POST-OPERATIVE SHOCK AND SHOCK-LIKE CONDITIONS TREATMENT BY INFUSION IN LARGE VOLUME *

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A number of theories have been advanced to explain shock. One of the most popular of these has been the neurogenic theory which ascribes the fall of blood pressure and consequent shock to exhaustion of the vaso-motor centre. Mitchell, Morehouse, and Keen [1] suggested this possibility near the close of our Civil war. Crile [2] has been its chief sponsor in more recent years.

During the World war, a Research Committee was appointed by the British Government to study traumatic shock. Largely through the activities of this committee, important observations and discoveries bearing upon shock were made. Dale and Laidlaw [3] were able to produce typical shock in animals by the intravenous injection of histamine, and thereby to study shock under experimental conditions. Upon opening animals, moribund from this type of shock, they found the heart executing muscular beats of moderate vigor, although the arteries were pulseless. The veins were not distended, and if clamped, they filled very slowly from the periphery. A large part of the blood, in fact, had disappeared from active circulation. It appeared that the weakness of the heart beat was due to a reduced inflow from the veins and not to any essential cardiac deficiency.

In the shock of wounded soldiers Robertson and Bock [4] and Keith [5] have demonstrated a diminution in blood volume, even in the absence of significant hæmorrhage. In experimental shock Gasser, Erlanger and Meek [6] have made similar observations.

The investigations of Cannon, Fraser and Hooper,[7] Taylor,[8] and Robertson and Bock [9] showed that a concentration of corpuscles existed in the capillaries as compared with the veins. Since the ratio of corpuscles to plasma in the veins remained approximately normal, it was assumed that there was a loss of plasma from the capillary channels. These observations have been amply confirmed, both in clinical and in experimental shock.

A large part of the blood which is out of active circulation is to be found, concentrated and stagnant, in the capillaries. Here additional loss of volume occurs by continuous passage of plasma through the capillary walls. Such diminution of volume of actively circulating blood has been called oligæmia, or exæmia, and is now believed to be a very important factor in the production and aggravation of shock.

The inevitable result of oligæmia is impaired effectiveness of circulation and diminution of oxygen supply to the tissues. Because of inadequate

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oxygen supply it is probable that changes in osmotic pressure conditions, and other physico-chemical changes not well understood, occur in the capillary areas. That these result in a further loss of circulating fluid is suggested by Hill and McQueen,[10] whose explanation of the sequence of events in shock we find attractive: "With a falling blood pressure as in shock, there is a general constriction of arterioles to maintain sufficient pressure to supply the heart and brain. The general capillary field is sacrificed to the arterial pressure in the brain and heart. A sure Nemesis awaits this method of restoring blood pressure if carried too far. De-oxygenation sets in in the capillary area, the osmotic pressure of the tissue cells rises and these imbibe more fluid, the viscosity of the concentrated blood increases, the capillary wall may suffer and become increasingly permeable. The kinetic pressure available in the capillary area, already a small fraction of a few millimetres of mercury, is inadequate to maintain the blood flow and the heart, so carefully shielded from oxygen want by vaso-constriction elsewhere, finds itself with progressively less and less blood to propel." This chain of events leads to a further decrease in blood pressure and a continuation of the vicious cycle with ultimate complete failure of circulation.

Hill,[11] Krogh,[12] Hooker [13] and Rich [14] have demonstrated that there are numerous capillary channels which under ordinary conditions do not function, but which dilate to receive blood when local tissue needs demand it. The dilation of these capillaries in answer to oxygen want in the tissues probably constitutes an important factor in the loss of blood from the main circulating channels.

Another factor in the production of oligæmia which we believe should be stressed is dehydration. Maintenance of proper blood volume depends to a great extent upon a sufficient reserve of fluid in the tissues or tissue spaces. If a deficiency of circulating blood is threatened it is obvious that the tissue fluids cannot be drawn upon unless they exist in adequate amounts. It is well known that patients who have lost much fluid, as from prolonged vomiting or diarrhœa, are more likely to fall into shock than are patients whose fluid balance has been maintained. Direct loss of circulating fluid through hæmorrhage has long been recognized as an important factor in the production of shock. In the presence of dehydration the loss of a small amount of blood may assume an importance out of all proportion to the amount of blood lost.

A great deal may be done to prevent shock. Diligence in this direction is better than the most skillful treatment after shock is established. An ample supply of fluid should be insured before, after, and sometimes during operation. It is easier to maintain fluid balance than to restore it. Occasionally, however, one is confronted unexpectedly by shock. It may arise under circumstances which are out of control, or it may occur after the most careful preparation and operative procedure.

A plan of treatment for shock has been outlined recently by Cowell.[15] He recommends the following measures: 1, Application of warmth; 2, Pro-

cure mental rest; 3, Relief of pain; 4, Restoration of deficient circulation by giving fluids by mouth, rectally, or by 10 per cent. glucose-saline solution intravenously (one litre in two hours); 5, Increase of intra-cellular oxygenation by insulin hypodermically (five units at beginning and at end of infusion); 6, If operation is needed, by choosing a local or gas-oxygen anæsthesia. Similar suggestions have been made by Fraser [16] in a post-war publication.

In civil practice, cold does not generally play so great a part as in war injuries, but maintenance of warmth is not to be overlooked. Procuring mental rest and relief of pain are important as early measures. In well-established shock, mental unrest and pain usually are strikingly absent. There is instead a remarkable apathy, and sensibility to pain is blunted. Often a needle may be thrust into the vein, or the vein may be cut down upon with little or no disturbance to the patient.

It is chiefly with restoration of deficient circulation that we are concerned. Cardiac stimulants, in the absence of organic cardiac disease, are generally uncalled for. Likewise the use of adrenalin to raise blood pressure is to be condemned. Acting as it does upon the arterioles, its effect is to raise the blood pressure in the arterial tree. But, as pointed out by Cannon,[17] this does not improve volume flow in the capillaries. ". . . Merely a higher arterial pressure is not the desideratum in the treatment of shock, but a higher pressure which provides an increased nutritive flow through the capillaries all over the body. This can be obtained . . . only by increase of the volume flow." Bainbridge and Trevan,[18] injecting adrenalin slowly into the veins of anæsthetized dogs for twenty minutes or longer, at a rate sufficient to raise and maintain blood pressure moderately high, found upon stopping the injection that the animals' blood pressure fell to a low level, with the development of typical shock.

To restore effective circulation it is necessary to restore and maintain the volume of circulating fluid. The importance of an abundant supply of water has been emphasized. Administration by mouth is often impossible or inadequate. The same may be said of rectal instillations. Fluid given subcutaneously, even in large amounts, is likely to be ineffective if shock is at all profound. It is absorbed so slowly that elimination keeps pace with absorption, and the blood volume is not increased. Intravenous injection is the only method by which volume of circulating fluid may be augmented quickly and surely.

For this purpose a number of fluids have been used. Whole blood has many advantages, especially if there has been hæmorrhage. It supplies erythrocytes as well as volume. Furthermore, it does not readily escape from the vessels. Its greatest disadvantages are the lack of ready and adequate supply, and the technical procedures involved in its administration.

The substitutes for blood most frequently employed are solutions of sodium chloride, glucose, acacia, and their combinations. It has long been known that normal salt solution may be safely used for intravenous administration. Clinical experience with it in the treatment of shock has heretofore

been disappointing. Fraser and Cowell,[19] treating shock from war wounds, gave as much as two pints of normal saline solution intravenously to a number of patients. They obtained a temporary rise of blood pressure which was followed by a rapid fall to a level sometimes below the original. Drummond and Taylor [20] record similar conclusions as to the value of salt solutions. Cannon [21] and Bayliss [22] ascribe this failure to the prompt passage of the solution from the capillaries into the tissue spaces. Hypertonic salt solutions proved no better than the normal solution.

Hypertonic glucose solutions have been employed with the idea of increasing blood volume, both directly and by extraction of tissue fluids, and at the same time supplying carbohydrate. It was hoped that glucose might be the means of combating the acidosis of shock. Cannon [23] found, however, that the blood sugar in shock is above normal, and that it is not related to the decreased alkali reserve. Acetone bodies were absent from the urine. The acidosis of shock is not the same as that of diabetes. Thalhimer,[24] Fisher,[25] Beresow,[26] and others, have reported good results by combining the use of insulin with glucose, Padgett and Orr,[27] however, treating experimental shock with glucose solutions, obtained almost identical results whether given with or without insulin. It seems probable that the beneficial effects of glucose are due largely to the water introduced with it.

The principal fault found with the simple crystalloid solutions is that they have generally failed to maintain blood pressure. A primary elevation has been obtained, but has been quickly lost as the solution escaped from circulation.

To overcome this difficulty Bayliss [28] introduced the use of gum arabic. A solution of 0.9 per cent. sodium chloride, containing 6 to 7 per cent. gum arabic has the viscosity of blood and the osmotic pressure of its colloids. It is chemically inert and apparently harmless. It will not pass through the capillary walls and retards the passage of water. Good results from its use have been reported by Drummond and Taylor,[29] who are quoted by Bayliss. Farrar [30] and Randall [31] likewise have found it useful in the field of gynecology and obstetrics. On the other hand, there have been unfavorable reports. Lee [32] mentions two patients in whom he thinks death was hastened by acacia. Hanzlik and Karsner [33] report anaphylactoid phenomena in animals injected with solutions of acacia in saline. Whatever its eventual status in the treatment of shock may be, acacia has not yet come into very extensive use. In addition to the fear of it, whether justified or not, there is the difficulty, in general hospital practice, of securing reliable preparations.

To specifically combat the exæmia of shock we may inject into the vein a fluid, such as blood itself, or the gum-salt solution of Bayliss, with the idea of having it remain in the blood vessels. With the increase of blood volume, blood pressure will be raised, oxygenation of the tissues will be increased, and the vital condition of all the cells in the body will be improved. If a sufficient amount of water can be given afterward, one may expect a lowering of osmotic pressure in the tissues, and the establishment of a balance with

the blood stream. Unfortunately, there are disadvantages which limit the use of these two substances.

Instead of using a fluid which is expected to remain in the blood vessels, we may select a solution such as normal salt or glucose. These, as we know, readily escape from the blood vessels, and are taken up by the fixed tissues, or stored in the tissue spaces. This probably occurs, at least partly, in answer to the physiological demands of the tissues. If solutions of sodium chloride or glucose are to be effective in the treatment of fully developed shock, they should be given in large quantity. In the first place, fluid requirements of the tissues must be met. In the second place, stagnant erythrocytes must be mobilized, viscosity of the blood reduced and blood volume increased. To do this we must fill a circulatory system which, owing to capillary dilation and an increase in number of open capillaries (Krogh[34]), is more capacious than under normal conditions.

We believe that the previous failures with salt solutions have been due to the administration of insufficient quantities. By injecting large amounts of fluid we have had gratifying results in a number of cases treated at St. Luke's Hospital during recent months. In nearly all the cases we have used normal salt solution, often with the addition of varying amounts of glucose. The solution has been given intravenously in amounts ranging from 2000 cubic centimetres to 8000 cubic centimetres at a single injection. The usual amount required has been about 4500 cubic centimetres. It has been given at the rate of about 500 cubic centimetres in ten to twenty minutes. Altogether forty infusions of saline and glucose have been given to thirty patients. Six patients have received a transfusion of blood (500 cubic centimetres) in addition to infusion. As far as we have been able to judge, all the cases treated by infusion were benefited to some extent. In several cases the treatment seemed to be a life-saving measure. In other cases, who eventually died, we have felt that death was due, not primarily to shock, but to the morbid condition which had produced shock.

The post-operative shock-like conditions observed resulted from a considerable variety of diseases. The signs leading to the diagnosis of shock, however, were much the same. The measurable indications of shock following radical mastectomy, for example, differed little from those observed in the "toxæmia" of intestinal obstruction, or of general peritonitis. Delbet[35] and Olivecrona[36] have already called attention to the similarity between traumatic shock and the shock-like condition associated with peritonitis. Similarity in response to treatment has likewise been striking and suggests that a variety of causes may lead essentially to the same condition. Whether it is called "toxæmia" or shock makes little difference so far as the treatment is concerned.

The infusion treatment, in common with other kinds of shock treatment, is most effective when administered early, and when the initiating cause or disease has been removed. Its best results are seen when shock is due simply to a severe operation, uncomplicated by infection, or other factors

not easily eliminated. It is less likely to be permanently beneficial when shock has been produced by a continuing cause, such as peritonitis, or other severe infection. In cases of this type the patient may be brought out of the shock-like state. Then, if his powers of resistance to the disease are good, he probably will go on to recovery. If, however, the infection is overwhelming, shock may again supervene. Infusion can be repeated. The ultimate outcome may be either recovery or death. The infusion treatment in such cases has been useful in so far as it has permitted the patient to combat the disease without the added complication of shock. Infusion, of course, has no specific effect on the underlying disease.

Changes in blood chemistry immediately following infusion have not been striking. Such later changes as have occurred are explainable upon the basis of improved circulation and increased elimination.

The clinical improvement observed in some cases has been impressive. Some of the most interesting changes noted have been the return of normal color to ashen features, often as striking as if a transfusion of blood had been given; a return of tone to the facial muscles, notably those of the eyelids and mouth; a return from lethargy, or even unconsciousness, to relative mental alertness; a slowing of the pulse, with improved quality; the rapid increase of blood pressure, with stabilization near the normal pressure; return of renal function, with disappearance of "urinary suppression"; and, in general, the transition from a precarious condition to one of relative safety.

If given under careful supervision, we believe the administration of the large amounts suggested is safe. In no case have there occurred alarming signs or symptoms of cardiac or respiratory embarrassment. Three patients became a little restless and apprehensive and appeared slightly dyspnœic. In these cases the rate of flow was reduced and symptoms abated. Two patients developed severe chills during infusion. In both of these unbuffered stock solutions of 5 per cent. glucose were being used. The onset of chill occurred in one case after the administration of 1500 cubic centimetres. The infusion was immediately discontinued and no harm resulted. In the second case the chill did not appear until after 3500 cubic centimetres of glucose solution had been given. In this case the glucose was stopped but infusion was continued with normal saline. During the administration of saline solution the chill disappeared. In general, we have had more favorable results when saline was used either alone, or with the addition of prepared ampules of glucose solution. When glucose has been given with the salt solution, we have usually added 50 cubic centimetres of a 50 per cent. solution to 1000 cubic centimetres of normal saline. We have observed little difference whether the saline is given with or without glucose.

We are not unmindful that frequent warnings have been issued stressing the dangers attending intravenous injections of fluids, particularly if given in large amounts. Fatalities following infusion have been recorded in the literature. Acute cardiac dilatation has been cited as a frequent cause of

death. While we have encountered nothing of this kind, we nevertheless would urge constant vigilance. It has been our policy to have the patient under the immediate observation of one of the house staff during the entire infusion. The pulse has been watched at all times, and the blood pressure recorded after the administration of each 500 cubic centimetres of fluid.

Of thirty treated patients, eight died. The causes of deaths were as follows: general peritonitis (three cases), gangrenous ileo-colitis (one case), carcinoma of kidney (one case), intestinal obstruction (one case), liver abscess, tertiary lues, and diabetes mellitus (one case); and carcinoma of breast, bilateral (one case). Death occurred from seven to seventy-two hours after infusion, and from fourteen hours to eight days after operation. Five autopsies were done. In none was there evidence of cardiac dilatation. Two cases, both women, who were moribund when the infusion was begun, showed some pulmonary œdema. The two lungs in one case weighed at autopsy 1200 grams. The lungs of the other case weighed 1310 grams. The average weight for the lungs of a female is given by Morris [37] as 1023 grams.

In the field of animal experimentation there is abundant evidence that any excess of sodium chloride or glucose solution is quickly eliminated from the blood stream. Smith and Mendel,[38] working with rabbits, made intravenous injections of various isotonic solutions in amounts equivalent to the estimated blood volume of the animal. The solutions were injected in two minutes' time. Repeated blood volume determinations were made afterward. In the case of 0.9 per cent. sodium chloride solution, it was found that the larger part of the solution disappeared within the first five minutes after injection, and in the majority of cases, blood volume returned to normal within half an hour.

Lamson and his associates [39] obtained similar results with dogs. Gasser and Erlanger,[40] Smith,[41] and others, have obtained parallel results with glucose solutions.

To establish the relative merits of glucose and sodium chloride solutions in the treatment of shock, further work is needed. Cases of our own who have had blood sugar determinations done before treatment have shown normal, or above normal, content. As a rule plasma chlorides likewise have been normal. It is likely that the greatest value of both glucose and sodium chloride is as an adjunct to the safe administration and retention of water.

The following cases are recorded to illustrate the effects of treatment:

ILLUSTRATIVE CASES

I. Case No. 70388.—E. B., white, female, forty-two years. *Operation.*—February 9, 1929, for congenital anomaly of pancreas, producing obstruction of the common bile duct and chronic pancreatitis.

Post-Operative Course.—February 10, 1929, 11 A.M. Face very pale, yellowish color; eyes sunken, half closed; lips and tongue dry; fibrillary twitchings of muscles; completely unconscious; death seems imminent. *Infusion.*—4300 cubic centimetres normal saline solution with 100 grams glucose, and ten units insulin.

Time	Fluid	Temp.	Pulse	Resp.	Remarks
11:30 A.M.	0	106.6	Too rapid and feeble to count	5 (irreg., gasping)	Apparently moribund
12 NOON	1000		180	9 (irreg.)	Color better
12:30 P.M.	2000		168	9 (irreg.)	Color good
1:00	2800	106.3	156	11	Voided 100 c.c. (acetone-0)
1:45	3200	105	150	13	Twitchings have ceased; conscious; thirsty
2:45	4100		136	12	Vomited
3:15	4300	104	126	12	Comfortable; absolutely conscious
Infusion discontinued.					
9:00 P.M.		101.3	120 (strong)	16	Retaining fluids by mouth

Note.—Blood pressure readings were, unfortunately, not recorded.

Result.—Uneventful recovery.

II. Case No. 69571.—White, male, twenty-six years. Adm. B. P. 120/80. *Operation.*—February 27, 1929. Trans-duodenal excision of common duct stone.

Post-Operative Course.—February 28, 1929. Transfusion of blood, 600 cubic centimetres. Developed duodenal fistula, second post-operative day, profuse leakage. *Infusion.*—March 8, 1929. Pale, jaundiced gray color; skin, lips and tongue dry; eyes moderately sunken; thirsty; prostrated; mentally depressed and talks about dying. *Infusion.*—4500 cubic centimetres of normal sodium chloride solution.

Time	Fluid	Temp.	Pulse	Resp.	B. P.	Remarks
1:30	0	99	120	20	98/86	See above
1:45	500		108	20	110/80	
2:00	1000	98.4	108	20	118/80	
2:15	1500		108	14	125/80	Asks for water
2:30	2000		106	14	132/80	More alert, asks questions about treatment
2:45	2500		105	12	142/85	
3:00	3000		105	14	140/80	
3:15	3500		106	13	145/80	Voided 576 cubic centimetres
4:00	4000		106	13	148/80	
4:20	4500		106	13	150/82	
Infusion discontinued.						

Time	Fluid	Pulse	Resp.	B. P.	Remarks
6:00		103	16	134/80	
10:00		92	18	132/78	
March 9, 1929		82	16	134/80	Duodenal tube passed beyond fistula for feeding
March 10, 1929		90	18	130/80	

	Urea N	CO_2	Chlor.	Sug.	Bile Ind.
Note—Adm. B. P.—120/80					
Bl. Chem. 3/8/29 before infusion	71.5	57.6	4.5		15
Bl. Chem. 3/9/29 20 hrs. after	50.0	61.7	5.25	125	13

Result.—Alarming symptoms disappeared after infusion. A second infusion of small amount was given a few days later. Patient made a good recovery.

III. Case No. 72760.—White, female, thirty years. *Operation.*—March 19, 1929. Laparotomy and drainage for general peritonitis, streptococcus hæmolyticus, of undetermined origin.

Post-Operative Course.—March 21, 1929. Face generally pale with slight flushing of cheeks; eyes sunken with bluish discoloration about them; lips dry; skin moist; abdomen much distended; constant vomiting of brownish, foul-smelling material; anxious, but mentally clear; critically ill. *Infusion.*—5000 cubic centimetres normal saline.

Time	Fluid	Temp.	Pulse	Resp.	B. P.	Remarks
9:20 P.M.	0	104.8	122	23	133/80	Restless, complains of thirst
9:44	1000		106	26	140/74	Alternately dozing and awake. Asks for water
10:27	3000		114	36	144/74	
11:15 Infusion discontinued.	5000	105.4	116 (Good)	40	148/76	Again restless, anxious to be left alone, dyspnœic

	R. B. C.	Hgb.	W. B. C.	P.	L.
Bl. Ct. before inf.....	4,600,000	90	28,800	90	10
Bl. Ct. after inf.....	3,400,000	92	12,800	88	12
Bl. Ct. 10 hrs. after inf.....	4,000,000	90	10,800	88	12

Progress.—March 22, 1929. Condition worse. Gastric lavage at 6 P.M. yielded foul fluid. Passing some gas by rectum. Two watery stools. Distention still very great.

Second Infusion.—March 22, 1929. 3500 cubic centimetres 5 per cent. glucose (unbuffered), plus 600 cubic centimetres normal saline.

Time	Fluid	Temp.	Pulse	Resp.	B. P.	Remarks
7:30 P.M.	0	103.0	96 (weak)	24	110/64	Voided 700 cubic centimetres during infusion. Had chill toward end
9:00	4100	104.8	136 (good)	40	165/80	

Infusion ended.

	R. B. C.	Hgb.	W. B. C.	P.	L.	Bl. U. N.	CO_2	Sug.	Chlor.
Before inf.....	4,800,000	92	11,000	90	10	35.7	37.2	167	5.85
Immed. after.....	4,200,000	90	6,000	84	16				
12 hrs. after.....						29.4	39	125	6.

Result.—Gradual improvement after second infusion to complete recovery.

IV. Case No. 71898.—White, female, forty years. Adm. B. P. 126/82. *Operation.*—April 2, 1929. Supra-vaginal hysterectomy for fibromyoma.

Post-Operative Course.—April 3, 1929. Developed symptoms and signs of peritonitis. *Infusion.*—April 4, 1929. Definite severe general peritonitis, streptococcus hæmolyticus. Infusion of 5000 cubic centimetres normal saline solution.

Time	Fluid	Temp.	Pulse	Resp.	B. P.	Remarks
2:55 P.M.	0	104.2	126	30	98/72	Face pale and moist. Tongue and lips parched. Distended; apathetic
3:30	2500		120	27	120/65	
4:05	5000	102.4	120	32	135/70	Face flushed; pulse well sustained. Voided 250 cubic centimetres
Infusion discontinued.						
8:00		102	103	26	128/80	

	R. B. C.	Hgb.	W. B. C.	P.	L.	B. U. N.	CO_2	Sug.	Chlor.
Immed. before inf.	5,800,000	102	28,600	92	8	18.5	40.9	133	6.25
Immed. after inf..	4,000,000	84	28,800	90	10	12.5	39	118	6.5

Second Infusion.—April 7, 1929. 2000 cubic centimetres saline plus 1000 cubic centimetres 5 per cent. glucose.

Time	Fluid	Temp.	Pulse	Resp.	B. P.	Remarks
5:55 P.M.	0	104.4	160	44	94/66	Face pale. Breathing shallow. Mouth open
6:40	3000	103.4	146	51	116/62	Face flushed
Infusion discontinued.						
April 8, 1929		103	142	44	106/70	Some gas expelled

Third Infusion.—April 9, 1929. 4500 cubic centimetres saline with 50 grams glucose and 5 cubic centimetres digitan.

9:00 A.M.	0	106.4	150	40	70/?	Semi-conscious, condition grave
12:00 NOON	4500	102	130	45	114/66	Fully conscious, much improved
Infusion discontinued.						
8:00 P.M.		101.6	112		112/70	

Fourth Infusion.—April 10, 1929. 5,500 cubic centimetres saline with 50 grams glucose.

9:50 A.M.	0	104.8	135	18	64?/50?	In coma, face white. Eyes fixed; apparently dying
12:15 P.M.	5500		134	20	80/50	
Infusion discontinued.						
6:00						Unconscious. Shows some œdema of legs

Result.—April 11, 1929. Died.

Autopsy.—80 cubic centimetres fluid in right pleural cavity; 60 cubic centimetres fluid in left. Both lungs show moderate œdema and advanced congestion in both inferior lobes. Right lung weighs 710 grams. Left lung weighs 600 grams. *Heart* is normal. *Abdomen.*—Acute fibrinous peritonitis.

V. Case No. 71944.—White, male, thirty-six years. *Operation.*—April 6, 1929. Gastro-enterostomy for duodenal ulcer. Cholecystectomy for chronic cholecystitis.

Post-Operative Course.—April 7, 1929. Twenty-four hours after operation, patient appeared quite ill; frequent vomiting; profuse sweating; face pale, lead gray color; eyes sunken; extremely prostrated; very apathetic; response to questions is slow but accurate. *Infusion.*—April 7, 1929. 5000 cubic centimetres normal saline solution.

Time	Fluid	Temp.	Pulse	Resp.	B. P.	Remarks
12:40 P.M.	0	100.3	120 (thin)	18	110/85	Drowsy
12:55	1000		102	22	125/75	Color better. More alert
1:10	2000		99	21	130/75	
1:25	3000		98	24	135/70	
1:40	4000		100	24	140/75	Talkative
2:05	5000	100.3	96	21	140/75	Marked clinical improvement
Infusion discontinued.						
April 8, 1929	3 P.M.				128/75	
April 9, 1929	3 P.M.				128/75	

Result.—Recovery.

VI. CASE No. 75570.—White, female, thirty-four years. *History.*—Seven days after delivery of a full-term baby, patient developed symptoms of acute intestinal obstruction. *Operation.*—September 27, 1929. (Three days after onset of symptoms.) Laparotomy revealed obstruction of upper small intestine due to volvulus. Adhesions freed and obstruction relieved with minimum of trauma and very little loss of blood.

Post-Operative Course.—Two hours after operation patient presented the typical picture of shock. *Infusion.*—2000 cubic centimetres normal salt solution (two hours after operation).

Time	Fluid	Pulse	Resp.	B. P.	Remarks
3:30 P.M.	0	140 Weak	24	80/64	Unconscious; condition poor
3:40	500	124 Slight Imp.	24	92/64	Cold
3:50	1000	120 Fair	24	110/68	Warmer; conscious
4:00	1500	112 Good	24	110/64	Conversing
4:10	2000	110 Strong	24	108/66	Asks for water
Infusion discontinued.					
September 29, 1929		84		125/80	

Note.—Urinary output during night before operation was 45 cubic centimetres. Catheterization two hours after infusion yielded 290 cubic centimetres.

Result.—Patient made a good recovery.

VII. CASE No. 74384.—White, male, forty-seven years. *History.*—Patient entered hospital with uniformly enlarged thyroid; general symptoms of hyperthyroidism; heart enlarged; auricular fibrillation; basal metabolism, plus 96. After one month of medical treatment, basal metabolism dropped to plus 15, and patient was operated upon. *Operation.*—September 5, 1929. Subtotal thyroidectomy.

Post-Operative Course.—Twelve hours after operation patient was extremely prostrated; pale; lips and tongue dry; very dyspnœic, breathing with mouth open; thirsty but too dyspnœic to drink, except small sips. Pulse rapid and weak, but regular.

Infusion.—3600 cubic centimetres normal salt solution twelve hours after operation.

Time	Fluid	Temp.	Pulse	Resp.	B. P.	Remarks
9:00 P.M.	0		156	40	90/50	Condition critical
10:30	3600		144	32	155/65	Face flushed; respiration easy

Note.—Most striking features were return of excellent color to face, improvement in quality of pulse, and relief of dyspnœa. At end of infusion patient asked for water and drank a full glass without difficulty. Twenty-four hours after infusion the blood pressure was 150/65.

Result.—Recovery.

SUMMARY

1. Whatever the absolute cause of shock may be, the essential fact of shock is de-oxygenation of the body tissues.

2. De-oxygenation occurs from impairment of circulation.

3. The impairment of circulation results from diminution of blood volume *in circulation*. This loss is due to stagnation of blood in the capillary areas, and to escape of plasma from the capillary channels. Hæmorrhage and dehydration are frequent factors.

4. To rationally combat shock, restoration of volume of blood in effective circulation is of first importance.

5. Physiological sodium chloride solution has heretofore been tried as a medium to replace lost volume and has been generally discarded.

6. By using physiological sodium chloride solution, with or without glucose, in amounts much larger than have usually been employed, we have consistently obtained gratifying results in the treatment of shock.

7. The danger of producing acute cardiac dilatation or pulmonary œdema has not been apparent in a relatively short series of cases.

8. We practise and urge, however, constant vigilance during the administration of large amounts of solution.

9. We believe that the results obtained to date with this method of treatment warrant continuation of its use.

BIBLIOGRAPHY

[1] Mitchell, Morehouse and Keen: Report to the Surgeon General, 1864, Circular No. 6, p. 17.

[2] Crile: An Experimental Research into Surgical Shock, Philadelphia, 1897. Blood Pressure in Surgery, Philadelphia, 1903. Anoci-association, Philadelphia, 1913.

[3] Dale and Laidlaw: Journal of Phys. 1910, vol. lxi, p. 318. Memorandum upon Surgical Shock and Some Allied Conditions, Report of Medical Research Committee, February, 1917.

[4] Robertson and Bock: Report of Shock Committee, English Medical Research Committee, August, 1918, No. 25, p. 226.

[5] Keith: Report of Shock Committee, English Medical Research Committee, No. 27, March, 1919, p. 5.

[6] Gasser, Erlanger and Meek: Am. Journ. Physiol., 1919, vol. l, p. 31.

[7] Cannon, Fraser and Hooper: Report of Shock Committee, English Medical Research Committee, December, 1917, No. 25, p. 73; see also Journ. Am. Med. Assoc., 1918, vol. lii, p. 527.

[8] Taylor: See footnote 2, Report of Shock Committee No. 25, p. 76.
[9] Robertson and Bock: Report of Shock Committee, August, 1918, No. 25, p. 216.
[10] L. Hill and McQueen: The Lancet, 1921 (July) vol. ii, p. 65.
[11] L. Hill: Further Advances in Physiology, Arnold, 1909, p. 143.
[12] Krogh: Journ. of Physiol., 1919, vol. lii, p. 457.
[13] D. R. Hooker: Am. Journ. Physiol., 1920–21, vol. liv, p. 30.
[14] Rich: Journ. Exp. Med., 1921, vol. xxxiii, p. 287.
[15] Lt. Col. Ernest Cowell: Proceedings of the Royal Soc. of Med. (War Section), July, 1928, vol. xxi, pp. 39–46.
[16] Prof. John Fraser: British Journ. of Surg., January, 1924, vol. xi, pp. 410–425.
[17] Walter B. Cannon: Traumatic Shock, D. Appleton and Company, 1923, pp. 175–176.
[18] Bainbridge and Trevan: Memorandum upon Surgical Shock and Some Allied Conditions, Medical Research Committee, February, 1917, p. 5.
[19] Fraser and Cowell: A Clinical Study of the Blood Pressure in Wound Conditions, 1917, Mem. No. 2 of the Shock Committee, pp. 1–26.
[20] Drummond and Taylor: The Use of Intravenous Injections of Gum Acacia in Surgical Shock, 1918, Mem. No. 3 of the Shock Committee.
[21] Walter B. Cannon: Traumatic Shock, D. Appleton and Company, 1923, p. 177.
[22] Bayliss: Intravenous Injection in Wound Shock, London, 1918, p. 24.
[23] Walter B. Cannon: Acidosis in Shock, Hæmorrhage, and Gas Infection, and A Consideration of the Nature of Wound Shock, Mem. No. 2 of the Shock Committee, 1917, pp. 41–57 and pp. 67–83.
[24] William Thalhimer: Insulin Treatment of Post Operative (non-diabetic) Acidosis. Journ. of Am. Med. Assoc., August, 1923, vol. lxxxi, pp. 383–385.
[25] D. Fisher: Insulin-Glucose Treatment of Shock. Surg. Gyn. and Obs., August, 1926, vol. xliii, pp. 224–229.
[26] S. L. Beresow: Insulin-Dextrose Treatment of Surgical Shock, Zentralblatt f. chir., December, 1926, vol. liii, pp. 3214–3217.
[27] Padgett and Orr: Surg. Gyn. and Obs., June, 1928, vol. xlvi, pp. 783–788.
[28] Bayliss: Intravenous Injection in Wound Shock, London, 1918, p. 80.
[29] Drummond and Taylor: The Use of Intravenous Injection of Gum Acacia in Surgical Shock, Mem. No. 3 of the Shock Committee, 1918.
[30] Farrar: Surg. Gyn. and Obs., 1921, vol. xxxii, p. 328.
[31] Randall: Journ. Am. Med. Assoc., September, 1929, vol. xciii, p. 845 (No. 11).
[32] R. Van A. Lee: Journ. Am. Med. Assoc., 1922, vol. lxxix, pp. 726–728.
[33] Hanzlik and Karsner: Anaphylactoid Phenomena from the Intravenous Administration of Various Colloids, Arsenicals and Other Agents, Journ. of Pharm. and Exp. Therap., 1920, vol. xiv, pp. 379–422.
[34] Krogh: See [12].
[35] Delbet: Bull. et Mém. de la Soc. de Chirur., 1917, Part 11, vol. xliii, p. 2121.
[36] Olivecrona: Acta Chir. Skand., 1922, vol. liv, pp. 559–634.
[37] Morris: Human Anatomy, B. Blakiston's Son & Co., 7th Ed., p. 1268.
[38] Smith and Mendel: Adjustment of Blood Volume after Injection of Isotonic Solutions of Varied Composition, Am. Journ. of Physiol., September, 1920, vol. liii, p. 323, No. 2.
[39] Lamson, Abt, Oosthuisen and Rosenthal: Journ. of Pharm. and Exp. Therap., July, 1923, vol. xxi, p. 401, No. 6.
[40] Gasser and Erlanger: Am. Journ. of Physiol., 1919, vol. i, p. 104.
[41] Smith: Intravenous Injection of Fluid and Repeated Blood Volume Determinations. Bul. of Johns Hopkins Hosp., September, 1925, vol. xxxvii, p. 177.