SURGICAL SHOCK FROM A CLINICAL STANDPOINT.

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It is generally conceded that the pathology of shock is not thoroughly understood, but it is also generally alleged that the predominant factor is a paresis of the circulatory system, especially of the heart and arteries. The clinical manifestations of shock, when analyzed in accordance with undisputed physiologic facts, will not permit us to accept this view without question. The essential condition in shock is a profound disturbance of the entire vasomotor and sympathetic systems, but this disturbance is in the nature of a hyperirritation rather than a paresis.

This disturbing influence may reach the vasomotor center through various channels, as, for instance, by direct irritation of the sympathetic nerves in abdominal operations; by crushing injury to the skeletal nerves, as in railroad injuries; and through the medium of the brain, as in sudden fright; or two or more of these factors may unite as a causative influence, as in railroad injury, where the influence of sudden and great fright is added to the crushing of the large nerve trunks. Through whatever channel the impression on the sympathetic nerve centers may be received, the effect is the same.

The clinical aspect of mental shock differs from that of traumatic shock only in point of duration. There is the same sudden pallor and clammy perspiration, the same

abolition of the pulse to a greater or less degree, the same relaxation of the sphincters and sense of absolute physical prostration.

But in mental shock these conditions are somewhat evanescent because of the evanescent nature of the exciting cause. That the pathology of mental, surgical, and traumatic shock is essentially the same has, I believe, never been questioned, and that the clinical manifestations of shock, from whatever cause, can be explained most completely by the theory of excessive irritation of the sympathetic system is, I believe, capable of satisfactory demonstration.

The principal symptoms of typical, uncomplicated shock are pallor, more or less livid in character; clammy perspiration; a small, sometimes imperceptible, but very rapid pulse of low tension; a tendency to relaxation of the sphincters and to suppression of urine, more or less marked, and a sense of mental and physical lethargy. The pallor of shock has been designated as "livid," in contradistinction to the waxy pallor of hemorrhage, because the arteries and arterioles alone are comparatively empty, the veins remaining filled; while in hemorrhage both arteries and veins are empty to a greater or less extent. This explains the bluish color of the mucous membranes and of the finger-nails so often seen in profound shock.

There is a condition of arterial anemia, with venous engorgement. Now how can this condition be satisfactorily explained on the theory of paresis of the circulatory system? In no other condition does arterial paresis result in empty arteries. Section of the sciatic nerve, whereby the vasoconstrictor nerves of the peripheral arteries are divided, results necessarily in complete and typical vasomotor paralysis; but the peripheral arteries become distended and filled with blood, and the surface is flushed.

That in shock there is a condition of general circulatory paresis involving both arteries and veins, whereby the blood supposedly collects in the large veins, is a theory not sup-

ported by the facts. The large veins of the abdomen, etc., are distended, but the peripheral veins are also filled. If it were not so you would have the waxy pallor of hemorrhage rather than the livid pallor of shock. Moreover, if there were general arterial paresis the arteries would not be contracted and empty. There is normally a certain "tone" to the arteries—a continuous action of the constrictor nerves—whereby the blood is kept circulating. Paralysis of these constrictors, whereby this "tone" is lost, invariably results in dilatation of the arteries, which remain filled with arterial blood.

I believe it to be a physiologic fact, though I have never seen a record of experiments tending to prove or disprove it, that there is a correlation in the functions of the arterial and venous vasomotor nerves whereby the equilibrium of the circulation is maintained; that is, that stimulation of the arterial vaso-*constrictors* coincides with stimulation of the venous vaso-*dilators*, and *vice versa*. If this be admitted, the clinical manifestations of shock are easily explainable on the theory of excessive irritation or stimulation of the entire sympathetic system, but affecting specially the constrictor nerves of the arteries and the vasodilator nerves of the veins. But whether the venous vasodilators are at the same time stimulated or not, the results would be the same owing to the greater contractile power of the arteries. This theory clearly explains the spasmodic action of the heart, the empty and contracted arteries, and the venous engorgement—conditions which are made manifest by the rapid, small, almost imperceptible pulse, with the peculiar pallor. But the stumbling-block to the acceptance of this theory has always been the fact that arterial tension is extremely *low* in shock, and we always expect it to be *high* when the vaso-constrictors are stimulated. This in reality should not only not be a stumbling-block, but is a condition that we ought to expect if we accept the theory that I am endeavoring to substantiate.

Without referring to any theory, it is a fact demonstrated by autopsies that in the first stages of shock the heart is thrown into a state of violent contraction—so violent in some cases as to be absolutely tetanic, causing death. Agnew, in his *System of Surgery* (p. 377), says: "When a fatal termination is to follow, death may be sudden, produced by heart spasm, the ventricles forcibly contracting and then ceasing to beat." The writer in the *American Text-book of Surgery* says that in sudden deaths from shock "the heart has been found contracted and empty." In the *Medical and Surgical Reporter* of October 12, 1895, a case is related of a woman who, upon being suddenly told of her son's death, fell to the floor and expired. Autopsy showed *rupture of the left ventricle of the heart.* It certainly cannot be contended that death occurred in these cases from *paresis* of the heart and arteries.

In cases that do not prove immediately fatal the same condition exists, only in a less intense degree; the heart is in what I may term a tetanoid condition, contracting rapidly and forcibly, but relaxing very imperfectly. Thus but little blood enters the left ventricle, and consequently but little passes from the ventricle to the arteries; so that however strongly the arteries may be contracted, the tension is necessarily low because of scanty blood-supply. This very condition of the heart and arteries which I claim exists in profound shock has been experimentally demonstrated in the laboratory, where severe stimulation of the cervical sympathetic caused spasm of both the heart and arteries and *low arterial tension.*[1]

Thus, however we may theorize about a condition of paresis of the heart and arteries in shock, the record of post-mortem examinations shows that the heart is contracted and empty, even ruptured; and laboratory experiments have repeatedly produced the same condition that we see in shock by

[1] Landois and Sterling's Manual of Physiology, p. 106.

electrical *stimulation* of the vasomotor system. By reason, then, of this cardiac and arterial spasm affecting the entire arterial system there is of necessity venous engorgement, causing the clinical manifestations of livid pallor. This same arterial anemia, existing alike in the brain and spinal cord, would necessarily cause the sense of mental and physical prostration that is often so pronounced in profound shock. This has also been repeatedly demonstrated by experiment, notably by Brown-Séquard, who brought about a condition of temporary paraplegia simply by causing profound arterial anemia of the spinal cord.

The symptom of profuse and clammy perspiration can only be explained by the theory of hyperirritation of the sympathetic nervous system. No other theory will meet the conditions. In profound shock the skin is pale, the surface temperature is low, and yet there is profuse perspiration. This is caused by *stimulation* of those secretory branches of the sympathetic system which are distributed to the sweat-glands, and can be caused in no other way.

In 1875, special secretory nerves distributed to the sweat-glands were described by Goltz. Since then various experiments have been made demonstrating their nature, and also that the secretion of perspiration is entirely independent of vascular conditions.[1] Thus stimulation of the peripheral end of a divided sciatic nerve will cause perspiration, even after the limb is amputated; also, if after section of the nerve the leg be exposed to a high temperature it becomes suffused with blood, but remains dry. The secretory nerves of the sweat-glands are, of course, paralyzed; but if electricity be applied to the peripheral end of the nerve a profuse perspiration is at once induced by reason of *stimulation* of the secretory nerves. Therefore, since paresis of the secretory nerves of the sweat-glands does not cause perspiration, and since (as demonstrated by experiments) the secretion of per-

[1] Howell's Text-book of Physiology.

spiration does not in any way depend on vascular conditions, it is a fair conclusion that the perspiration of shock *is caused by stimulation* of the secretory nerves of the glands.

On the other hand, the secretion of urine depends entirely upon the condition of the circulation through the kidney, no secretory nerves for the kidney having been discovered. The quantity of urine secreted depends on the pressure and supply of blood to the kidneys. Thus *general* arterial paresis would lessen the quantity of urine secreted because, although the arteries of the kidneys would be filled with blood, the pressure would be very low; but more especially would we look for this condition in shock, in which the arteries are contracted and yet the pressure is low, the arteries being incompletely filled, owing to the tetanoid condition of the heart. To this condition, then, is due the very scanty secretion of urine that is so characteristic of profound shock; and this condition is directly caused by excessive vasoconstrictor stimulation.

Again, the relaxation of the sphincters, which is a more or less constant condition in profound shock, and is especially noticeable in mental shock, is not dependent on circulatory conditions, but is another strong witness to the hyperirritation of the entire sympathetic system. The peristaltic movements of the rectum and intestines are entirely involuntary and under the control of the sympathetic system, while the control of the external sphincter is largely voluntary and governed by the cerebro-spinal system. Therefore, when by reason of sudden and severe fright a condition of mental shock is induced, there is an instantaneous sense of relaxation of the anal sphincter. This is certainly induced by sudden and powerful stimulation of the sympathetic nervous system, whereby active peristalsis is provoked, and, at the same time, by reason of the cerebral and spinal anemia induced by the sudden arterial spasm, there is a more or less complete inhibition of the cerebro-spinal control of the external sphincter, and involuntary defecation may occur.

Therefore, when we regard surgical shock from the standpoint of the clinician, and when we reason from physiologic facts, which are beyond dispute, to the conditions which we see in shock, we are forced to the conclusion that the true pathology of uncomplicated shock is a hyperirritation of the entire sympathetic system. We shall be confirmed in that belief if, after careful analysis, we find that line of treatment most beneficial which coincides with this theory. The remedies that hold the confidence of operators are comparatively few—opium, strychnine, intravenous saline infusion, and external heat. To these should be added nitrite of amyl and nitroglycerin. The effect of nitrite of amyl is so evanescent that its use is generally confined to carrying the patient through the last stages of the operation. Nowhere have I been able to discover a record of ill effects from its use under such conditions, and often have I seen a rapid, almost imperceptible pulse restored to comparative fulness by its free administration. Its use in uncomplicated shock is never other than good, and yet it is a powerful vasodilator. It relieves arterial spasm, and is in no sense a circulatory stimulant. The action of nitroglycerin is similar. It is a remedy from which we may rightly expect much, but it must be given as strychnine is given, in doses that would ordinarily prove almost toxic.

We are not dealing with a condition of moderate arterial contraction; but we have to overcome intense spasm, and extraordinary doses will be required. Its use is not generally popular because of its well-known sedative and relaxant effect on the arteries—a condition already existing, according to the generally accepted theory of vasomotor paresis. By reason of this theory strychnine is at present the most popular drug in the treatment of shock because of its well-known action as a circulatory stimulant; but the universal testimony is that strychnine is useless unless given in large doses or in small doses very frequently repeated. For instance, Dr. J. B. Hall says, in the *British Medical Journal*, November

25, 1899, in discussing shock: "While $\frac{1}{4}$ grain (of strychnine) is a rather large dose, and should not be given as a routine practice, there is scarcely any use in giving less than $\frac{1}{10}$ grain when strychnine is really indicated." Many other authors express the same opinion, and the experience of all who have derived any benefit from its use corroborates it.

Now let me quote from accepted authorities in therapeutics. H. C. Wood says: "It has been shown by experiments of Klapp that the primary stimulation of the vasomotor centers (by moderate doses of strychnine) is followed by fall of arterial pressure and vasomotor palsy; also, that very large doses produce an immediate depression of the vasomotor centers and fall of blood-pressure." Bartholow says: "A large, toxic dose of strychnine will paralyze instead of stimulate the vasomotor center in the medulla, and thus prevent any rise of blood-pressure." Dr. Carl Heineman has found that "large doses cause diminished frequency of cardiac movements, with diastolic pauses." La Housse has shown that "large doses slow the action of the heart by a paralyzing influence on the intracardiac ganglia." Therefore, if shock consists essentially in a *paresis* of the vasomotor system, strychnine in the doses always recommended is one of the most dangerous agents that could be used.

Now as to the use of normal saline solution. Dawbarn was, I believe, the first to use it as a remedy for surgical shock; but now, properly used, it is the remedy most universally relied on and most useful. I say properly used, because in order to get the greatest benefit it should be used as Dawbarn originally advised—an intravenous injection of normal salt solution heated to about 115° or 118° F. Many have objected to the temperature advised, thinking it too high; but bear in mind that the sympathetic ganglia in the walls of the heart and the arteries are in a condition of extreme irritation, and the musculature is in a state of spasm. By the time the small stream of saline solution reaches the

heart it has been diluted by the mass of blood filling the vena cava, and the temperature of the blood that flows into the heart and through the lungs before it reaches the left ventricle and the contracted arteries is but little above normal—just enough to exert a powerfully sedative effect on the irritated ganglia and muscles. We all know that moist heat only moderately high, applied to the surface, causes a red, suffused condition of the integument by reason of paresis of the cutaneous arterioles and capillaries. The same result follows when the moist heat is applied within the arteries.

Dawbarn erroneously attributed the beneficial effect to the stimulation of the paretic heart and arteries, but certainly the heat in the small amount of saline solution in the vena cava would be so dispersed that the effect would not be stimulating.

Again, the solution should be administered intravenously, because thus the heat is brought more directly to the irritated ganglia, and the entire effect, mechanical and otherwise, is brought about much more certainly and rapidly; and even if there were more danger in this method than when administered subcutaneously, still the risk should be taken because the benefit is commensurately greater.

When all that is desired is the mere mechanical effect of supplying the solution to replace blood lost by hemorrhage, the subcutaneous method with the solution at a temperature of 100° F. will suffice; but in profound shock infuse a solution at a temperature of at least 115° F. directly into a vein.

DISCUSSION.

Dr. Willis E. Ford.—I am very glad to hear such a valuable paper on shock. I have revised my own views somewhat as to shock within the past week. This last week I lost a patient, for the first time, under circumstances which gave me the feeling that perhaps I have been mistaken in some of my theories

regarding surgical shock. A week ago last Monday I operated on a woman for a large fibroid tumor. I had some difficulty in removing the tumor on account of impaction. However, I completed the operation inside of fifty minutes, and the woman went off the table in good condition. She was forty years of age, and weighed 110 pounds. She was a very intelligent woman, and was not at all depressed or apprehensive, nor was there anything surrounding the circumstances of operation which could in any way be questioned. I completed the operation before three o'clock in the afternoon, and at eight o'clock I was summoned to the hospital at once. The woman was in collapse. I made a hasty examination, and thought she was having a hemorrhage. I found this was not so. Her pulse, which I could barely feel at the wrist, was slow. Her temperature was normal. Her surface was cold, and she was profusely perspiring. She was perfectly conscious and not at all apprehensive. She had none of the nervous excitement which most women have in connection with internal hemorrhage. Her pulse-rate made me think that she was not having a hemorrhage. That, added to my physical examination, excluded hemorrhage, and made me think it was some form of shock. I thought, possibly, I had tied off the ureters. The woman was catheterized and four ounces of perfectly normal urine obtained. She remained in a condition of collapse which deepened until the wrist pulse was entirely lost, and I could hardly hear her heart beating for thirty-six hours. During this time we applied external heat, and I gave, as I always do, and as she had before she went into this condition of shock, saline enemata, ten ounces every three hours, which is the ordinary rule in the hospital. I tried that in preference to the intravenous administration of salines, and I do it in all cases after a serious surgical operation. I supposed she would die, and I sent for her husband, who had not given me any account of any previous illness pointing to disease of the heart or of the kidneys. The next morning, when I went back to the hospital, she was in pretty much the same condition. I was told by the nurse that at midnight two ounces of urine had been drawn, and at eight o'clock the next morning she was not able to get more than two drops. Then I was certain that I had tied off both ureters. I made an examination again and found it was not true. She was

given the thirtieth of a grain of strychnine every two hours; a saline enema of ten ounces every hour, very hot, and she had an infusion of digitalis leaves, freshly made, half an ounce every two hours during that day. Her stomach was in good condition. She took water, and she wanted something to eat. She was perfectly sane and quiet. We could not feel the pulse at the wrist during any part of this day. Toward evening she had a sudden rise of temperature to 104°. Immediately after that she passed a large quantity of urine. The next night she passed thirty-six ounces of urine, which was free from albumin, casts, or blood. It was normal. She went on for three or four days in a comfortable condition, with a temperature under 100°, and with a pulse of such great tension that I stopped the administration of strychnine and heart stimulants but gave saline enemata, and gave her milk and water freely by the mouth. After three days in this condition all symptoms had apparently ceased, and she was in a fairly normal state. At the end of a week she again went into a collapse; urine was again entirely suppressed. After ten hours we drew off ten ounces of urine which was filled with blood and casts, both granular and hyaline, in great abundance. From that time she failed, developed uremic coma, and died on the ninth day. No autopsy.

I was deeply impressed with the history of this case. After the pulse returned we failed to find anything the matter with the heart or with the urine. After the prolonged collapse from shock she recovered so fully that I had no suspicion of any further trouble. Her husband went home. Eight days after the operation she went into a second collapse, with stoppage of urine. A curious condition noted was cyanosis, which occurred over the legs. They became blue and purple, and looked like purpuric spots covering the surface of the limbs and a part of the body, although her finger-nails did not seem to show very much cyanosis at any time, neither did her lips show marked cyanosis, which we so often see. I think she must have had an old myocarditis. I have seen several cases of serious disturbance of the heart following the administration of ether where a history of some obscure heart disease could account for the accident. Physical examination does not always reveal the condition of the heart muscle.

DR. PHILANDER A. HARRIS.—The value of that most important measure, intravenous injection of normal salt solution, is recognized by all of us. I only wish to speak of a point which the writer of the paper did not cover or make sufficiently clear, and that is the temperature of the solution. When we speak of the temperature of the solution it is necessary to consider several factors which affect the temperature of the injected fluid as it is delivered into the median basilic vein. If, in our haste, we improvise a fountain, employing a rather large tube to conduct the solution downward from the fountain, taking the precaution, which I think is valuable, namely, of making the infusion rather gradual, taking at least forty or possibly fifty minutes to give a pint or two of normal salt solution; then the fluid will enter the vein at a temperature of from fifteen to twenty-five degrees lower than that in the fountain. If anyone doubts this, all he has to do is to take a moderately large tube, put the fountain a few feet above the patient, time the rapidity of the flow, and by personal observation verify the truth of my assertion. He will find the radiation of heat from the tube has reduced the temperature of the solution fifteen, twenty, or twenty-five degrees below that in the reservoir, according to the temperature of the room; consequently, we must remember that a small tube is better than a large one on account of the lessened radiation of heat, and we should remember that if we are using a large tube and want to give the solution slowly, the temperature should be more than in the fountain. We can use a solution at a temperature of 130° or 140°, provided we employ half an hour or more for the giving of two pints of normal salt solution.

DR. LAPTHORN SMITH.—We are not able to say as yet that there is no such thing as surgical shock. In my earlier operations I saw many patients die from shock; but during the last one hundred laparotomies I have only lost one or two patients from shock. There are several good reasons for the difference, and I believe my experience must be the same as that of other operators. First of all, we are enabled in the majority of cases now to perform a laparotomy without seeing the bowels. We cover the bowels over with hot cloths, and they are not touched. That is one great thing, for every irritation of the sympathetic is reflected on the heart and bloodvessels. Furthermore, we do not do

prolonged operations as we used to do, and the shorter the operation the less the shock in most instances. Formerly we deluged our patients with hot water, and in evaporating it became iced water almost. That has been done away with. We apply hot, dry towels and do all we can to keep the patient dry. With no depression from the anesthetic, no depression from loss of blood, no depression from cold water in washing the patient, and by giving hot injections per rectum when needed, I expect that at the next meeting or two, if anyone reads a paper on shock, it will be only to say that there is no such thing as shock.

Dr. Reuben Peterson.—If I understand the matter correctly, the paper was not on the prevention of shock, but the treatment of it after it is present, and it seems to me in the discussion we should not lose sight of that fact. Whatever may be our good fortune in the future, we certainly have shock at the present time. Every operator has shock, and will probably have it to contend with in some of his cases for some years to come. Shall we give stimulants or sedatives for shock? I was fortunate enough to be associated with Dr. Boise at the time he began his investigations, and you will remember that in a paper he read some years ago he detailed a case which led him to think along the lines he has set forth. That was a case in which the woman was septic, in which all kinds of cathartics and stimulants had been administered in order to produce movement of the bowels. Everything failed, and then the doctor reasoned that, inasmuch as they had all failed, he would try exactly the opposite treatment, and he gave the patient a good dose of morphine. The result was the woman had a free movement of the bowels, and recovered from the septic condition. Believing, as I do, that the arguments set forth are correct, I have adopted his plan in many cases of shock due to sepsis, and I have found it much better to give sedatives under these circumstances than try to stimulate the patient. We all know when we have a case of surgical shock from a railway accident it is useless to try to effect anything by stimulants. A sedative will work far better than a stimulant in the majority of cases. The same holds true with reference to sepsis. Recently, before the Chicago Gynecological Society, a case was reported where it was thought that the shock was due to intestinal obstruction. I stated at the time it seemed to me

that the intestinal obstruction was not so much due to an adhesive band as to sepsis, for the reason that he tried massage of the abdominal wall without avail; yet when he gave a dose of morphine the patient's bowels moved in two or three hours, and the result was recovery. Therefore, the practical deductions to be drawn from the paper are that we should cease to stimulate patients when they are shocked from sepsis, and try sedative treatment.

DR. HUNTER ROBB.—I agree entirely with what Dr. Boise has said with reference to this interesting subject.

I simply wish to say a few words about those instances in which death is supposed to be due to shock following abdominal operations. The remarks made by Dr. Ford remind me of some cases that have come under my observation during the past few years. In several of these instances death followed an abdominal operation, and despite a very thorough bacteriologic examination of the tissues no micro-organisms could be demonstrated, nor was it possible to find any satisfactory cause of death. But although from a careful examination of the urine of these cases before operation no evidences of any involvement of the kidney could be determined, subsequently to the operation marked pathologic changes appeared in the urine secreted. Moreover, microscopic examination of the renal tissues disclosed the presence of inflammatory changes. Such findings would certainly seem to suggest the possibility that the anesthetic may have produced the fatal result, at least in a certain number of cases, and that not shock but inflammatory changes in the renal tissues induced by the anesthetic were responsible. Before accepting such a view, however, in any given case a careful examination of the pelvis from a bacteriologic stand-point is necessary in order to exclude the possible presence of a peritonitis which may have existed without giving rise to any definite clinical manifestations during life.

DR. HENRY D. FRY.—I would like to ask Dr. Ford as to the character of the physical examination he made by which he excluded hemorrhage. I would also ask him if he made an autopsy in this case.

DR. FORD.—Examinations of the heart and of the urine were made a few days before I operated by men other than myself, who are perhaps better able to judge of the soundness of the

woman. In examining the patient for hemorrhage no clotted blood was found. The woman had never had any peritonitis. I did not have the opportunity of making an autopsy. I believe it was uremia secondary to the condition of shock which stopped the heart. I think we did have shock and she had uremia. She rallied from shock in thirty-six hours, and it is my belief that she had some obscure disease of the kidney which was not recognizable before.

DR. FRY.—The reason I have asked Dr. Ford a question regarding hemorrhage is because I think shock is rare, and secondary hemorrhage frequent when coming on several hours after operation. I do not believe we can find out whether or not there is secondary hemorrhage by examination through the vagina. The only way to tell is to reopen the abdominal wound sufficiently to get down to the peritoneal cavity to determine whether or not there is blood in there. We may have hemorrhage and no clotting, and be unable to distinguish it by a vaginal examination, especially where the symptoms of shock come on some hours after the operation. I have on several occasions had the symptoms of shock occur some hours after operation, have reopened the abdomen, and found that it was hemorrhage. I believe that the principal point to bring out here is to realize that fact, which is only too common, and to assign the cause of death to shock, when I believe, in the vast majority of cases, it is hemorrhage.

DR. FORD.—You can tell by the pulse.

DR. FRY.—There is no symptom by which you can tell that a hemorrhage is going on. Some years ago I read a paper before this Society in which I tried to bring out the symptomatology of hemorrhage. I had a patient die from secondary hemorrhage with an elevated temperature. We are usually led by text-books to believe that where we have hemorrhage we have a subnormal temperature. In that case the patient died with a temperature above normal and steadily rising. I do not believe we can rely on any symptom or set of symptoms in regard to secondary hemorrhage, and there is only one way to determine it, and that is to reopen the abdomen.