two excellent examples of this form of research on the problem have recently been presented. They are the relation of fat emboli to conditions diagnosed as surgical shock, as shown by Bissell² and by Porter,³ and wound shock of Cannon⁴ and his coworkers. While in neither case have all the factors involved been explained, the positive fact that cases which the surgeon diagnoses as shock may be classified is of the greatest importance.

There is, I believe, one general statement that can be made to include all cases clinically diagnosed as shock, namely, that the fundamental cause of death in each instance is a failure of physiologic compensation. From the therapeutic standpoint one of the most important problems to solve in connection with shock is to determine why some persons fail to compensate for the various procedures to which they are submitted.

I have discussed in a previous paper⁵ the effect of the anesthetic in relation to shock, but the anesthetic is of so much importance, both in experimental and in postoperative shock, that its effect should be emphasized. Recently, while performing some experiments, I found that a low blood pressure was produced invariably within an hour or two after the beginning of anesthesia, regardless of the experimental procedures employed. These results were proved to be due to impure ether. Careful experiments in anesthetic control should always be performed before it is con-

* From the Mayo Clinic.

* Read before the Section on Pathology and Physiology at the Sixty-Ninth Annual Session of the American Medical Association, Chicago, June, 1918.

1. Mann, F. C.: The Peripheral Origin of Surgical Shock, *Bull. Johns Hopkins Hosp.*, 1914, **25**, 205-212.

2. Bissell, W. W.: Pulmonary Fat Embolism—A Frequent Cause of Postoperative Surgical Shock, *Surg., Gynec. and Obst.*, 1917, **25**, 8-22.

3. Porter, W. T.: Shock at the Front, *Boston Med. and Surg. Jour.*, 1916, **175**, 854-858.

4. Cannon, W. B.: A Consideration of the Nature of Wound Shock, *THE JOURNAL A. M. A.*, March 2, 1918, pp. 611-617.

5. Mann, F. C.: Shock During General Anesthesia, *THE JOURNAL A. M. A.*, Aug. 4, 1917, pp. 371-374.

cluded that an experimental procedure produces the symptoms of shock.

THE RELATION OF THE NERVOUS SYSTEM TO SHOCK

The relation of the nervous system, as a primary agent, to the condition which the surgeon diagnoses as shock is not clear. It is quite probable that the nervous system is a primary etiologic factor in some cases, particularly in those in which an anesthetic has not been employed. There are no experimental data extant in which such a relationship has been proved beyond a doubt. The results of numerous experiments that I have performed under light ether anesthesia have been, with the few exceptions previously recorded, uniformly negative. It certainly is possible to stimulate, either electrically or mechanically, one or all of the major nerves going to the limbs, for example, the sciatic and brachial plexus of a dog under constant surgical ether anesthesia, for as long as four hours, without producing the condition of shock. It should be emphasized that the marked fluctuation in respiration and blood pressure that occurs following such stimulation is not shock, and shock can be said to have occurred only when the cardinal signs of the condition are present at the end of the period of stimulation. In my experiments this has not occurred, but both respiration and blood pressure have quickly returned to approximately the normal condition. The same phenomenon follows section of the major nerves. Whether or not much reaction follows such a procedure depends mainly on the depth of the anesthesia. Under light anesthesia, section of the sciatic nerves and the brachial plexus produces marked changes in respiration and blood pressure; when deep anesthesia is employed, section of these nerves may produce only the slightest response in respiration or blood pressure. In either case shock does not follow section of the nerves. We have observed animals which, under light anesthesia, have had a normal blood pressure eight hours after section of the major nerves to each limb (Fig. 1). I am inclined to believe that, in most instances in which the primary factor is the nervous system, the cause will be found to be of the nature of inhibition, as held by Meltzer.⁶

Under only two conditions have I been able to produce death by nerve stimulation. One condition was produced by stimulating the nerve fibers that inhibited respiration when the animal was under deep anesthesia. Ether anesthesia seems to depress, and, when the tension is great enough, abolishes all respiratory reflexes

except one before respiration ceases. The reflex that it does not abolish before the respiratory center fails is the one that inhibits respiration. Instead of ether depressing this reflex, it is quite common for its action to be increased. Under deep etherization it is possible, in many instances, actually to kill the animal by prolonged stimulation of nerve fibers that inhibit respiration. The stimulation of some nerves, as the central end of the vagus and the superior laryngeal, usually inhibits respiration for a short period. As the stimulation is continued, however, respiratory movements soon return, owing either to a decrease in the reflex or, what is more probable, to an increase in the chemical stimulation of the nerve center. It is rarely possible, under light surgical anesthesia, to inhibit respiration by the stimulation of these nerves for a long enough period to jeopardize the life of an animal. As the ether tension is increased, the length of time the respiratory movements are inhibited is prolonged. Finally, in a large number of cases, under deep etherization, respiration fails to return, and blood pressure quickly falls, death ensuing. In other cases, while respiration is always inhibited for a time, recovery occurs. In the earlier experiments the blood pressure

was usually decreased, as much as one-half the normal pressure, before respiration could be inhibited long enough to produce death. However, it was found that by cautiously increasing the ether tension, many animals could be killed while the blood pressure was practically normal. The method of administering the anesthetic does not seem to be a factor in producing death. In most of our experiments the Connell⁷ apparatus was used, in some in-

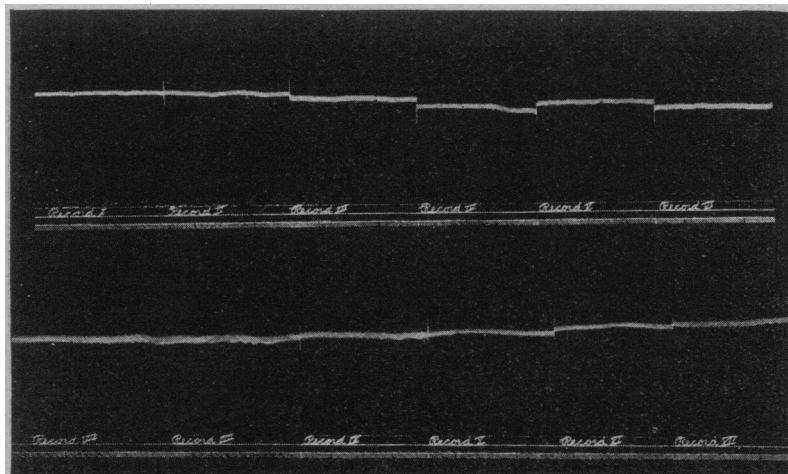


Fig. 1.—Kymograph record illustrating (1) an anesthetic control experiment and (2) the effect of section of the major nerves to each limb; Record I, normal blood pressure (140); Record II, immediately after exposing the sciatics and brachial plexuses; Record III, immediately after section of both sciatics and brachial plexuses. Each succeeding record was taken at intervals of an hour; thus, Record XII was taken nine hours after section of the nerves, and the blood pressure was 145.

stances a modification of McGrath's⁸ method, while in still others only the cone method was used. While it is possible that death in these cases may be due to factors other than the inhibition of respiration and asphyxia, there is no evidence to show it. I have never been able to obtain this result by stimulating nerves that did not inhibit respiration. Death will not occur while insufflation or artificial respiration is maintained, if the ether tension is not above that compatible with life. Death under deep etherization has not been produced except in connection with inhibition of respiration. The process producing death in such experiments seems to be as follows:

For some unknown reason, ether does not abolish reflexes that inhibit respiration as long as the respiratory center responds. Under deep etherization the threshold of the cells of the respiratory center is

7. Connell, K.: An Apparatus—Anaesthetometer—for Measuring and Mixing Anesthetic and Other Vapors and Gases, *Surg., Gynec. and Obst.*, 1913, **16**, 245-255.

8. McGrath, B. F.: Anesthesia in Surgical Research, *Surg., Gynec. and Obst.*, 1914, **18**, 376-377.

6. Meltzer, S. J.: The Nature of Shock, *Arch. Int. Med.*, July, 1908, pp. 571-588.

greatly increased to the chemical stimuli. A point is thus reached at which the center will respond to the inhibitory reflex, and not to the increased carbon dioxide in the blood. At this time the stimulation of inhibitory fibers will produce death, and, owing to the deep etherization, quite quickly in many instances. Attempts have been made to produce the same result by both methods of stimulating the nerves other than by electricity or deep etherization. To date, however, all attempts have failed (Fig. 2).

It has been thought for a long time that inhibition of the heart, due to stimulation of the vagus, might produce death in mammals, and it has also been suggested that a reflex producing inhibition of the heart might also end fatally. In a few instances in this series of experiments such a result seems to have been produced. However, to the present time, death, owing to apparent reflex inhibition of the heart, has not been produced by stimulation of any nerve except the superior laryngeal, although it would seem that the central end of one vagus with the other intact would be effective. As this reflex disappears under deep etherization, it appears that death could occur only by reflex inhibition of the heart under light anesthesia. Such was the case in these experiments (Fig. 3).

What practical bearing such experiments have on the shock problem cannot at present be stated. Death is produced so suddenly and so strikingly under these conditions that it seems highly probable the same thing has occurred in man. It is quite possible that deep etherization and inhibition of respiration during the time in which operations are being performed in the region of the neck, axilla and diaphragm (regions in which traumatic procedures are prone to produce stoppage of respiration) are responsible for some of the sudden deaths on the operating table that the surgeon has diagnosed as due to shock.

THE RELATION OF FLUID VOLUME TO SHOCK

The conception that in most of the cases which the surgeon diagnoses as shock the patients are in a pathologicophysiological state, in which the cause of the symptoms is a loss of circulatory fluid, has been adversely criticized, because of the clinical distinction that is made between hemorrhage and shock. This has been due, I believe, to a failure to consider the fundamental likenesses and differences between the two conditions.⁹ The symptoms of this form of shock and of hemorrhage are, in the main, due to the loss of circulatory fluid. In general, after hemorrhage, the vascular system is capable of functioning, and the mechanism controlling fluid volume is able to at least partially compensate for the loss. In the condition of shock, however, not only is there a loss of circulatory fluid, but the mechanism which controls fluid volume is also, and possibly primarily, greatly impaired. That

is the reason why, in the former condition, intravenous injection of salt solution is of distinct value, while even the so-called colloidal solutions do not remain long in the circulation in the latter condition.

Janeway and Jackson¹⁰ have shown that a circulatory failure, which presents the typical signs of shock, may be produced in dogs by a partial occlusion of the inferior vena cava at its point of entrance into the thorax. This has been corroborated by other investigators.¹¹ It seems that, for the most part, the result is due to the effect of the occlusion on the portal circulation and the liver, because it is well known that ligation of the inferior vena cava at a point just below the entrance of the hepatic veins is a perfectly safe surgical procedure in most dogs. The collateral venous return is such that a ligature thus applied does not produce any of the symptoms of shock. The ligation of the portal vein will always cause death in a few hours. Even partial occlusion of this vein, as sometimes occurs in a badly made Eck fistula, will produce death in a couple of days.

In a recent series of experiments, I have attempted to determine the relation of the volume of capillary and venous beds to the signs of shock. Only a brief preliminary report can be made of these experiments.

The method consisted in including in a strong ligature all the structures to each limb except the major

artery. In this manner the major artery was allowed to pump blood into the limb, from which all venous and lymph return was obstructed. The results of a sufficiently large series of experiments are in general agreement. The first effect of such a procedure is a slight and ordinarily transient rise in blood

pressure. The blood pressure, as a rule, soon decreases and at the end of two hours it has only about half its initial value. At this time the animal generally exhibits the signs of shock. If the ligatures are then removed, recovery usually takes place. When the ligatures are left on for a long period of time, there may be an initial rise in pressure following their removal, but it subsequently decreases. When the ligatures have been applied for a very long period, the removal produces a further drop in blood pressure, and eventually, death. To a lesser degree these results may be obtained when only three limbs are used. From the results of such experiments it would seem that a condition producing stasis in a large capillary field would produce the signs of shock. It should be emphasized that simple vasomotor dilatation will not cause this condition. Section of the nerve supply to all the limbs does not produce the signs of shock (Fig. 1).

THE TREATMENT OF SHOCK

The treatment of shock may be divided into: (1) general measures, (2) the use of drugs, (3) attempts

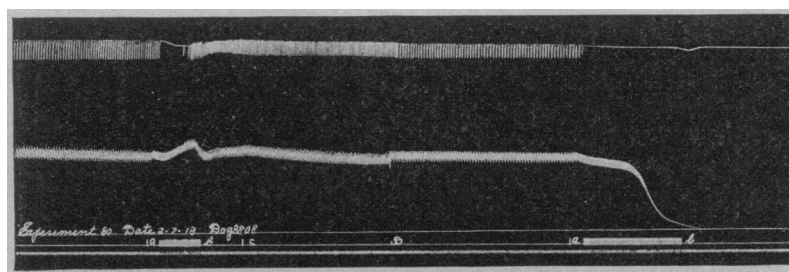


Fig. 2.—Kymograph record showing the effect of stimulating the central end of the vagus under light and deep anesthesia. Normal blood pressure was 115. At signal *a*, ether was disconnected and the control end of the left vagus was stimulated at *b* until respiratory movements returned. During the interval *D* (sixteen minutes) ether tension was increased. At *a* the ether was discontinued, and at *b* the vagus was again stimulated, and death occurred.

9. Mann, F. C.: Shock and Hemorrhage: An Experimental Study, *Surg., Gynec. and Obst.*, 1915, **21**, 430-441.

10. Janeway, H. H., and Jackson, H. C.: The Distribution of Blood in Shock, *Proc. Soc. Exper. Biol. and Med.*, 1914-1915, **12**, 193-197.

11. Erlanger, Joseph; Gessell, Robert, and Gasser, H. S.: An Experimental Study of Surgical Shock, *THE JOURNAL A. M. A.*, Dec. 22, 1917, pp. 2089-2092.

to restore the fluid volume, and (4) special measures. In studying any form of treatment of shock experimentally, it is necessary carefully to standardize the experimental procedures. In this series of investigations the routine method of experimentation was as follows:

The animal (dog) was etherized in a closed cabinet, intubated, and a constant surgical anesthesia maintained by means of the Connell apparatus. The carotid blood pressure was recorded with a mercury manometer. Marked changes in the temperature were prevented by the judicious use of an electric pad. After a normal record had been obtained, the abdominal viscera were exposed and gently sponged, about every fifteen minutes, with dry gauze. When the blood pressure had decreased and remained rather stationary at the desired point, which occurred usually from about one to two hours after exposure of the viscera, the viscera were returned to the abdominal cavity and the wound was repaired. After waiting a sufficient length of time to determine definitely that the blood pressure did not increase, the procedure designated to improve the condition of the animal was instituted. The blood pressure was taken as a criterion of the condition of the animal because it is the easiest indication to record and compare. It should be emphasized that the anesthetic was constant throughout the experiment. This removes the possibility of the ether producing an error in either the interpretation of the blood pressure record or the general condition of the animal. Careful anesthetic control experiments were performed, the etherization being maintained at the same tension and for a length of time equal to the shock experiment. It should be noted that

practical conclusions can be drawn only from the results obtained as applying to a condition in which the signs of shock were produced by exposure of the abdominal viscera. If the blood pressure is allowed to decrease until it is much less than half the normal pressure, it is rarely possible to restore it by any known method. This is an important point to consider in placing a value on any method of treatment.

The general measures employed consisted of placing the animal in the head-down position, and the application of heat, etc. A slight amount of benefit has been obtained by such methods in experimental shock. In fact, it was found to be of distinct value to keep a heating pad under the animal throughout the experiment, care being taken to apply only a moderate amount of heat.

Drugs are employed for one or two purposes, either as a stimulant to the circulatory system, as strychnin and camphorated oil, or to produce vasomotor constriction, as epinephrin or pituitary extract. The results of the experiments corroborate our previous investigation on the use of stimulants in experimental shock. In none of the experiments was any benefit derived.

The value of the use of vasoconstrictors in the treatment of shock is still an open question. In the first place, although the decrease in blood pressure is of great importance in shock, it is not known whether or not its increase by means of vasomotor constriction is in itself of much permanent benefit to the organism. In the second place, none of the vasomotor constrictor drugs produce a very prolonged effect. In experimental shock it is not possible to maintain blood pressure for a very long period of time near to the normal value by the use of the vasoconstrictor drugs. In our experience pituitary extract produced a more prolonged action, and seemed to be of somewhat greater benefit than epinephrin.

A large number of artificial fluids have been devised with which to attempt to restore the volume of fluid in shock. Our series does not yet include experiments from which positive conclusions may be drawn in regard to all of these. However, they have furnished enough data to justify some tentative conclusions in regard to their use in experimental shock.

1. Physiologic sodium chlorid solution is the least valuable of all the artificial fluids, although hypertonic sodium chlorid solutions are of value.

2. The making of the artificial fluid alkaline definitely enhances its value.

3. The use of glucose in the injected solution is also of definite value.

4. None of the saline solutions alone will maintain blood pressure for more than a very short period of time, even when it has been lowered to but a slight degree by exposure of the abdominal viscera.

5. The employment of the so-called colloidal solutions, such as those containing acacia or gelatin, is of

distinct value. The intravenous injection of these fluids will often restore and maintain the blood pressure for several hours after it has been decreased to at least one-half its normal value by the exposure of the abdominal viscera.

6. From these results it would seem that the ideal artificial fluid should contain (a) some substance to increase its colloidal properties, (b) an alkaline salt, and (c) glucose.

7. It should be noted that none of the artificial fluids will give as good results as whole blood or blood serum.

The value of transfusion in the treatment of shock is well known. In this series of experiments, citrated blood produced very good results. In order, however, approximately to restore blood pressure and maintain it, the amount transfused must be comparatively large. About 30 c.c. per kilogram produced the best experimental results. Such an injection will restore and maintain blood pressure under the experimental conditions outlined herein.

Blood serum seems never to have been used in the treatment of shock. In our experiments the intravenous injection of homologous serum has produced as good results, and, in most experiments, better

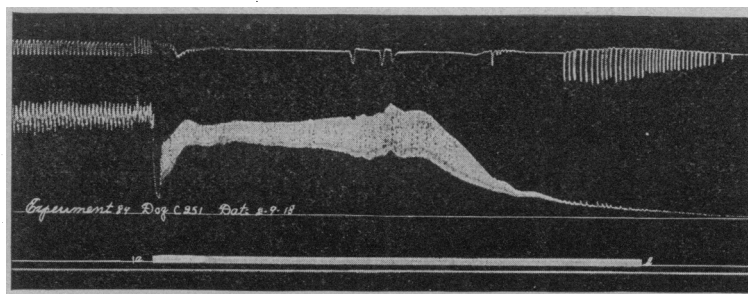


Fig. 3.—Kymograph record showing the effect of stimulating the superior laryngeal nerves under light anesthesia. At *a* ether was disconnected and signal *b* marks the period of stimulation of the superior laryngeal nerve, death occurring. This record is difficult to interpret, but it would appear that inhibition of the heart was the major factor producing death.

results, than any of the methods employed in the treatment of experimental shock. In none of the experiments in which the blood pressure had not decreased below one half of its original volume did the injection of serum fail to restore and maintain it for several hours. The best results were obtained by relatively large doses, 20 c.c. per kilogram. The serum was prepared by bleeding healthy dogs into sterile bottles which were placed on ice until it was desired to use the serum. From 35 to 40 per cent. of serum can be obtained in the dog in this manner from whole blood.

When blood serum, 20 c.c. per kilogram, is injected into the animal in which blood pressure has been decreased one-half, as the result of exposure of the abdominal viscera, it produces the usual rise in blood pressure and an increase in the amplitude of the heart, which occurs whenever an intravenous injection is made. However, the increase in blood pressure is usually greater, although it may take place more slowly, than when any of the other injection fluids are used, with the exception of whole blood when used in amounts equal to the amount of blood from which the serum was obtained. The blood pressure seldom decreases subsequently, or only to a slight extent, and usually, with the passage of hours, it increases until it may be greater than at the beginning of the experiment. I have not been able to obtain equally good results by any other method, except by the use of large amounts of whole blood.

Whether or not the use of serum will ever prove of any practical value, I do not know. The employment of large amounts of serum other than homologous serum would probably be very dangerous. It is possible that the latter serum might be of value under conditions in which the serum could be kept and whole blood could not be obtained.

The special measures employed in shock, such as rebreathing, have never been shown to be of much value under experimental conditions.

SUMMARY

The term "shock" is used by the surgeon in describing a definite clinical condition; it is probably due to a number of causes. In general, however, all cases may be included in two groups. One group contains the cases in which the clinical manifestation follows some time after the occurrence of the conditions incident to the shock. The other group includes the cases in which a severe or fatal condition supervenes immediately on receipt of the active agent. Experimentally, either condition can be produced by few of the methods which may be compared to their clinical manifestations. Chief of the methods by means of which a condition simulating the cases included in the first group can be produced experimentally is exposure of the abdominal viscera. The symptoms thus produced are due to a loss of circulatory fluid, probably due to, or associated with, a failure of the mechanism to control fluid volume. The signs of shock may be produced by the loss of an amount of circulating fluid that can be sequestered into capillary beds of venous trunks of the four limbs. The part the nervous system plays in the cause of shock is undetermined. It cannot be proved beyond doubt, experimentally, that shock is an etiologic factor, although clinically it seems to be definitely established that it is responsible for death in some cases, and in such cases it will probably be found to be of the nature of inhibition. This group might include a

large number of the cases contained in the second group. Experimentally, sudden death has been found to occur under deep etherization following stimulation of the nerves that inhibit respiration. It has also been produced under light etherization by the stimulation of nerves that produced an associated reflex inhibition of the heart. Either of these results may also occur clinically and the cause of death be described by the surgeon as shock. In the treatment of shock, experiments have not shown that the employment of drugs, either as stimulants or as vasoconstrictors, possesses very much value. The logical procedure, at least from the experimental standpoint, in the cases included in Group 1, would seem to be to attempt to replace the lost fluid. The best means of doing this is by the intravenous injection of large amounts of whole blood or blood serum. Some of the artificial solutions give good results. The ideal artificial fluid should contain, (a) some substance to increase colloidal properties, (b) alkaline salt, and (c) glucose.

RECONSTRUCTION OF THE COMMON BILE DUCT*

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Defects in the common bile duct may result from errors in operative technic, from strictures following ulceration or trauma, or from neoplasms. Since excision of the gallbladder has become a frequent practice, possibilities for injuring the common bile duct, particularly in inexperienced hands, have multiplied.

In an effort to devise some method that would be satisfactory in reconstructing the common duct, various procedures have been devised. Sullivan¹ described an operation in which a rubber tube is sutured into the stump of the hepatic or the remaining upper end of the common duct, carried into the duodenum, and surrounded with neighboring tissue and omentum. Molineus, according to Walton, has suggested the use of the appendix for reconstruction of the common duct. However, it is often difficult to secure a satisfactory appendix, the nutrition of the transplanted appendix would be very problematic, and it would carry bacteria it normally contains, which may cause cholangitis. It is for the last mentioned reason that it has been found unwise to connect the gallbladder with the colon instead of with the duodenum.

Lewis and Davis² have used experimentally transplanted fascia from the abdominal wall to repair defects in the common bile duct of dogs. Stropeni and Giacinto³ reported experiments in which a portion of a vein was used to fill a defect in the common duct.

Walton⁴ reviewed bile duct surgery and described an operation in which he uses a duodenal flap. Eliot⁵ has given the recent literature on repair of the bile

* Read before the Section on Surgery, General and Abdominal, at the Sixty-Ninth Annual Session of the American Medical Association, Chicago, June, 1918.

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints. A copy of the latter will be sent by the author on receipt of a stamped addressed envelop.

1. Sullivan, A. G.: Reconstruction of the Bile Ducts, THE JOURNAL A. M. A., Sept. 4, 1900, p. 774.

2. Lewis, Dean, and Davis, C. B.: Tr. Western Surg. Assn., St. Louis, December, 1913.

3. Stropeni and Giacinto: Zentralbl. f. Chir., 1914, 41, 190; abstr., Gior. d. r. Accad. di med. di Torino, 1914, 77, 21.

4. Walton: Surg., Gynec. and Obst., 1915, 21, 269.

5. Eliot, Ellsworth: Surg., Gynec. and Obst., 1918, 26, 81.