# The Nature and Treatment of Wound Shock and Allied Conditions

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Report No. 2, Special Investigation Committee, Medical Research Committee, (Great Britain)

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# PREFACE

**In August, 1917, the Medical Research Committee (Great Britain) appointed a Special Investigation Committee to undertake the coordination of inquiries into surgical shock and allied conditions, with a view to the better correlation of laboratory and clinical observations. The following were asked to serve on this committee:**

Prof. E. H. Starling, M.D., F.R.S. (Chairman).
Prof. F. A. Bainbridge, M.D., F.R.C.P.
Prof. W. M. Bayliss, D.Sc., F.R.S.
Prof. W. B. Cannon, M.D. (Harvard University).
Lieut.-Col. T. R. Elliott, M.D., F.R.S., R.A.M.C.
John Fraser, M.D., F.R.C.S. (Edin.).
Prof. A. N. Richards, Ph.D. (University of Pennsylvania).
Prof. C. S. Sherrington, M.D., Sc.D., F.R.S.
Col. Cuthbert Wallace, C.M.G., F.R.C.S.
H. H. Dale, M.D., F.R.S. (Secretary).

**The first report on "Intravenous Injections to Replace Blood" was published as a special Bulletin, Nov. 25, 1917. The second, on "Investigation of the Nature and Treatment of Wound Shock and Allied Conditions," which follows, was issued for official use, Dec. 25, 1917, and Sir Walter Fletcher, secretary of the Medical Research Committee, has given his consent to the publication of the papers in The Journal, as has also the military censor.**

**In sending the report, Dr. Cannon writes:**

"The work was done in a casualty clearing station a few miles from a part of the line where there has been constant fighting for three years with scarcely any change. Only the most serious cases which could not be reasonably moved any farther back were left with us for the town was repeatedly shelled and the station was twice bombed by aeroplanes during my stay. London, with its air raids, is a Paradise by comparison."

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# CLINICAL STUDY OF BLOOD PRESSURE IN WOUND CONDITIONS *

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Captain, M. C., R. A. M. C.
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## INTRODUCTION

For a period of over two years we have been reading and recording the blood pressures in a variety of wound conditions. Certain of the observations have been sufficiently interesting to warrant their recordance.

The results may be summarized under various headings:

(*a*) Control blood pressure readings.

(*b*) The blood pressure as observed in wound conditions.

(*c*) Blood pressure readings as studied subsequent to various intravenous infusions.

(*d*) Observations on the blood pressure in the first week of convalescence.

The readings published in this paper have been made at a casualty clearing station, except where otherwise stated.

## A. INSTRUMENTS EMPLOYED AND METHODS OF INVESTIGATION

We have employed several forms of mercury manometers, and of these generally the "Riva-Rocci" instrument. Lately we have been using a spring sphygmomanometer. It would appear that the mercury instruments give the most accurate readings of the systolic pressure, but it is difficult to obtain with them a correspondingly exact reading of the diastolic

* Discrepancies between the methods reported in this paper and those reported in later papers are accounted for by the fact that this paper had been completed and presented to the Medical Research Committee before the work which appears in the subsequent papers was begun.

pressure. The spring instrument, which from a mechanical point of view cannot be as accurate as a mercury column, certainly gives a more easily appreciated reading of the diastolic or minimum pressure. A great advantage of the former is its adaptability and small compass, allowing it to be easily carried in the pocket. With this instrument, many pressure observations on recently wounded men were made in the trenches, and in the advanced dressing stations of field ambulances. In certain cases in which the peripheral pulse can hardly be appreciated, it is necessary to read the pressure by means of the stethoscope. Such readings are very accurate, and a result can be obtained when no pulse can be felt.[1] Readings have been made of the systolic or maximum pressure, of the diastolic or minimum pressure, and of the pulse pressure.

### B. CONTROL BLOOD PRESSURE READINGS

A very large number of control readings were made from healthy soldiers. As was to be expected, a wide range of results was obtained. The readings of the systolic pressure were slightly lower than the records one would have accepted as normal in civil practice. Over a wide series of cases the average systolic or maximum pressure worked out at from 110 to 120, and the diastolic or minimum pressure at from 70 to 80, while the pulse pressure averaged 40 mm. We made what may be considered an interesting observation, that among soldiers engaged in the actual fighting, more especially infantrymen, the average systolic pressure worked out at a higher figure than among men of the same regiment in support, where they were only exposed to occasional fire.

It was noticed that the raised pressure of the fighting man tended to drop quickly when he was resting away from the firing line; for instance, a systolic pressure of 140 mm. dropped to 110 mm., or even to 100 mm.

Observations were made on a series of pilots and observers before and after a two hours' aeroplane bombing raid; the average systolic pressure was 135 mm., and it remained constant on return. In only one case the pressure was raised by 10 mm.

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1. Latterly we have used this method to the exclusion of all others.

## The Blood Pressure as Observed in Various Wound Conditions

Pressure readings taken in the line on various types of wounds,[2] at very short intervals after the patient had been wounded, show two distinct groups:

1. *The hypertension group*, in which the systolic pressure varies from 150 to 160 or even 170 mm.

2. *The hypotension group* (with primary shock), in which the pressure varies from 40 to 90 mm.

In the number of cases examined at this early period, there were practically none that occupied an intermediate position, that is, showing a normal blood pressure.

The pressure of patients in Group 1, after resting and the journey to the casualty clearing station, gradually falls, and in the case of a normal recovery with rest in bed the systolic pressure remains at a steady level between 110 and 120 mm.

In Group 2, the patients are cold, often pulseless, and many of them suffer from severe thoracic or abdominal wounds. Occasionally the anatomic lesion appears to be entirely out of proportion to the physiologic reaction; for example, a simple bullet wound of the buttock was accompanied by a systolic blood pressure of 50 mm. at the end of the three hours.

In this group intense primary shock is present, and with the occurrence of hemorrhage, secondary shock develops and the pressure continues to fall.

Following the application of treatment, arrest of hemorrhage and pain, with rest and warmth, the blood pressure may become partially reestablished.

### A. HEAD WOUNDS

1. *Scalp Wounds.*—These showed a slight elevation in blood pressure readings; all the cases which we had an opportunity of examining varied between 120 and 130 in systolic pressure, with a mean diastolic pressure of 100.

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2. As the result of further observations made in the front line trenches, the pressures of early wounded are divided into three groups (Cowell, E. M.: The Initiation of Wound Shock, p. 61): (*a*) Trivial wounds, in which there is no alteration in pressure or tension. (*b*) Moderately severe and not immediately endangering life, in which there is no primary shock, but in certain circumstances secondary shock develops. (*c*) Severe wounds, in which unavoidable primary shock is present.

2. *Compound Fracture of Skull with Dura Intact.*—These cases showed a high systolic pressure; with one exception all the cases that we had an opportunity of examining registered a systolic pressure of 140 mm. and over.

The single exception showed a systolic pressure of 100 mm., but such a low reading could be explained by the fact that associated with a very extensive fracture of the bone there was profuse hemorrhage from the meningeal and scalp vessels.

3. *Penetrating Wounds of Skull, That Is, Compound Fracture, Dura Torn and Foreign Body Retained.*—On

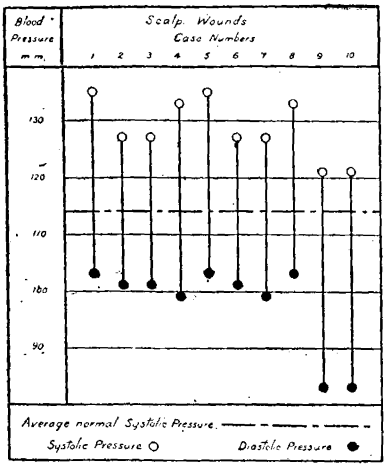


Chart 1.—Blood pressure in scalp wounds

examination of these cases it was found that as a rule a low blood pressure was registered. The systolic pressure varied from 60 mm. to 112 mm. over a series of cases. It was interesting to observe that the diastolic pressure closely corresponded to the systolic pressure with a correspondingly low pulse pressure. We can offer no explanation of this feature, which was relatively constant throughout this type of case. The wounds were such as not to result in increased intracranial pressure.

4. *Perforating Wounds of Skull.*—In types of perforating wounds—that is to say, with entrance and exit wounds—the blood pressure readings could be

divided into two groups, one in which the systolic reading was high, varying from 310 to 170 mm., and a second group in which the readings were low, varying from 100 to 110 mm. This subdivision corresponds to a certain anatomic distribution. The first group of

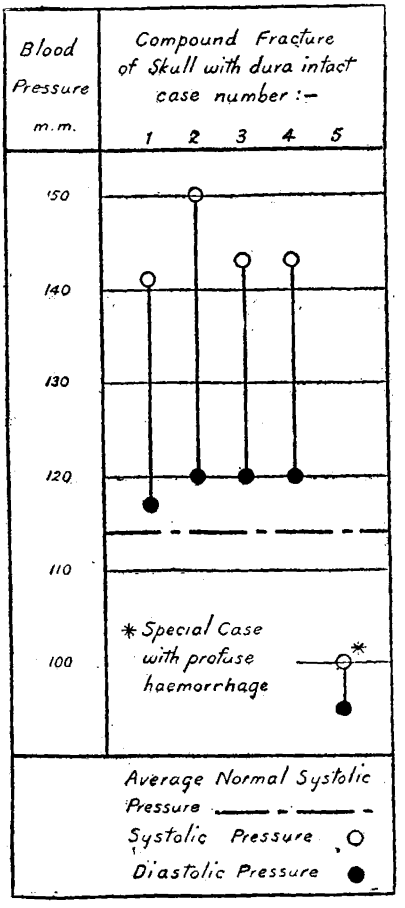


Chart 2.—Blood pressure in compound fracture with dura intact.

high pressure readings consisted of those in which the through-and-through wounds involve the ventricles, and were accompanied by hemorrhage into the cavities of the ventricles.

In the second group the wounds had involved the brain more or less superficially, and generally occurred in varying axes of the frontal and occipital regions.

Daily readings in patients recovering after operation for the second type of wound show that the systolic pressure is maintained at a constant level of from 110 to 120 mm. as long as convalescence is uncomplicated. The onset of meningitis is accompanied by a rise of blood pressure corresponding to the increase in the intracranial tension.

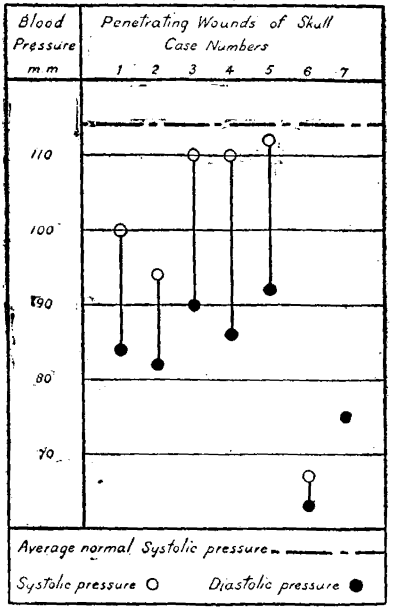


Chart 3.—Blood pressure in penetrating wounds of skull.

5. *The Effect of Anesthesia on the Blood Pressure Readings of Head Wounds.*—It was in regard to answering this question that our attention was first directed to blood pressure readings in various wound conditions. We had noticed a sequence of events somewhat as follows:

A man is admitted with one of the severe types of head wounds associated with tearing of the meninges and laceration of the brain; no examination is made of the blood pressure; operation is performed within an hour or two following

admission. In the later stages of the operation and subsequently, a case, which before operation had appeared promising, now entirely alters, the pulse rate becomes very rapid, unconsciousness deepens, the respiration rate is greatly increased, the patient becomes bathed in the most profuse perspiration, the temperature usually falls to a subnormal level, and death very rapidly supervenes.

It was difficult to discover any explanation of such a rapid change. The time was too short for sepsis to be the explanation, the amount of hemorrhage was never sufficient to explain the condition, and the injury itself was not one that involved any immediately vital

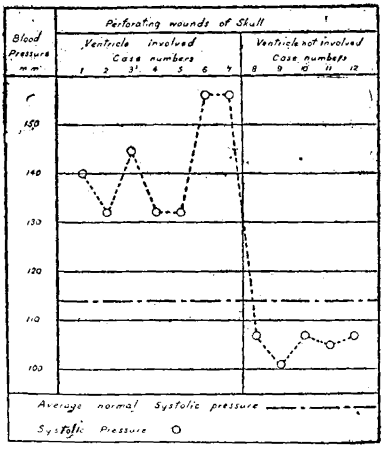


Chart 4.—Blood pressure in perforating wounds of skull.

part.[3] We began examining the blood pressures in these cases, and from such an examination we believed we had found the explanation of the disaster.

These are cases with a blood pressure which is either low or unstable. If operation is performed during the early stages of the case, the effect of the anesthetic, together with the extensive opening of the skull, is such as to produce a very marked fall of pressure. The level is so low that no further rise is secured, and the patient succumbs to a condition that clinically resembles acute shock.

3. Observations published in a later number of this series (Cannon, W. B.: Acidosis in Cases of Shock, Hemorrhage and Gas Infection, p. 47) show that this condition was probably related to a developing acidosis.

As an example of the unstable type of blood pressure, we have seen a case in which the systolic blood pressure before operation registered 164 mm.; taken immediately after operation the pressure had fallen to 16 mm. Death followed within thirty minutes.

With a view to overcoming the danger, our procedure was altered in two ways. If possible, operation was delayed for twenty-four and even for forty-eight hours, until, by successive readings, one had assured oneself that the pressure had acquired some degree of stability; the second modification was that in doubtful cases and in cases which demanded immediate operation, the operation was performed either with a local anesthetic or while the patient was under the influence of scopolamin and morphin. Since these modifications were adopted, disasters such as we have described have not been observed.[4]

6. *Summary of Results of Examination of the Blood Pressure in Head Wounds.*—1. Scalp wounds show no appreciable alteration in blood pressure.

2. Cases of compound fracture of the skull with dura intact show a relatively high blood pressure, averaging above 140 mm.

3. Penetrating wounds of the skull with free drainage are generally associated with a low blood pressure —from 60 to 112 mm.

4. In perforating wounds the blood pressure would appear to vary according to the anatomic distribution of the wound. If the wound has involved the ventricles, the blood pressure is high, varying from 130 to 170 mm.; if the wound is more superficial and has not involved the ventricles, the blood pressure is low, resembling the class in Summary 2.

5. The blood pressure subsequent to wounds of the head is apt to be unstable. If operation is performed under general anesthesia before the blood pressure has become stable, disaster is liable to ensue. The possibility of such an ill result can be diminished by delaying operation until the blood pressure has become stable or by performing the operation under local anesthesia.

4. These methods were employed before the rationale of preparing patients for operation, as given further on (Cannon, W. B.: Fraser, John, and Cowell, E. M.: The Preventive Treatment of Wound Shock, p. 93) had been worked out.

## B. ABDOMINAL WOUNDS

Before an analysis is made of the blood pressure in this type of wound, it will be of advantage to include a very brief summary of the cases, their clinical condition and relative blood pressures (Table 1).

From such a study as the table contains, the question arises: Can one with any degree of certainty draw definite conclusions from the blood pressure in supposed wounds of the abdominal cavity? We believe that we can with a minimum of error draw certain deductions.

First there are certain points to be considered. Cases 1 to 20 were observed at a casualty clearing station after a comparatively short journey, that is, within from three to six hours after being wounded. In Cases 21 to 30, with the exception of Case 27, more than ten hours had elapsed between receipt of the wound and admission to the casualty clearing station; in these the primary shock had abated.

Patient 27 was observed within six hours of being wounded. Patients 31, 32 and 33 were seen at an advanced dressing station, Patients 31 and 32 within three hours of being injured, and after a long and difficult carry over dangerous ground, and during a bitterly cold night. Patient 33 was quite fit when seen in the advanced dressing station, but very shortly afterward suffered marked collapse. Such a patient as this has been known to walk a mile after sustaining a penetrating wound of the abdomen, involving injury to the small intestine, and showing during this time a normal blood pressure and pulse. These patients soon become worse, and the collapse is then very intense.

The deductions we have thought it justifiable to draw are briefly as follows:

1. In patients seen on arrival at a casualty clearing station within six hours of being wounded, if there is an intraperitoneal injury of a hollow viscus, the blood pressure is low. In the series of cases of this description which we examined the systolic pressure varied from 50 to 100 mm.

2. When a period of from six to ten hours has elapsed, the pressure will probably have risen, for the primary wound shock is now beginning to pass off—the rest on the stretchers, the warmth and the sedative action of morphin are beginning to have effect.

TABLE 1.—SUMMARY OF ABDOMINAL CASES

| No. | Clinical Condition | Special Features | Blood Pressure |
|---|---|---|---|
| 1 | Wound of back; extraperitoneal wound of colon | Very slight hemorrhage; peritoneal cavity not opened | 90 dias<br>120 sys. |
| 2 | Wound of small intestine | General peritonitis; large amount of blood | 100 sys.<br>70 dias. |
| 3 | Multiple wounds of small intestine | Large amount of free blood | 85 sys<br>60 dias. |
| 4 | Small wound of anterior abdominal wall; two perforations of stomach | Moderate amount of blood in peritoneum | 70 sys.<br>40 dias. |
| 5 | Wound apparently perforating abdomen | Operation showed no perforation | 128 sys.<br>100 dias. |
| 6 | Wound apparently perforating abdomen | No operation; recovery... | 130 sys.<br>100 dias. |
| 7 | Large shell wound of abdomen | Injury to liver, colon and duodenum | 50 sys.<br>? dias. |
| 8 | Supposed wound of abdominal cavity | No perforation ..... | 150 sys.<br>100 dias. |
| 9 | Four perforations in small intestine | Large amount of blood in peritoneal cavity | 100 sys.<br>80 dias. |
| 10 | Perforation of cecum and transverse colon | General peritonitis; small amount of blood | 110 sys.<br>90 dias. |
| 11 | G. S. W. apparently perforating abdomen | Operation showed an extraperitoneal wound of rectum | 125 sys.<br>100 dias. |
| 12 | G. S. W. abdomen..... | Nonperforating ..... | 140 sys.<br>100 dias. |
| 13 | G. S. W. abdomen..... | Nonperforating ..... | 138 sys.<br>100 dias. |
| 14 | Two small wounds of small intestine; very stout man | Very slight hemorrhage.. | 140 sys.<br>95 dias. |
| 15 | G. S. W. abdomen..... | Injury to liver; abdomen full of blood | 138 sys.<br>100 dias. |
| 16 | G. S. W. abdomen..... | Injury to right kidney... | 140 sys.<br>100 dias. |
| 17 | G. S. W. abdomen.... | Injury to left kidney.... | 130 sys.<br>90 dias. |
| 18 | G. S. W. abdomen..... | Wound of liver; extreme hemorrage | 105 sys.<br>70 dias. |
| 19 | G. S. W. abdomen..... | Injury to prostate and bladder | 90 sys.<br>60 dias. |
| 20 | G. S. W. abdomen..... | Injury to liver ..... | 130 sys.<br>90 dias. |
| 21 | B. W. abdomen..... | Extraperitoneal wound of bladder; slight hemorrhage | 140 sys.<br>100 dias. |
| 22 | G. S. W. abdomen..... | Four perforations; small intestine; about 1 pint free blood | 120 sys.<br>95 dias. |
| 23 | G. S. W. abdomen..... | Four perforations jejunum and 1 in colon; about 1½ pints of free blood. | 120 sys.<br>93 dias. |
| 24 | G. S. W. abdomen..... | Injury to pancreas and spleen; about 1½ pints of free blood | 125 sys.<br>90 dias. |
| 25 | B. W. abdomen..... | Simple perforation of colon; no free blood | 110 sys.<br>80 dias. |
| 26 | G. S. W. abdomen..... | Perforated spleen, bruised kidney and colon; about 1 pint of free blood | 136 sys.<br>105 dias. |
| 27 | G. S. W. back..... | Six perforations; small intestine; profuse hemorrhage | 50 sys.<br>20 dias. |
| 28 | G. S. W. abdomen..... | Lacerated liver and kidney; about 2 pints of free blood | 110 sys.<br>85 dias. |
| 29 | G. S. W. abdomen..... | Bruising stomach; no hemorrhage | 120 sys.<br>90 dias. |
| 30 | G. S. W. abdomen..... | Tear of colon and small intestine | 120 sys.<br>90 dias. |
| 31 | B. W. pelvis..... | Observed in A.D.S.; exact wound unknown; long carry over the open | 50 sys.<br>30 dias. |
| 32 | B. W. pelvis..... | Observed in A.D.S.; probable small intestine lesion | 45 sys.<br>? dias. |
| 33 | G. S. W. abdomen..... | Observed in A.D.S.; probable small intestine lesion | 125 sys.<br>80 dias. |

3. At a period later than ten hours, the pressure begins to fall and a shocklike condition becomes evidenced; the change is due to sepsis and to loss of blood (secondary wound shock).

4. Perforating wounds of solid viscera of moderate severity appear to be associated with a relatively high blood pressure: wounds of the liver and kidney often exhibit a systolic reading of from 130 to 140 mm., and this even in cases in which the hemorrhage is considerable. We have observed a tear of the liver in the case of a soldier run over by a limber, in which the systolic pressure of 140 was maintained for twenty-four hours. At the end of this time the pressure fell suddenly and the patient succumbed.

5. Perforating wounds of the viscera which do not open into the peritoneal cavity are associated with a practically normal blood pressure, as in Case 1, an extraperitoneal wound of the colon, and Case 11, an extraperitoneal wound of the rectum.

6. Large wounds of the parietes are generally associated with a lower blood pressure than small wounds, even though the former may have produced much less visceral destruction than the latter. This is probably explained by the fact that in the former instance peritoneal blood readily escapes, while in the latter the hemorrhage continues to be retained.

We have made several observations regarding the effect on the blood pressure of opening the peritoneal cavity. Briefly, we found that if the abdominal cavity contained a large amount of blood, there was a very rapid fall of pressure as soon as the peritoneal cavity was opened, and the blood allowed to escape. If, on the other hand, the abdominal cavity did not contain much blood, the opening of the abdomen was followed by a temporary rise in blood pressure by as much as 20 mm. After ten minutes the blood pressure fell to slightly below the figure that it registered before operation.

### C. CHEST WOUNDS.

A summary of chest cases is given in Table 2.

From these readings there are a few conclusions that may be drawn:

1. Large open wounds of the chest with free entrance and exit of air are accompanied by a profound fall of blood pressure; this is evidenced in Case 9.

2. Patients with uncomplicated closed wounds of the chest who arrive at the casualty clearing station well cared for show normal pressure.

3. When severe internal hemorrhage has occurred and the patient has been exposed to the cold for some hours, or when infection has become established, hypotension is present and progressive.

TABLE 2.—SUMMARY OF CHEST CASES

| No. | and Clinical Condition | Special Features | Blood Pressure |
|---|---|---|---|
| 1 | G.S.W back and chest (penetrating). | Lower chest ..... | 120— 90 |
| 2 | G.S.W. chest wall .... | Non-perforating ..... | 138—100 |
| 3 | G.S.W. chest (penetrating) | Lower chest ..... | 130— 90 |
| 4 | G.S.W. chest (penetrating) | Upper chest ..... | 90— 60 |
| 5 | G.S.W. chest (penetrating) | Upper chest ..... | 90— 60 |
| 6 | G.S.W. chest wall .... | Non-perforating ..... | 136— 90 |
| 7 | G.S.W. chest (penetrating) | Upper chest ..... | 98— 80 |
| 8 | G.S.W. chest wall ..... | Non-perforating ..... | 128—100 |
| 9 | G.S.W. chest (penetrating) | Very large wound upper chest | 38— ? |
| 10 | G.S.W. chest (perforating) | Multiple wounds ..... | 110— 90 |
| 11 | G.S.W. chest (perforating) | Lower chest involving diaphragm | 138— 90 |
| 12 | G.S.W. chest (penetrating) | Right upper chest | 105— 80 |
| 13 | G.S.W. chest (penetrating) | Lower chest ..... | 130— 95 |
| 14 | G.S.W. chest (penetrating) | Multiple wounds ..... | 110— 90 |
| 15 | B.W. chest (perforating) | Upper chest ..... | 170—100 |
| 16 | G.S.W. chest (penetrating) | Surgical emphysema ..... | 130— 80 |
| 17 | Shell wound chest .... | Penetrating upper chest .. | 120— 90 |
| 18 | G.S.W. chest ..... | Very small hemothorax.... | 130— 85 |
| 19 | G.S.W. chest ..... | Lower chest, injuring liver and diaphragm | 138—105 |
| 20 | G.S.W. chest (open pneumothorax) | Observed in A.D.S. | 70— 35 |

4. Patients whose chest wounds are complicated by perforation or laceration of the diaphragm behave in the same way as Class 2 or 3.

The high pressure recorded in Case 15, under the influence of rest and morphin, rapidly dropped to 120, and remained so for the remainder of the time the patient was under observation. In Case 16 the pressure gradually dropped with the onset of a septic pericarditis.

## D. MULTIPLE WOUNDS AND WOUNDS OF EXTREMITIES

Out of a large number of observations we have selected a summary, representative of different types of wounds (Table 3).

TABLE 3.—SUMMARY OF CASES

| No. and Clinical Condition | Special Features | Blood Pressure |
|---|---|---|
| G.S.W Fractures—Lower Extremity— | | |
| 1 G.S.W. fracture—both thighs | Moderate hemorrhage .... | 100— 85 |
| 2 G.S.W. fracture—tibia.. | Moderate hemorrhage .... | 110— 90 |
| 3 G.S.W. fracture—both legs | Severe hemorrhage ..... | 68— 50 |
| 4 G.S.W. fracture—knee.. | Severe hemorrhage ..... | 30— |
| 5 G.S.W. fracture—tibia.. | Apparently slight hemorrhage | 70— 50 |
| 6 G.S.W. fracture—tibia.. | Severe injury, but slight hemorrhage | 143—100 |
| 7 G.S.W. fracture—both legs | Moderate hemorrhage .... | 96— 80 |
| 8 G.S.W. fracture—leg .. | Severe hemorrhage ..... | 100— 72 |
| 9 G.S.W. fracture—femur. | No hemorrhage ..... | 160—10 |
| 10 G.S.W. fracture—femur. | Moderate hemorrhage .... | 115— |
| 11 G.S.W. fracture—femur. | Moderate hemorrhage .... | 112— |
| 12 G.S.W. fracture—femur. | Seen in A.D.S. (two hours' carry) | 98— 70 |
| 13 G.S.W. fracture—tibia.. | Considerable hemorrhage.. | 90— 60 |
| 14 G.S.W. fracture—tibia.. | Hemorrhage and sepsis... | 65— 30 |
| 15 G.S.W. fracture—fibula. | Seen in A.D.S.; much pain | 140— |
| G.S.W Fracture—Upper Extremity— | | |
| 16 G.S.W. fracture—humerus | Slight hemorrhage ..... | 148—100 |
| 17 G.S.W. fracture—forearm | Severe hemorrhage ..... | 103— 80 |
| 18 G.S.W. fracture—humerus | Severe hemorrhage ..... | 105— 80 |
| 19 G.S.W. fracture—forearm | Severe hemorrhage ..... | 105— 80 |
| Face Wounds— | | |
| 20 Severe face wound .... | Severe hemorrhage ..... | 114— 80 |
| 21 Face wound and jam... | ..... | 128—100 |
| 22 Flesh wound ..... | Severe hemorrhage ..... | 138—100 |
| Simple Flesh Wounds— | | |
| 23 Flesh wound—arm ..... | ..... | 140—100 |
| 24 Flesh wound—leg ..... | ..... | 170—100 |
| 25 Flesh wound—arm ..... | ..... | 138— 90 |
| Multiple Wounds— | | |
| 26 Multiple wounds—legs.. | Slight hemorrhage ..... | 110— 90 |
| 27 Multiple wounds ..... | Slight hemorrhage ..... | 128—108 |
| 28 Multiple wounds ..... | Slight hemorrhage ..... | 118— 98 |
| 29 Multiple wounds ..... | Slight hemorrhage ..... | 100— 75 |
| 30 Multiple wounds ..... | Slight hemorrhage ..... | 100— 80 |

1. Compound fracture of the lower extremity, seen in the casualty clearing station, was generally associated with a considerable fall in blood pressure, more marked when the fracture affected the region of the knee joint. Undoubtedly the blood pressure read-

ing was largely affected by the amount of hemorrhage that had occurred. One finds in such a case as Case 6—a severe compound fracture of the tibia with a very small amount of hemorrhage—a systolic blood pressure registered of 143. On the other hand, Case 4—a compound fracture in the neighborhood of the knee, accompanied by severe hemorrhage—registered a systolic blood pressure of only 30.

2. One might expect compound fracture of the upper extremity to affect the blood pressure less than fracture of the lower extremity. We were surprised to find the comparatively low pressure which compound arm fractures generally registered; the remarks that are made as regards hemorrhage in wounds of the lower extremity equally apply in this connection.

3. In face wounds there is not much alteration of the blood pressure, unless there is an associated compound fracture of the face bones, when the pressure is generally lowered.

4. Multiple wounds of the body and extremities were accompanied by a considerable fall in blood pressure.

## Blood Pressure Readings as Studied Subsequent to Various Intravenous Transfusions

### A. PHYSIOLOGIC SODIUM CHLORID

For many years the intravenous infusion of physiologic sodium chlorid solutions (0.9 per cent. sodium chlorid) has been extensively used in shock and in collapse after hemorrhage. We confess we have been greatly disappointed with the results obtained, and blood pressure readings confirm the clinical disappointments that we have experienced. Chart 5 is typical of many that we have recorded.

Private C. M. was admitted with a simple fracture of the right humerus and a severe shell wound, involving the right knee. The wound was sustained at 9 p. m., and he was admitted to the advanced dressing station by 10 p. m. He was then in a collapsed and pulseless condition. He was treated by the application of warmth, and half a pint of physiologic sodium chlorid solution was given subcutaneously in each flank. By 1 a. m., that is, three hours after admission to the advanced dressing station, his condition was said to have so

far improved as to warrant his removal to the casualty clearing station. On admission to the casualty clearing station he was found to be extremely collapsed; a radial pulse could only occasionally be felt; the systolic blood pressure barely registered 30 mm. of mercury; no diastolic pressure was read. Two pints of physiologic sodium chlorid solution were administered intravenously; immediately the systolic pressure rose to 75 mm., but after a period of about twenty minutes it

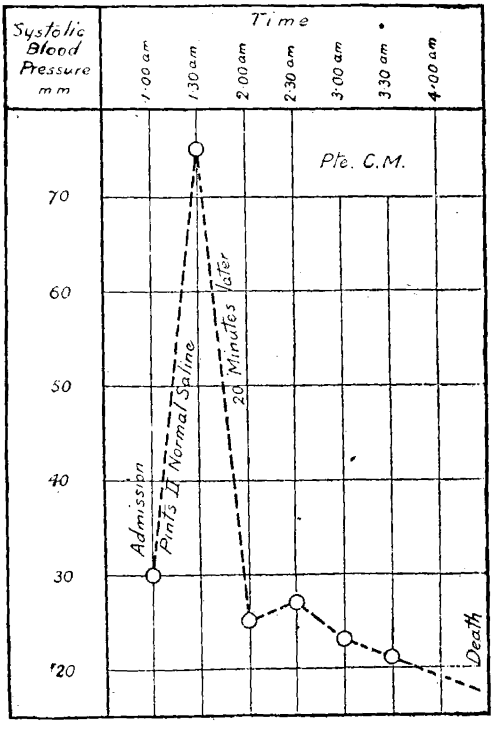


Chart 5.—Systolic blood pressure with administration of 2 pints of physiologic sodium chlorid solution.

rapidly began to fall, and within one hour after the administration it had fallen actually to a lower level than before the infusion had been given. There was no further rally, and death occurred about three hours later.

The blood pressure (systolic) readings are graphically recorded in Chart 5.

It would appear that the introduction of the physiologic sodium chlorid solution induced a condition of

hydremic plethora—that is, a dilution and increase in the total volume of the blood. The kidneys, the skin and the lymph channels excrete the excess of fluid, there is profuse perspiration, and presently the blood is actually less in bulk and more concentrated than it was before. This is in keeping with the observations of Lazarus Barlow, who has shown that the specific gravity of the blood at first falls from 1.064 to 1.054, and later rises to 1.067; in other words, there is at first a dilution of the blood and afterward an actual concentration.

### B. RINGER'S SOLUTION AND ITS MODIFICATIONS

The fluid known as Ringer's solution has the following composition: sodium chlorid, 0.9 per cent.; potassium, 0.03 per cent.; calcium, 0.02 per cent., and a trace of sodium carbonate.

In place of this we have been using a hypertonic solution, as recommended in a publication of the Medical Research Committee, and constituted thus: sodium chlorid, 2 gm.; potassium chlorid, 0.05 gm.; calcium chlorid, 0.05 gm.; water, 100 c.c.

From its intravenous use we have obtained satisfactory results, and the clinical history and blood pressure chart of a typical and successful case is recorded.

Sergeant A., R. F. A., was admitted with a severe shell wound of the pelvis, injury to the bladder and prostate and profuse bleeding. On admission the respective pressures were: systolic, 90 mm.; diastolic, 60 mm. Immediate operation was necessary. Subsequently there was intense collapse—the systolic pressure registered only 25 mm., the diastolic pressure about 20 mm. Two pints of hypertonic solution were administered intravenously, and in immediate response the pressures rose, respectively, to 100 mm. and 80 mm. One hour later they had fallen to 65 mm. and 50 mm. Thereafter the pressure steadily began to rise, and six hours later it had reached 100 mm. and 80 mm.

The readings were maintained, and an eventual recovery was made.

Chart 6 graphically illustrates the blood pressure (systolic) readings in this case.

### C. COLLOIDAL SOLUTION

At the suggestion of Prof. W. Bayliss and Col. Cuthbert Wallace, A. M. S., we have used intravenously in

cases of hypotension due to profound shock and the toxemia of gas gangrene a solution of gum acacia.

We had first arrived at the formula we have used with undermentioned cases, when the report of the Medical Research Committee was published, March 24. The formula we employ is as follows: calcium chlorid (B. P.), 0.075 gm.; sodium chlorid, 1.325 gm.; gum acacia, 2 gm.; water, 100 c.c. A double strength solution is conveniently made and kept in sterilized bottles—sterilization should be repeated each week

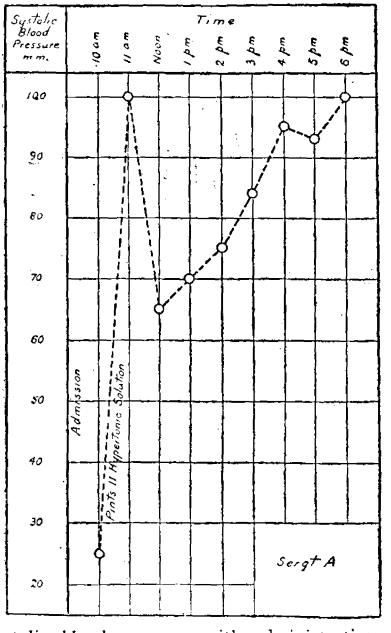


Chart 6.—Systolic blood pressure with administration of 2 pints of hypertonic solution.

This solution is made up as follows: calcium chlorid (B. P.), 13 grains; sodium chlorid, 232 grains; gum acacia, 350 grains; water, 1 pint.

In comparison with the formula recommended by the Medical Research Committee, we give less sodium chlorid and more calcium chlorid, while we omit the potassium salt entirely. With this solution good results have been observed when from 15 to 20 or even 30 ounces of the solution are given intravenously. The

solution was given slowly at the rate of 5 ounces in five minutes, and its heat maintained at a temperature of about 120 F. in the reservoir. It passed from the reservoir through a compte-gouttes chamber and entered the body by means of a small glass cannula tied into cephalic or saphenous veins. The infusion was repeated after twelve or twenty-four hours, if necessary.

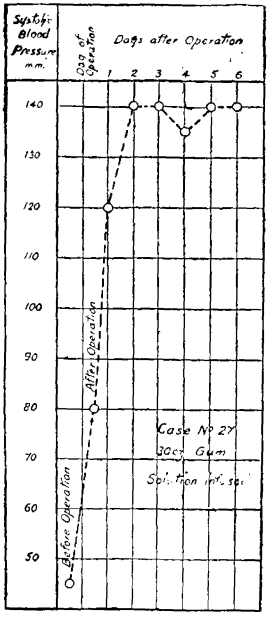


Chart 7.—Systolic blood pressure with administration of an infusion of 30 ounces of gum solution.

Chart 7, taken from Case 27, is added to illustrate graphically the blood pressure (systolic) response in a successful case.

From a study of the cases quoted above it may be seen that the pressure almost invariably rises within a few minutes of the infusion being begun. This fact often enables the necessary operative treatment to be carried out, even in the worst type of case which presents itself at a casualty clearing station—such a case, for example, as shows a hypotension pulse with general collapse and associated with shock, hemorrhage and toxemia.

TABLE 4.—SUMMARY OF CASES IN WHICH GUM TRANSFUSION WAS EMPLOYED

| Case | Clinical Condition | Special Features | | | Amount | Pressure | | Days | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Shock | Hem. | G.G.* | | Before | After | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| | | | | | Oz. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. |
| 1 | Compound fracture of femur | + | + | — | 10 | 50 | 120 | 115 | — | — | — | — | — | — |
| 2 | Compound fracture of femur | + | + | + | 15 | 70 | 115 | 120 | 140 | — | — | — | — | — |
| 3 | Compound fracture of femur | + | + | — | 20 | 90 | 90 | — | — | — | — | — | — | — |
| 4 | Compound fracture of femur | + | + | ++ | 15 | 70 | 80 | 100 | — | — | — | — | — | — |
| 5 | Compound fracture of femur | ++ | + | — | 20 | 50 | 125 | — | — | — | — | — | — | — |
| 6 | Torn femoral vein | — | ++ | — | 15 | 90 | 100 | 108 | — | — | — | — | — | — |
| 7 | Gas gangrene of thigh | — | — | + | 20 | 70 | 110 | 110 | 110 | 110 | 108 | 110 | — | — |
| 8 | Compound fracture of tibia | + | + | ++ | 15 | 70 | 90 | 110 | 118 | 118 | — | — | — | — |
| 9 | Perforating wound of abdomen | + | + | — | 15 | 80 | 110 | 110 | 120 | 112 | 110 | — | — | — |
| 10 | Compound fracture of tibia (amputation) | + | + | + | 20 | 70 | 120 | — | — | — | — | — | — | — |
| 11 | Compound fracture of tibia | + | + | + | 15 | 80 | 130 | 110 | 130 | — | — | — | — | — |
| 12 | Compound fracture of tibia (amputation) | — | — | — | 10 | 140 | 140 | 120 | — | — | — | — | — | — |
| 13 | Wound of calf | — | — | ++ | 15 | 20 | 115 | 65 | Died | — | — | — | — | — |
| 14 | Massive wound of shoulder | + | + | + | 15 | 50 | 90 | 90 | Died | — | — | — | — | — |
| 15 | Compound fracture of tibia (amputation) | + | + | + | 20 | 65 | 80 | 100 | 110 | — | — | — | — | — |
| 16 | G.S.W. thigh | — | — | ++ | 15 | 120 | 115 | 80 | Died | — | — | — | — | — |
| 17 | Compound fracture of femur and pelvia | ++ | + | — | 20 | 80 | 80 | Died | — | — | — | — | — | — |
| 18 | Compound fracture of humerus (amptn.) | + | + | + | 20 | 80 | 85 | 110 | 118 | 120 | — | — | — | — |
| 19 | G.S.W. thigh | — | — | + | 15 | 100 | 108 | 90 | 112 | 108 | 112 | 104 | 98 | 100 |
| 20 | Compound fracture of fibula (amputation) | — | — | + | 15 | 100 | 100 | 98 | 102 | 90 | — | — | — | — |
| 21 | Perforating wound of buttock | + | + | + | 15 | 90 | 100 | 95 | 90 | 104 | 90 | 100 | — | — |
| 22 | G.S.W. multiple | + | + | + | 12 | 95 | 130 | 115 | 122 | 120 | — | — | — | — |
| 23 | G.S.W. of shoulder (amputation) | + | + | ++ | 15 | 100 | 120 | 90 | 80 | 80 | Died | — | — | — |
| 24 | Compound fracture of femur | + | + | ++ | 15 | 80 | 100 | 112 | 108 | 100 | — | — | — | — |
| | | | | | | | | Repe ated | | 110 | 104 | 120 | 110 | 120 |
| 25 | G.S.W. multiple | + | + | ++ | 20 | 60 | 80 | 75 | Died | — | — | — | — | — |
| 26 | G.S.W. thigh | Chron ic hypo . pulse | | | 20 | 100 | 110 | 90 | 110 | 90 | 100 | — | — | — |
| 27 | G.S.W. abdomen | ++ | ++ | — | 30 | 45 | 80 | 120 | 140 | 140 | 138 | 140 | — | — |
| 28 | Penetrating buttock | + | + | ++ | 30 | 80 | 90 | Death | — | — | — | — | — | — |
| 29 | G.S.W. scrotum | + | + | + | 10 | 50 | 70 | 100 | 105 | Death | — | — | — | — |
| 30 | Compound fracture of tibia (amputation) | + | + | + | 20 | 80 | 100 | 100 | 104 | — | — | — | — | — |
| 31 | Double amputation both legs | + | ++ | + | 30 | 30 | 85 | 80 | Death | — | — | — | — | — |
| 32 | G.S.W. shoulder | + | ++ | + | 30 | 35 | — | Death | — | — | — | — | — | — |
| 33 | G.S.W. abdomen | + | + | — | 20 | 30 | 90 | 80 | 80 | — | — | — | — | — |
| 34 | Compound fracture of femur | + | + | + | 20 | 30 | 90 | 85 | 90 | — | — | — | — | — |
| 35 | Perforating wound of buttock | + | — | ++ | 20 | ? | 80 | 80 | 85 | 85 | — | — | — | — |

* G.G.— Gas gangrene.

TABLE 5.—PRESSURE OBSERVATIONS DURING CONVALESCENCE

| No. | Clinical Condition | Special Features | Blood Pressure History, Days | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
| | | | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. | Mm. |
| 1 | Penetrating shell wound of leg | Abscess formed third day; note fall of blood pressure | 122 | 130 | 115 | 120 | 130 | 120 | 120 | — | — | — | — | — |
| 2 | Shell wound of knee joint | Slight temperature third day; perfect result | 120 | 130 | 118 | 130 | 130 | 118 | 130 | 130 | 118 | 122 | 138 | 138 |
| 3 | Compound fracture of tibia | Joint infected eighth day; septicemia and death | 150 | 120 | 130 | 120 | 130 | 148 | 138 | 122 | 125 | 110 | 100 | 90 |
| 4 | Shell wound of elbow joint | Healed with slight superficial sepsis | 120 | 115 | 112 | 110 | 110 | 110 | 115 | — | — | — | — | — |
| 5 | Penetrating chest wound | Sepsis, pericarditis | 130 | 115 | 115 | 115 | 100 | 100 | 90 | 90 | — | — | — | — |
| 6 | Penetrating chest wound | Uncomplicated recovery; no hemorrhage in thorax | 120 | 130 | 140 | 140 | 140 | 140 | — | — | — | — | — | — |
| 7 | Perforation of frontal lobe | Operation second day; a febrile convalescence | 120 | 140 | 120 | 130 | 120 | 118 | 120 | 118 | 120 | 120 | 120 | 122 |
| 8 | Penetration of frontal lobe | Slight febrile convalescence | 120 | 110 | 100 | 100 | 105 | 100 | 102 | 100 | 102 | 105 | 100 | 100 |
| 9 | Shell wound of buttock | Pulseless on admission (warm rectal glucose) | 60 | 90 | 90 | 80 | 90 | — | — | — | — | — | — | — |
| 10 | Shell wound of calf | Hypotension; pulse on admission | 90 | 100 | 100 | — | — | — | — | — | — | — | — | — |
| 11 | Wound of knee joint | Second day gangrene developed and amputation done | 130 | 120 | 120 | 130 | 150 | 140 | — | — | — | — | — | — |
| 12 | Shell wound of buttock | Low tension; no sepsis | 98 | 102 | 104 | 102 | 100 | 110 | — | — | — | — | — | — |
| 13 | G.S.W. of thigh | Gas gangrene on second day; pressure fell 30 mm. in four hours | 120 | 100 | 120 | 125 | 130 | 120 | — | — | — | — | — | — |
| 14 | B.W. of thigh | Admitted after fight in air; slight wound | 134 | 120 | 120 | 115 | 112 | — | — | — | — | — | — | — |
| 15 | Slight multiple wounds | A febrile convalescence | 135 | 100 | 100 | 110 | — | — | — | — | — | — | — | — |

When it has been possible to remove the source of infection, the rise of pressure is progressive in the subsequent twenty-four hours, and the pressure is maintained during the early critical days of convalescence.

In Case 27 the patient was admitted apparently moribund. After an hour's rest with warmth and rectal glucose-saline injection, he was submitted to laparotomy; there were about 2 pints of free blood in the peritoneal cavity, and six perforations of the intestine required suture. Intravenous infusion

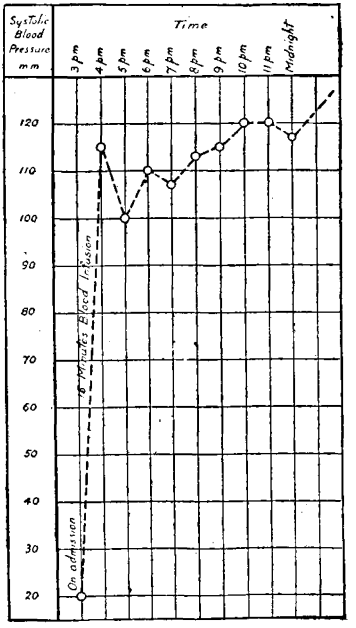


Chart 8.—Systolic blood pressure with fifteen minutes of direct blood transfusion.

of gum was commenced with the operation. When the operation was completed, thirty minutes later, the pressure had risen to 80 mm., and a fair pulse was palpable at the wrist. The following day the pressure was 120 mm.; it rose later to 140 mm., and remained at this figure during an uneventful convalescence.

### D. DIRECT BLOOD TRANSFUSION

We have had an opportunity of observing in several cases the results of direct blood transfusion. The

transfusion was necessary on account of severe arterial hemorrhage, and was carried out by the direct method, as suggested by Basset and Fullerton. The time of the transfusion varied from fifteen to twenty-two minutes. It is difficult to estimate with any degree of exactness how much blood was transferred during these respective periods, but we calculate roughly that from 500 to 700 c.c. would be about the proper estimate.

The following are the notes and the blood pressure chart of one of these cases:

Lieutenant ——, admitted with a severe shell wound of the left thigh: the femoral vessels were torn; no tourniquet had been applied, and the patient was practically moribund from loss of blood.

After the local wound had been dealt with a donor was secured, and direct transfusion of blood begun. Transfusion was continued for fifteen minutes. Clinically there was a most dramatic improvement, and within the course of a few hours the pulse rate had fallen from 150 to 95. The systolic blood pressure on admission was very difficult to estimate; but it was estimated at about 20 mm. Before the transfusion had been completed it rose to 115; one hour later it had fallen to 100. At the end of another hour it had risen to 110, and about this level it remained for the first twenty-four hours; thereafter it attained and remained at a level of about 120.

The blood pressure readings were as shown on charts.

### E. GLUCOSE SOLUTION

We have no records of blood pressure readings following the infusion of hypertonic glucose solutions.

### F. OTHER MEASURES

From time to time various drugs are advocated, such as pituitary solution, epinephrin, caffein, camphor, etc. At present there is not sufficient evidence to show that one of these drugs takes precedence over the others.

### G. CONCLUSIONS

1. In cases of profound shock accompanied by loss of blood, excellent results are obtained from direct blood transfusion.

2. Injection of the calcium hypertonic gum solution will produce an immediate rise of pressure in hemorrhage cases or cases of hypotension, complicated by toxemia.

This rise may tide the patient through an operation. If the source of the infection is removed, the tension will remain supported.

3. In milder cases of shock and hemorrhage, infusion with hypertonic saline is useful.

4. Results obtained after infusion with physiologic sodium chlorid solution have been unsatisfactory.

## Pressure Observations in the First Week of Convalescence as an Aid to Prognosis and to Treatment

An extensive number of pressure charts have been collected, showing the behavior of the tension not only before and during operation, but also in the first week of convalescence. We have thus a series of controls that show such complications as the onset of sepsis or gas gangrene, and illustrate the subsequent history of intravenous infusion for shock, hemorrhage and toxemia.

An uncomplicated wound running a favorable course shows a steadily maintained blood pressure. With the onset of gas gangrene or sepsis there is a sudden fall in pressure (Case 13). Occasionally a long sustained hypotension pressure reading may be observed (Cases 9 and 12), even though the patient is quite well and no sepsis is present.

A steadily rising or maintained high pressure reading, even in a severe wound, may be taken as a most favorable prognostic sign (Cases 2 and 6).

We acknowledge our indebtedness to Col. Cuthbert Wallace, C. M. G., A. M. S., and to Professor Bayliss, for repeated suggestions and assistance; to Lieutenant-Colonel Winder, R. A. M. C., and Lieut.-Col. A. H. Safford, R. A. M. C., for permission to investigate the subject; to Lieut.-Col. T. R. Elliot, R. A. M. C., and the Medical Research Committee for the various instruments which were employed in the investigation.

# SOME ALTERATIONS IN DISTRIBUTION AND CHARACTER OF BLOOD IN SHOCK AND HEMORRHAGE

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## INTRODUCTION

The importance of traumatic shock as a serious complicating factor of wounds and of surgical operations, and its mysterious nature, have combined to stimulate investigation into its causes and into methods of dealing with it. That investigation of shock has been difficult and baffling is indicated by the number and variety of the theories put forth to account for it. The difficulties lie not only in the obscure character of shock itself, but also in complications introduced by attendant conditions, such as hemorrhage and sepsis. Under these circumstances the most hopeful mode of obtaining insight is by gathering as many facts of observation as possible, with the chance that the facts may suggest the nature of the process or processes that are occurring.

The observations of Sherrington and Copeman[1] that intraperitoneal operations on animals raise the specific gravity of the blood, the confirmatory studies of Cobbett[2] and also of Vale,[3] indicating a concentration of the blood in conditions that induce shock, and the experiments of Mann,[4] showing that the amount of blood stagnant in the tissues of an animal in shock may be more than 50 per cent. above the amount in the normal animal—all these evidences indicate that

1. Sherrington and Copeman: Jour. Physiol., 1893, **14**, 83.
2. Cobbett: Shock, in Allbutt: System of Medicine, London, 1897, **3**.
3. Vale: Med. Rec., New York, 1904, **46**, 325.
4. Mann: Surg., Gynec. and Obst., 1915, **65**, 380.

examination of the blood might yield significant facts. Warfare provides shock cases in large numbers. At a casualty clearing station, where only the most severely wounded were admitted for treatment, we have had an unusually favorable opportunity to study their conditions, and at Col. Cuthbert Wallace's suggestion we undertook observations on the physical features of the blood in these cases. The present account is based on records of observation in ninety-eight cases of shock and hemorrhage, and in fourteen control cases.

The routine examination consisted of a count of the red blood corpuscles (referred to henceforth as "red count"), estimation of the hemoglobin by use of the Haldane hemoglobinometer, and determination of the percentage of corpuscles in the blood, either by capillary hematocrit or by larger graduated tubes, if the amount of blood permitted. When desirable to keep the blood for some time unclotted, potassium oxalate crystals were added in minimal amount; but when this was done, the hematocrit readings were made at once in order to avoid any change of corpuscle volume through the action of the added salt.

Although the blood examinations were made in a routine manner, we have kept in mind certain questions to which we hoped to find more or less definite answers:

1. Are these changes in certain physical features of the blood peculiar to cases of shock and of hemorrhage?

2. Are there typical postoperative blood changes in these cases?

3. What are the effects on the blood of intravenous infusions?

4. May the data provided by blood examinations be helpful in prognosis?

We propose to present and consider our results under these headings.

## The Blood Changes Peculiar to Shock

In discussing the blood changes in shock it will be desirable to distinguish between cases of severe or extreme shock, as seen at the casualty clearing station, and those of moderate character. To some extent the

judgment of these conditions is based on an extensive clinical experience of one of us (J. F.) in connection with blood pressure determinations in shock cases.[5] Combined with this clinical judgment were pressure readings. Roughly, the moderate cases had a systolic pressure over 90 mm. of mercury, and the severe cases not over 70 mm.

The first noteworthy characteristic of the blood in shock is a high capillary red count. In Chart 1 is presented the red count in twenty-seven cases classified as severe traumatic shock. In all but eleven of the cases the count was 6 million corpuscles or higher, and in eight cases it was more than 7 million cor-

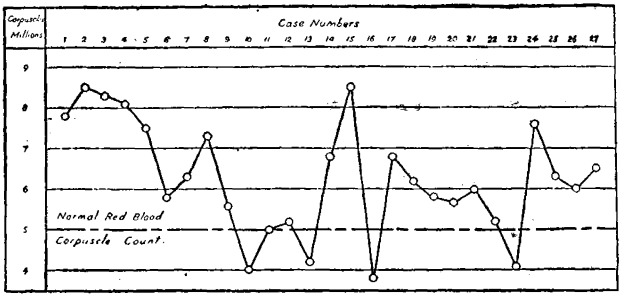


Chart 1.—Red corpuscle counts in twenty-seven cases of severe shock.

puscles.[6] When hemorrhage as a complicating factor tending to reduce the blood count is considered, these high counts are striking. They indicate that in shock a concentration of the blood occurs, at least in the superficial capillaries.

Whether or not the concentration found in capillary blood is true of all the blood can be determined by counting capillary and venous samples taken at the same time. When this is done, a more or less marked discrepancy between the two is revealed. The capillary samples have been taken from widely separated parts of the body—from the lobe of an ear, from a finger or from a toe; the venous samples from an arm vein. In Chart 2 are plotted the observations in seven cases

5. Fraser, John, and Cowell, E. M.: Clinical Study of Blood Pressure in Wound Conditions, p. 9.

6. These counts were made by means of a Thoma instrument made by Hawksley; it was compared with a Thoma-Zeiss instrument, and a difference of only 10,000 corpuscles in 5,000,000 was found.

of severe shock, four cases of moderate shock, and five cases in which no shock was present. The strikingly higher capillary than venous red count in the severe cases, amounting frequently to 2 million corpuscles, was reduced in the moderate cases, but even in these the difference is still nearly a million corpuscles per cubic millimeter.[7] In the final group of cases, in which no shock was present, the capillary count was uniformly slightly higher than the venous, but as compared with the results in the other two groups, the difference is neglible. These observations have been made independently by two observers.

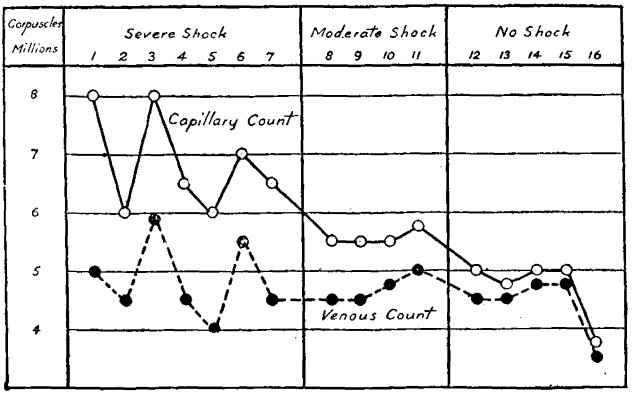


Chart 2.—Comparison of red counts, capillary and venous, in cases of severe and moderate shock, and in patients without shock.

Bürker[8] states that in the same person blood from the finger tip and ear is the same with reference to hemoglobin content and red counts within an error of 1 per cent., and that blood from an elbow vein, when flowing free, is indistinguishable from that of the ear or finger. Control observations made by us on normal individuals did not reveal greater differences than 3 per cent. between capillary and venous counts; and comparative counts of capillary and venous blood taken before rising from bed in the morning proved that the discrepancy is not due merely to inactivity.

7. Capt. E. Emerys-Roberts has reported to us verbally that he has made similar observations in cases of shock, and recently the results have been confirmed by Capt. Eric Taylor.

8. Bürker: Tigerstedt's Handbuch der Physiologischen Methodik, Leipzig, 1912, 2, Part 5, p. 4.

From the foregoing considerations it is clear that the difference between capillary and venous red counts varies roughly with the degree of shock, and, since the venous count is approximately normal, the difference is due to concentration of the blood or stagnation of corpuscles in the capillaries.

In all probability the low temperature typical of patients in shock is an important factor in producing the increased corpuscular content of the capillaries. It is known that blood drawn from a cold finger contains a larger number of corpuscles in a given volume than that drawn from the same finger after it has been warmed.[9] In cases of shock, simultaneous counts of venous blood and samples from capillaries of the ear, the finger, and the mucous membrane of the mouth show significant differences. In one case of slight shock the venous red count was 5,360,000, the ear and finger capillary counts 6,450,000 and 6,280,000, respectively, and the buccal count 5,600,000. The capillaries of the mouth, less exposed to loss of heat than those of the skin, contained blood nearer the venous blood in concentration than did those of the skin, but still indicating stasis.

When the capillary stagnation has become established, it does not promptly disappear. We have seen a patient who had recovered from severe shock, but whose hands from wrist to finger tips, in spite of being warmed, were still bluish-gray with stagnant blood.

Hematocrit determinations of the volume per cent. of corpuscles in capillary and venous blood have also been made. In each instance duplicate tests were carried out in order to avoid any error that might arise from adhesion of blood to the wall of the capillary tube. The results confirmed the discrepancy between capillary and venous samples that was found in the counts. The capillary corpuscle volume was greater than the venous by amounts ranging from 12 to 33 per cent.

The difference between capillary and venous blood in shock was further confirmed by hemoglobin determinations. In cases thus compared, the capillary hemoglobin readings exceeded the venous by amounts ranging from 6 to 29 per cent. In the case cited at

9. Gulland and Goodall: The Blood, London, 1914, p. 61.

the end of the second paragraph above, the hemoglobin reading of the blood in the still abnormal fingers was 114 per cent., that of the recovered capillaries of the ear, 104 per cent.

A comparison of some of the blood counts and hematocrit and hemoglobin determinations in our cases is presented in Table 1.

As mentioned before, shock is frequently complicated by hemorrhage. In these conditions the capillary red count may be low, but when compared with the venous red count the discrepancy between the two at

TABLE 1.—VENOUS AND CAPILLARY RED COUNTS, WITH HEMATOCRIT AND HEMOGLOBIN READINGS, IN SOME CASES OF LOW BLOOD PRESSURE

| Initials* | Blood Pressure | | Red Counts (in millions) | | Hematocrit | | Hemoglobin | |
|---|---|---|---|---|---|---|---|---|
| | Diastolic | Systolic | Venous | Capillary | Venous | Capillary | Venous | Capillary |
| P. K. | 34 | 52 | 3.8 | 5.6 | — | — | — | — |
| A. S. | 38 | 62 | 4.5 | 6.4 | 30 | 41 | — | — |
| E. G. | 40 | 64 | 6.2 | 8.5 | 30 | 47 | 88 | 113 |
| F. H. | (near | death) | 4.0 | 6.0 | 31 | 43 | — | — |
| D. H. | 48 | 64 | 4.2 | 5.5 | 37 | 41 | 80 | 95 |
| S. D. | 48 | 72 | 4.7 | 5.3 | 30 | 35 | 75 | 84 |
| W. W. T. | † | 50 | 5.3 | 6.4 | — | — | 92 | 98 |
| W. C. | 58 | 76 | 4.5 | 5.5 | — | — | — | — |
| S. F. S. | 58 | 80 | 4.9 | 5.3 | — | — | — | — |
| T. R. | 70 | 92 | 5.2 | 5.6 | 39 | 44 | 107 | 111 |
| J. H. C. | 80 | 102 | 5.8 | 6.9 | 41 | 45 | 95 | 105 |

* Further information about some of these patients may be obtained by finding these initials in Table 1, Cannon, W. B.: Acidosis in Cases of Shock, Hemorrhage and Gas Infection.
† Irregular.

once appears. In other words, when hemorrhage complicates shock, the blood in the peripheral capillaries contains relatively more corpuscles in a given volume than that in the veins, though in both the number is reduced.

Observations from day to day on the capillary blood in cases of shock have shown that there is gradually a drop to normal or below in the count and in the hemoglobin and hematocrit readings. In some instances this return to normal has occurred on the second day; in others it may not have been completed for three or four days. A fairly typical case of the slower recovery is the following:

CASE 69 A.—Private L. was admitted suffering from wounds of the face and arm, with fracture of skull; early gas infection, severe; moderate shock.

The red count on admission was 7,510,000; the next day it was still high (7,560,000); on the third day it fell slightly to 7,280,000, and on the fourth day it was 5,610,000. Counts made on five days during the following week disclosed no further important change. The hemoglobin and hematocrit readings likewise fell during the first three days, though not so sharply as the red count. The record is presented in Chart 3.

The influence of cold in producing stagnation or concentration of blood in the capillaries has been mentioned. It is in the capillary region that the corpuscles are most exposed to contact with the vascular wall, that is, in this region and in the finer arterioles friction

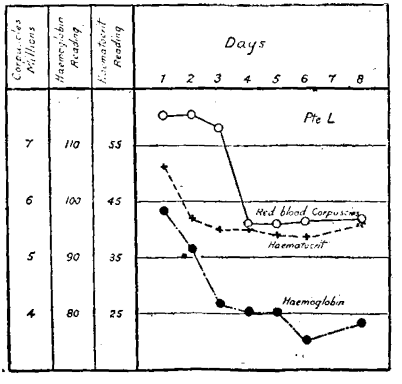


Chart 3 (Private L.).—Count of red corpuscles, with hematocrit and hemoglobin readings.

is greatest, and the energy stored in the arterial pressure is mostly used up. When arterial pressure is low, as in shock, there is naturally a tendency of the blood corpuscles to gather in the place of greatest resistance.[10] And if these channels are differentiated by cold, so that some (the warmer) offer easier courses, and others (the cooler) more difficult courses for the blood to take, the accumulation of corpuscles, especially in the capillaries of the skin and limbs, may reasonably be accounted for as a partial stasis. The blood thus checked in capillary areas would be out of currency, and by failure to return to the heart would contribute to a lowering of arterial pressure.

10. Cohnstein and Zuntz: Arch. f. d. ges. Physiol., 1888, **42**, 326.

## The Blood Changes Peculiar to Hemorrhage

Many wounded men have lost much blood; they are not suffering from infection, and they appear to have only such elements of the shock complex as the hemorrhage itself may induce. The question arises as to whether under these circumstances examination of the blood discloses any features that will differentiate hemorrhage from shock.

Blood counts of hemorrhage cases show several distinctive features. The capillary red count is usually much lower than that of shock alone, but is by no means as low as the pallid appearance of the patient might lead one to suspect. In twenty-one cases classified chiefly as hemorrhage, the average capillary count at the time of admission or shortly thereafter was about 5,000,000 corpuscles, with variations ranging from 5,800,000 to 3,900,000. In a few of these cases in which the venous blood was also counted, it proved considerably lower than the capillary count. This is what might be expected, for in severe hemorrhage the blood pressure is reduced, and the cooling of the body induces stasis, just as in shock.

Another feature of hemorrhage that seems fairly characteristic is a relatively low hemoglobin reading. In the twenty-one cases of hemorrhage, the hemoglobin percentage was 72. If a count of 5,000,000 corpuscles is taken as normal, the color index would be only 0.72, and even with 6,000,000 taken as normal, the index is only 0.9. In shock cases the index is approximately 1, with 6,000,000 corpuscles regarded as the normal number.

The primary reduction in the count and in the hemoglobin percentage in cases of hemorrhage is followed by a further fall. The following instance is illustrative:

Private G. was admitted with severe wounds of the left leg. Both tibial vessels were severed, and there had been profuse bleeding. There was marked pallor of all cutaneous and mucous surfaces. The blood examinations on admission and on successive days disclosed a fall, and later the beginning of recovery. On admission the red count was 5,100,000, the hemoglobin 68 per cent. For the next three days both figures fell; on the third day the red count was 3,070,000, the hemoglobin 39 per cent. This tendency continued, but at a slower rate, until the sixth day, when the count was

1,900,000 and the hemoglobin 35 per cent. From this point a betterment began, and on the eighth day the count was 2,550,000, the hemoglobin 38 per cent. On the fifth day there were present in the blood film very small cells resembling microcytes. Throughout the examination no poikilocytosis was observed.

We have had several opportunities of confirming, on men who have served as donors in the transfusion of blood, the changes seen in the foregoing case. In these conditions there could be no question of shock or sepsis. The initial red count of the case illustrated in Chart 4 was 5,600,000, the hemoglobin 102 per cent. Transfusion was performed by the direct method, and it was estimated that about 700 c.c. of blood passed from the donor. Some hours later the capillary count was still high, 5,700,000, but the hemoglobin reading had fallen to 88 per cent. The changes during the

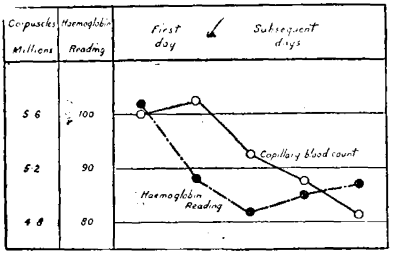


Chart 4.—Red count and hemoglobin percentage in a case of hemorrhage.

next few days are shown in the chart. The typical posthemorrhagic fall occurred, but the hemoglobin was relatively lower than the count. In shock cases with hemorrhage as a noteworthy feature, there is usually a relatively low hemoglobin content of the blood.

We may infer from the foregoing evidence that a low hemoglobin reading is highly suggestive of a hemorrhage's having occurred.

## Postoperative Blood Changes

Tracing the course of blood changes in shock cases has led to numerous observations being made both before and after operation. In all these cases the operation was performed with warmed ether as the

anesthetic. The results of the observations may be thus summarized:

(*a*) When an operation is associated with considerable bleeding, both hemoglobin and red count are reduced; but, as in the hemorrhage cases, the hemoglobin falls relatively more than the count. This observation has been confirmed in numerous instances. The following case is illustrative:

CASE 18 A.—The patient had a severe compound fracture of femur. Before admission the hemorrhage had been slight; the red count was 5,600,000, hemoglobin 90 per cent. The necessary operation was accompanied by much bleeding, and the count then was 4,600,000, the hemoglobin 50 per cent. The fall in the hemoglobin percentage in this case was excessive; as a rule it amounted to about 10 per cent.

(*b*) When the operative hemorrhage is slight, the count and hemoglobin reading after operation are commonly higher than before, and the longer the operation the greater the changes are likely to be. Subjoined is an illustrative case:

CASE 14 A.—A patient was admitted with a wound of the abdomen through which a portion of the small intestine protruded; the intestine itself was damaged. Hemorrhage had been slight. The red count before operation was 6,400,000, hemoglobin 112 per cent. The operation was completed with a minimum of bleeding; about 1 foot of small intestine was resected. The operation lasted forty-five minutes, and at the end the red count was 8,000,000, the hemoglobin 130 per cent.

Perspiration is commonly a prominent feature of these cases during the course of the operation—a condition which suggests that the concentration of the blood that we have noted is at least in part due to loss of fluid from the body. That it is not due solely to a stasis of blood in peripheral capillaries is shown by observation of venous blood before and after operation. In one instance, in which there was profuse sweating during the operative procedure, the venous hematocrit determinations showed a rise of corpuscle volume from 32 to 40 per cent. in forty-two minutes.

A puzzling feature that may be mentioned here is the change observed in the circulating blood when a considerable quantity of blood, previously shed, but retained in one of the body cavities, is removed. The removal is followed by a marked drop in the hemoglobin percentage and in the red count. An explana-

tion of the phenomenon is lacking; it has been repeatedly observed.

CASE 41 A.—The patient had sustained a penetrating wound of the chest, with much hemorrhage into the left pleural cavity. On the day of admission the red count was 8,000,000, hemoglobin 80 per cent.; the next day the count was again 8,000,000, hemoglobin 78 per cent. On the following day 2 pints of fluid blood were aspirated from the chest. Within eighteen hours thereafter the red count had fallen to 5,000,000, and the hemoglobin percentage to 52. Twenty-four hours later the count was 4,500,000, hemoglobin 56 per cent.

In some cases operation seems to induce a fragmentation of the red corpuscles.

## BLOOD CHANGES OBSERVED AFTER VARIOUS INJECTIONS

As means of treating hemorrhage and shock, transfusion of blood and injections of solutions of salt and of gum have been widely employed. We have made observations on the blood before and after such treatment has been tried.

### A. BLOOD TRANSFUSION

From 500 to 700 c.c. of blood have been transfused from donor to recipient, either directly, artery to vein, or by means of the Kimpton tube. No diluting or chemical substance has been added to the blood. Naturally transfusion is done when the blood count is low, and, as is to be expected, a great improvement occurs in the recipient's condition. The following case illustrates the degree of improvement that may take place:

CASE 100 A.—The patient had received severe multiple wounds; one leg had been blown off. The red count before transfusion was 1,900,000, hemoglobin 31 per cent. It was estimated that about 600 c.c. of blood were transfused directly from a donor. Twelve hours later the red count was 3,000,000, hemoglobin 56 per cent. Twenty-four hours later the count was 2,500,000, hemoglobin 60 per cent. Thenceforth there was steady improvement.

### B. INJECTION OF GUM SOLUTION

A 7 per cent. solution of gum acacia in physiologic sodium chlorid solution has been advocated by Bayliss[11] to raise blood pressure in shock and after hemorrhage.

11. Bayliss: Proc. Roy. Soc., 1916, **89**, 380.

We had occasion to note the effect of the injection of this solution on the blood count:

CASE 64 A.—The patient had sustained a severe abdominal wound, with profound collapse. A pint of 6 per cent. solution of gum acacia was injected intravenously. Before the injection the red count was 4,500,000, hemoglobin 84 per cent. Three hours after the injection the count was 3,800,000, and the hemoglobin 78 per cent.

The persistence of the dilution of the blood in this case is noteworthy.

### C. HYPERTONIC SALINE INJECTIONS

The hypertonic solution was made according to the formula: sodium chlorid, 2.0; potassium chlorid, 0.05; calcium chlorid, 0.05, and water 100. Injection of this fluid produces effects that are illustrated in the cases given.

CASE 65 A.—The patient had sustained a compound fracture of femur; the limb was almost severed; there was considerable hemorrhage. In the first blood examination the capillary count was 5,290,000, hemoglobin 84 per cent.; the venous count 4,700,000, hemoglobin 75 per cent. Immediately after operation a pint of hypertonic salt solution was injected intravenously. Five hours later the capillary count was 4,600,000, hemoglobin 70 per cent.

CASE 60 A.—The patient had a compound fracture of tibia and fibula, with considerable bleeding. There was early gas infection. The patient's appearance was pallid. Shortly after admission the capillary red count was 6,330,000, hemoglobin 102 per cent.; the venous count was 4,200,000, hemoglobin 94 per cent. After operation 2 pints of hypertonic salt solution were injected intravenously. Six hours later the capillary count was 4,120,000, hemoglobin 55 per cent.

It is noteworthy in these cases that the capillary red counts, some hours after the injection, were much lower than the original capillary counts, but corresponded fairly closely with the original venous counts. This change may be interpreted as a disappearance of the capillary concentration, probably due in part, at least, to improvement in the circulation. The striking feature, however, is the greatly lowered hemoglobin reading when the larger amount of the hypertonic solution was injected. Why this change occurs is difficult to explain. It is a serious disturbance in an important element of the blood, however, and sug-

gests that care should be exercised to avoid introducing unnecessarily large amounts of hypertonic solutions.

## The Value of Blood Examinations for Prognosis

Only repeated examinations of the blood are of prognostic value; conclusions cannot be drawn from a single observation. After this conditional statement we would call attention to the two following points:

### A. The Significance of Continued Concentration of Peripheral Blood

The concentration of capillary blood, which occurs in shock, if persistent, appears to indicate an unfavorable prognosis, and an increasing concentration is a significant precursor of a fatal outcome. The interrelation between the clinical condition and the persistence of capillary concentration is illustrated in the following cases:

Case 78 A.—The patient was admitted with a bullet wound of the abdomen. Operation disclosed two perforations of the ascending colon. On admission the capillary red count was 8,300,000, hemoglobin 96 per cent. The blood was examined on each of the following four days:

July 13, red count 7,800,000; hemoglobin 104 per cent.
July 14, red count 6,600,000; hemoglobin 98 per cent.
July 15, red count 7,400,000; hemoglobin 98 per cent.
July 16, red count 7,100,000; hemoglobin 99 per cent.

During this period the clinical condition of the patient was precarious; there was restlessness, persistent thirst, and low blood pressure, with rapid pulse.

On the fifth day the capillary blood count suddenly fell to 5,200,000, hemoglobin 82 per cent.; and on the next day still farther, to 3,700,000, hemoglobin 68 per cent. This change synchronized with a striking improvement in the general clinical condition, and an uninterrupted recovery followed.

Case 14 A.—The patient was admitted with a gunshot wound of the abdomen, and perforation of the small intestine. The capillary red count was 6,489,000, hemoglobin 112 per cent. At the end of twenty-four hours the concentration had increased, and the count then was 8,700,000, hemoglobin 130 per cent. This case terminated fatally within the next twenty-four hours.

Whether the *persistent* concentration of the capillary blood is due to a circulation inadequate to establish and maintain a uniform distribution of corpuscles, or

is due to some chemical alteration in the corpuscles or in the capillary walls, we do not wish at this time to consider. The observations recorded in the foregoing cases, however, we have had repeated opportunities to verify.

### B. THE SIGNIFICANCE OF PROGRESSIVE DILUTION OF THE BLOOD

As previously pointed out, the intial concentration of the capillary blood normally passes away after a few days. The blood count falls as if the peripheral blood were being diluted or the stagnant corpuscles being swept away. In association with loss of blood, dilution occurs from increase of plasma, but this process also is short lived. A dilution of the blood which continues to progress beyond the fourth or fifth day after injury is ominous, and the longer the dilution continues the more unfavorable it becomes. In the absence of repeated hemorrhage, progressive dilution signifies the presence of sepsis, and generally the involvement of the blood stream (septicemia). The following case is illustrative:

CASE 4 A.—The patient was admitted with a severe wound of the buttock and high compound fracture of the femur. The daily blood examinations are recorded in Table 2.

TABLE 2.—DAILY EXAMINATIONS IN PROGRESSIVE DILUTION OF BLOOD

| Day | Capillary Red Count | Hemoglobin, per Cent. | Day | Capillary Red Count | Hemoglobin, per Cent. |
|---|---|---|---|---|---|
| 1 | 5,400,000 | 85 | 5 | 2,500,000 | 54 |
| 2 | 4,500,000 | 66 | 6 | 2,300,000 | 56 |
| 3 | 4,500,000 | 62 | 7 | 2,200,000 | 54 |
| 4 | 3,900,000 | 61 | 8 | 2,000,000 | 50 |

The local wounds were irrigated with flavine, and were doing so well that it was difficult to account for the progressive deterioration of the blood. On the eighth day the patient left our care, but we learned afterward that the deterioration continued, and eventually the patient succumbed to a septicemia.

We have recorded the foregoing case as a single instance of the progressive dilution of the blood, obscure in origin, and followed by septicemia, but we have made several confirmatory observations.

## Summary

In cases of shock as seen at a casualty clearing station in conditions of warfare, the red count of blood, taken from various capillaries, is higher than that of blood taken from a vein. The discrepancy is greater the more profound the shock, and not infrequently is as much as 2,000,000 corpuscles per cubic millimeter. Since the venous count is approximately normal, the condition is due to a stagnation of corpuscles in the capillaries. The observations by means of blood counting have been confirmed by hematocrit and hemoglobin determinations.

This condition once established in shock is only gradually recovered from; the recovery sometimes requires two or three days.

After hemorrhage, and in cases of shock complicated with hemorrhage, the hemoglobin reading is relatively low compared with the red count.

After operation attended by hemorrhage, the hemoglobin reading is again relatively low compared with the count. If the operation has not been accompanied by hemorrhage, the count and the hemoglobin content of the blood may be higher than before, probably owing to loss of fluid from the body.

Transfusion of blood naturally raises both the count and the hemoglobin reading. Injection of a gum solution leads to a dilution of the blood that may persist for some hours. Intravenous administration of a large amount (2 pints) of hypertonic salt solution may markedly reduce the hemoglobin content of the blood; a smaller amount (1 pint) in our experience has not had this effect. Injection of the salt solution reduces the capillary stasis.

Continued concentration of the capillary blood for several days after injury accompanies a continued unfavorable clinical condition. Disappearance of the concentration is a signal of improvement. Continued dilution of the blood, after the fourth or fifth day, is ominous.

# ACIDOSIS IN CASES OF SHOCK, HEMORRHAGE AND GAS INFECTION

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## INTRODUCTION

In the attempt to obtain more facts concerning the conditions that prevail in cases of persistent low blood pressure, it seemed desirable to examine the blood for certain chemical alterations. Among these were changes in the alkali reserve and in the sugar content. A reduction of the alkali reserve would be indicated specifically by less than the normal amount of sodium bicarbonate in the blood. This is the condition of "acidosis" in the sense defined by L. J. Henderson[1] and by Van Slyke and Cullen.[2] The sugar content might be significant as to the nature of the acidosis, and would also throw light on other processes in the body.

The circumstances under which these studies have been conducted required the use of simple apparatus. Fortunately, the instrument invented by Van Slyke,[3] by which the capacity of the blood plasma to take up carbon dioxid can be determined rapidly and accurately permits observations on this feature of the blood to be made even where few laboratory conveniences are available. The results given by this instrument are expressed in volumes per cent. of carbon dioxid, which the plasma holds after being exposed to an atmosphere containing 5.5 per cent. of the gas (the concentration existing in the alveoli, and present in the final air of an extreme expiration.) Any figures lower than 50 per cent. in adults indicate acidosis. In drawing the blood, a syringe was used. In most cases no difficulty was found in thrusting the needle into an elbow vein made prominent by drawing a rubber tube around the upper arm. Care was exercised not to exert such

1. Henderson, L. J.: Science, 1913, **37**, 389.
2. Van Slyke and Cullen: Jour. Biol. Chem., 1917, **30**, 291.
3. Van Slyke: Jour. Biol. Chem., 1917, **30**, 347.

pressure when the tube was fastened as to shut off the arterial flow. As the blood entered the syringe, the piston was gently withdrawn. When sufficient blood had been taken (about 10 c.c.), the tube was loosened and the needle then withdrawn. The blood was transferred to a flask containing a few crystals of neutral potassium oxalate. Agitation of the sample was avoided. The plasma was promptly separated in a centrifuge. All utensils used were carefully washed with water, alcohol and ether. In making the determinations a check was invariably obtained, and in most cases a further exposure of the plasma to the alveolar air was made before the second reading. All the results presented have the values gained by these precautions.

The methods employed in estimating blood sugar was the easy and reliable one devised by Myers and Bailey.[4]

That the impaired circulation in cases of shock would probably lead to a condition of acidosis was pointed out by Yandell Henderson[5] in 1910. Later Crile[6] and his co-workers reported results which indicated that acidosis is present in various clinical conditions, including shock. Recently Henderson has revised his well known acapnia theory of shock,[7] and now interprets the low carbon dioxid content of the blood as probably not a primary acapnia, but as a consequence of acidosis.

The present study includes an examination of forty-seven cases of low blood pressure, whether due to shock alone or complicated by hemorrhage and gas bacillus infection (Table 1). Observations have been made on the relation of acidosis to blood pressure, pulse and respiration; the sugar content of the blood; the effects of anesthesia and operation on existent acidosis and low blood pressure, and the influence of alkaline treatment in cases of extreme acidosis.

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4. Myers and Bailey: Jour. Biol. Chem., 1916, **24**, 149.
5. Henderson, Yandell: Am. Jour. Physiol., 1910, **27**, 167, 174.
6. Crile: The Origin and Nature of Emotions, Philadelphia, 1915, p. 227.
7. Henderson, Yandell; Prince, A. L., and Haggard, H. W.: Observations on Surgical Shock, THE JOURNAL A. M. A., Sept. 22, 1917, p. 965.

TABLE 1.—OUTLINE OF CASES

| Initials | Date, 1917 | Respiratory Rate | Pulse Rate | Blood Pressure (Systolic, Diastolic) | | $CO_2$ Capacity | | Nature of Wounds. Remarks |
|---|---|---|---|---|---|---|---|---|
| | | | | Before Operation | After Operation | Before Operation | After Operation | |
| | | | | | | % | % | |
| F. A..... | July 3 | — | 136 | 92-58 | — | 41 | — | Gas infection left arm, also scapular muscles. D. |
| S. D..... | July 3 | 33 | 144 | 82-59 | — | 46 | — | G.S.W. thigh muscles; lay out 48 hours; gas infection. D. |
| A. J. R.... | July 3 | 48 | 132 | 63-32(?) | — | 24 | — | G.S.W. both legs, left foot, left arm; typical "air hunger" respiration; no radial pulse. D. |
| W. G..... | July 4 | 32 | 140 | 68-50 | — | 40 | — | G.S.W. buttock and right foot; not much hemorrhage; gas infection. D. |
| H. J. H.... | July 7 | 32 | 144 | 62-40 | — | 37 | — | Shell fracturing right femur, left tibia, left fibula; penetrating chest wound right axilla; lips and finger nails bluish; second $CO_2$ reading taken at death; other readings slightly before. D. |
| | July 7 | 42 | 144 | 58-42 | — | 22 | — | |
| V. G. G..... | July 8 | 50 | 152 | 59-38 | — | 24 | — | G.S.W. chest, leg, abdomen, colon, face; blood taken as dying. D. |
| H..... | July 10 | 24 | 144 | 78-56 | — | 30 | — | G.S.W. loin, elbow; multiple tears in intestine; lips, fingers, ears, forearms cyanotic; legs with cyanotic patches. D. |
| P. K..... | July 12 | 32 | 126 | 52-34 | — | 20 | — | G.S.W. face, fracture left femur, right tibia; blood taken at death; gas in left thigh. D. |
| A. S..... | July 12 | 34 | 144 | 62-38 | — | 35 | — | G.S.W. right arm and side, buttock and abdomen; gas in buttock. D. |
| E. G..... | July 14 | 31 | 144 | 64-40 | — | 21 | — | Compound fracture left ulna and radius, arm nearly severed; G.S.W. right arm and left side; intestine and omentum protruding; blood taken as dying. D. |
| C. P. H.... | July 15 | 20 | 144 | 76-46 | — | 41 | 28 | Sh. wd. right thigh (compound fracture); fracture left arm; face wounds. D. |
| T. R..... | July 15 | 44 | 144 | 92-70 | — | 49 | — | Bullet wound chest; lips and hands blue; temperature 95, mouth. E. |
| J. H. C..... | July 25 | 15 | 72 | 102-80 | 50-28 | 47 | 40 | Sh. wd. right arm, compound fracture; shock developed in operation; $NaHCO_3$ by mouth. E. |
| | July 26 | — | 120 | 112-88 | — | 66 | — | |
| O. B..... | July 26 | 20 | 120 | 102-64 | — | 48 | — | Wounds of back, muscles and spines of vetebrae, ? abdomen. D. |
| S. D..... | July 27 | 26 | 120 | 72-48 | 54-20 | 38 | — | Sh. wd. right leg; severe comminuted compound fracture; much bleeding. Given $NaHCO_3$ by mouth. E. |
| | July 28 | 20 | 96 | 102-68 | — | — | — | |
| J. F. J..... | July 27 | 24 | 134 | 80-58 | — | 43 | — | Multiple sh. wd. right leg, left arm, right forearm, chest, right hand. D. |
| F. A. T..... | July 31 | 24 | 136 | 98-80 | 64-48 | 56 | 40 | Compound fracture left tibia and fibula; many slight wounds; early gas infection. D. |
| S. W. B.... | Aug. 1 | 60 | 144 | — | 56-20 | — | 26 | Wound right leg; readings taken after operation; respiration rapid and deep; air hunger. D. |
| F. L..... | Aug. 1 | 22 | 136 | 92-62 | — | 47 | — | Sh. wd. left arm (compound fracture); very thirsty. E. |
| W. C..... | Aug. 9 | 24 | 116 | 76-58 | — | 49 | 40 | Wound right arm perforating brachial artery and vein; much hemorrhage; pulseless on admission. E. |
| H. T..... | Aug. 10 | 28 | 96 | 70-48 | — | — | — | G.S.W. right chest; hemorrhage; given glucose and $NaHCO_3$ every two hours during first night. E. |
| | Aug. 11 | 32 | 96 | 88-64 | 82-58 | 67 | 61 | |
| H. H..... | Aug. 15 | 32 | 144 | 78-56 | — | 48 | — | Wounds both buttocks; later extensive gas infection. D. |
| B. S..... | Aug. 18 | 28 | 120 | 82-58 | 58-36 | 58 | 46 | G.S.W. left thigh, arm, face, side (penetrating abdomen); four tears in colon, shock, gas nfection, hemorrhage. D. |
| W. A. T.... | Aug. 21 | 16 | 104 | 76-36 | 62-28 | 46 | 38 | G.S.W. buttocks and perineum, muscles below buttocks mashed and torn; some gas infection. D. |
| R. G..... | Aug. 23 | 32 | 144 | 50-32 | — | 50 | — | Compound fracture femur; multiple sh. wounds, femur, buttock, chest; severe gas infection; had been given $NaCHO_3$ and glucose by mouth. D. |
| G. J. H. H. | Aug. 24 | 24 | 138 | 90-62 | 64-46 | 50 | 44 | Sh. wds. right ankle, left leg (fractured), buttock; sighing respiration. E. |
| T. E. C..... | Sept. 6 | 30 | 136 | 66-42 | — | 42 | — | G.S.W. right buttock, considerable hemorrhage, lips, face, conjunctiva pale. D. |
| G. K..... | Sept. 7 | 30 | 144 | 84-60 | 58-36 | 46 | 27 | G.S.W. both arms, left thigh, left foot, compound fracture right thigh; respiration rose to 48 after operation; $NaHCO_3$ lowered it to 25. D. |
| G. W..... | Sept. 8 | 28 | 130 | 74-46 | — | 48 | — | G.S.W. right leg, left foot; compound comminuted fracture femur. D. |
| F. B. N..... | Sept. 9 | 26 | 144 | 72-42 | 104-50 | 36 | — | G.S.W. shattering foot, wounds right thigh, right shoulder; alkaline prophylaxis. E. |
| J. S..... | Sept. 10 | 30 | 140 | 42-28 | 88-44 | — | — | G.S.W. smashing right foot; left foot, both hands, and eye wounded; alkaline prophylaxis. D. next day. |
| G. D..... | Sept. 12 | 28 | 106 | 90-62 | — | 50 | — | G.S.W. arm (fractured), left leg (fractured), wounds right thigh and abdomen; temperature 95 by mouth; D. in anesthesia. |
| G. R..... | Sept. 14 | 20 | 120 | 92-62 | 74-42 | 58 | 46 | G.S.W. left leg, right foot blown off; much sweating during operaton; corpuscle volume rose 32 to 40 per cent. E. |
| G. L..... | Sept. 14 | 22 | 128 | 68-46 | 66-38 | 39 | 39 | G.S.W.; compound fracture right femur; multiple small wounds left thigh, left foot, right elbow; much hemorrhage; alkaline prophylaxis. D. Secondary gas infection. |
| A. H. P.... | Sept. 16 | 20 | 128 | 72-48 | — | 47 | — | Multiple wounds, shoulder, both buttocks, back; shot through left kidney. D. |
| W. S..... | Sept. 16 | 24 | 132 | 98-84 | — | 49 | — | G.S.W. left thigh, femoral artery and vein opened; much hemorrhage. D. |
| W. B..... | Sept. 24 | 18 | 132 | 64-36 | — | 36 | — | G.S.W. left leg (compound fracture), scalp wound; slow respiration; cranial (?), morphia (?). D. |
| J. B..... | Sept. 24 | 18 | 114 | 54-20 | 112-40 | 42 | 58 | G.S.W. head, left eye, fracture both bones left leg; left foot blown off; alkaline prophylaxis. D. second day. |
| R. N..... | Sept. 25 | 20 | 118 | 96-50 | 100-60 | 40 | 63 | Severe gas infection leg, compound fracture left tibia and fibula; alkaline prophylaxis. E. |
| F. W..... | Sept. 26 | 28 | 132 | 98-70 | 110-85 | 34 | 68 | Compound fracture left thigh, wound left hand; alkaline prophylaxis. E. |
| W. G..... | Sept. 26 | 28 | 120 | 92-66 | — | 46 | — | Sh. wd. right leg, foot, left thigh. E. |
| W. H..... | Sept. 27 | — | — | 90-40 | 82-42 | 57 | 50 | Sh. wd. back, penetrating abdomen; liver lacerated; much blood in abdomen. D. |
| O. C. R..... | Sept. 28 | — | — | — | — | 50 | — | G.S.W. lower jaw shot away, thigh wound extending to abdomen; much loss of blood; readings one hour and five hours after wound. D. |
| | Sept. 28 | 28 | 132 | 42-28 | — | 40 | — | |
| H. G..... | Oct. 1 | 24 | 136 | 70-46 | — | 52 | — | G.S.W. face, neck, left foot. E. |
| A. S..... | Oct. 3 | 20 | 122 | 84-56 | 86-62 | 60 | 52 | Bomb wound buttock, penetrating abdomen; nine tears in intestines; much bleeding. E. |
| H. H..... | Oct. 3 | — | — | 62-50 | 62-46 | 42 | — | Left foot blown off; alkaline prophylaxis. E. |
| | Oct. 4 | 24 | 96 | 104-72 | — | — | — | |
| W. H. S.... | Oct. 5 | — | 80 | 120-90 | 95-60 | 50 | 44 | Compound fracture skull; shock developed during operation. E. |

NOTE.—G.S.W. = gunshot wound. Sh. wd. = shell wound. Alkaline prophylaxis = injection of 4 per cent. sodium bicarbonate at start of operation (see Cannon, W. B.; Fraser, John, and Cowell, E. M.: The Preventive Treatment of Wound Shock). D. = died. E. = evacuated base hospital. The data given in this table are mainly based on examinations made shortly after arrival of the patient in the casualty clearing station or at the time of operation. Numerous other observations were made in the cases, some of which are reported in these papers in connection with other features of shock; identification by initials will usually correlate the cases.

## The Relation of Acidosis to Blood Pressure, Pulse and Respiration

### A. Relation to Blood Pressure

In forty-seven different coincident determinations of blood pressure and carbon dioxid capacity, a rough relation between the two was found. In general the lower the blood pressure the lower the alkaline reserve, that is, the greater the acidosis. This relation is illustrated in Table 2.

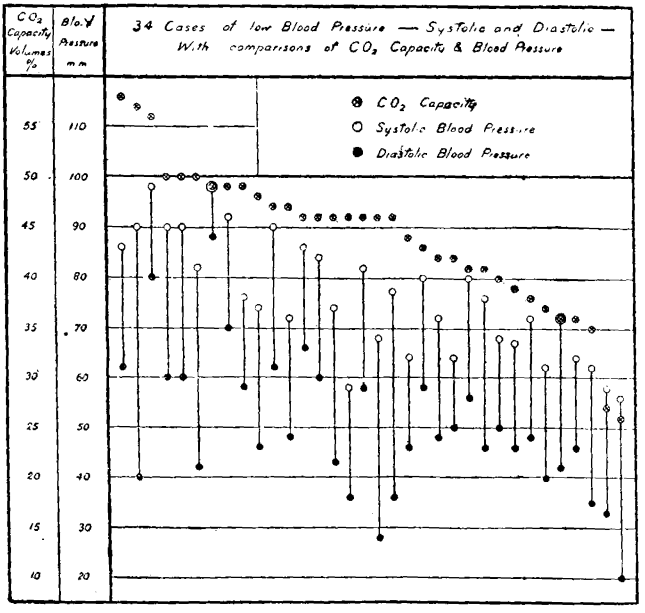
TABLE 2.—RELATION BETWEEN CARBON DIOXID CAPACITY AND BLOOD PRESSURE

| No. of Cases | Carbon Dioxid Capacity | Mean Arterial Pressure |
|---|---|---|
| 6 | 20 to 29 (av. 24) | 49 mm. hg |
| 8 | 30 to 39 (av. 35) | 59 mm. hg |
| 26 | 40 to 49 (av. 44) | 61 mm. hg |
| 7 | 50 to 59 (av. 53) | 69 mm. hg |

It is noteworthy that in forty of the forty-seven cases the mean blood pressure was below 60 mm. of mercury, and that in all these there was a carbon dioxid capacity less than 50 volumes per cent.—a condition of acidosis was present. Furthermore, as the average carbon dioxid capacity was low, the average mean pressure was likewise low. In the chart are presented the records of blood pressure, systolic and diastolic, in thirty-five cases of shock, hemorrhage and gas infection, arranged in the order of decreasing carbon dioxid capacity (increasing acidosis). From this group moribund patients treated with alkaline drink have been eliminated. The first six of these cases had a carbon dioxid capacity of 50 per cent. or higher, and are not to be regarded, therefore, as instances of acidosis. The remaining twenty-nine cases range roughly in correspondence with the state of the blood.

In ten cases classified as uncomplicated shock, the average systolic and diastolic pressures when the blood samples were taken were 77 and 52 mm. of mercury, and the average carbon dioxid capacity was 41 per cent. In seven cases of clear hemorrhage, the relation again was 77 and 52 in blood pressure to 47 per cent. carbon dioxid capacity. And in thirteen cases with gas infection prominent, the average pressures were

70 and 43, with 38 per cent. as the average of the carbon dioxid readings. These groups are perhaps too small to permit conclusions to be drawn. The figures suggest, however, that hemorrhage alone is not attended by as great a reduction of the alkali reserve as is shock (when the blood pressures are equally reduced), and again, when the blood pressure is low, as in the cases of gas infection, the alkali reserve is likely to be correspondingly low.




Records of blood pressure in thirty-five cases of shock, hemorrhage and gas infection, arranged in the order of decreasing carbon dioxid capacity (increasing acidosis).

These observations add another to the known similarities between hemorrhage and shock. In both, the alkaline reserve is lessened. Milroy[8] has recently pointed out that hemorrhage, experimentally induced, is attended by reduction of reserve alkali, and that thereupon exposure of the plasma to the same carbon dioxid concentration as before the hemorrhage devel-

8. Milroy: Jour. Physiol., 1917, **51**, 272.

ops an H-ion concentration considerably higher than before.

In the cases under consideration no observations were made on the H-ion concentration of the blood. Probably the concentration was not much raised when the alkali reserve was only slightly below normal. The activity of the respiratory center in some cases, however, indicated a markedly increased concentration. These conditions will be dealt with later.

From the evidence presented above, the conclusion is warranted that bodily states characterized by reduced blood pressure and consequently by defective circulation are accompanied by a diminished alkali reserve, and that as a general rule the lower the pressure the lower the reserve. This acidosis should not be compared with that which may occur acutely after a quick run or other sharp exercise. That may be quite as extreme as any change seen in shock, but it is temporary, and the alkaline stores in cells and in other body fluids than the blood may soon compensate for the sudden reduction of available alkali in the blood, and oxidative processes rapidly restore the normal conditions. In the cases under consideration, on the other hand, some process has been going on for hours (often six or eight) that has brought about the state observable at admission of the patient. The progressive character of the process was shown in a case in which the carbon dioxid capacity one hour after the wounding was 50 per cent., and five hours later, with no corrective treatment, had fallen to 40 per cent. The condition has a gradual rather than an acute onset.

### B. RELATION TO PULSE

With low arterial pressure, a rapid pulse may be expected. In the series of cases here reported the heart was seldom beating faster than 144 per minute. Possibly that is a limiting rate for continued action. The average rates were somewhat below this number. Table 3 is arranged on the basis of increasing diastolic pressures.

Since the average pulse rates are fairly uniform for different ranges of low blood pressure, and since, as shown above, there is a relation between the lowness of the pressure and acidosis, it follows that there is no definite relation to be found in our cases between

the diminished alkali reserve and the rapidity of the heart beat. If the pulse is considered in these cases arranged on the basis of their carbon dioxid capacity, the average rate for a carbon dioxid capacity of 24 per cent. is 136; of 35 per cent., 135, and of 44 per cent., 130.

### C. RELATION TO RESPIRATION

The chemical stimulus increasing respiration is an increase in the H-ion concentration in the arterial blood. As L. J. Henderson[9] has shown, a large amount of acid may be added to a bicarbonate solution similar in concentration to that of the blood before any considerable increase of acidity occurs, so long as the carbon dioxid passes off. The H-ions of the blood do not increase to an important degree, therefore, in

TABLE 3.—THE RELATION OF ARTERIAL PRESSURE TO PULSE

| No. of Cases | Average Pressures | | Pulse | |
|---|---|---|---|---|
| | Systolic | Diastolic | Av. Rate | Range |
| 5 | 51 | 23 | 133 | 114–144 |
| 9 | 60 | 35 | 134 | 108–152 |
| 13 | 72 | 44 | 133 | 112–150 |
| 8 | 80 | 55 | 133 | 116–144 |
| 7 | 89 | 62 | 128 | 106–144 |

spite of reduced alkali, if pulmonary ventilation prevents an accumulation of carbonic acid. Only when this process fails, or acids increase to such a degree as seriously to encroach on the neutralizing power of the bases of the blood, does the increased H-ion content affect in a marked degree the respiratory center. From a study of the alkali reserve, by the Van Slyke method, in a large number of surgical cases Caldwell and Cleveland[10] report no change of respiration when the carbon dioxid capacity was between 43 and 50 per cent., and also none between 36 and 43 per cent. when that condition was stationary; but if the reserve was *diminishing* and had reached that range, hyperpnea was almost always apparent.

The apparatus necessary to record the volume of respired air in the cases under consideration was not

9. Henderson, L. J.: The Fitness of the Environment, New York, 1913, pp. 149-151.
10. Caldwell and Cleveland: Surg., Gynec. and Obst., 1917, **25**, 23.

at hand, but the rate of breathing was taken. In some of these cases the respiratory rate was probably reduced to a greater or less degree by morphin, which was regularly administered to seriously wounded men in amounts varying from ¼ to ½ grain, with an occasional larger dosage. In Table 4, the chest cases and the abdominal cases have been omitted, for in them the pain due to respiratory movement may modify the breathing in a way complicating the influence of the blood.

As the figures show, the rate increased as the alkali reserve fell, but the change, as was to be expected, became more marked as the limit of the reserve was more nearly approached. The character of the respiration was not noteworthy except in the cases of extreme acidosis, that is, with a carbon dioxid capacity in the

TABLE 4.—RELATION OF CARBON DIOXID CAPACITY TO RESPIRATION

| Number of Cases | Carbon Dioxid Capacity | Average Respiratory Rate |
|---|---|---|
| 17 | 40 to 49 (av. 44) | 24 |
| 7 | 30 to 39 (av. 35) | 28 |
| 6 | 20 to 39 (av. 24) | 44 |

region of 30 per cent or lower. In some instances the breathing was deep and vigorous, as in true "air hunger," and at the rate of 40 or 50 times per minute. These conditions have been met especially in cases of infection with the gas bacillus.

### The Sugar Content of the Blood

Lack of food, or subsistence on a carbohydrate-free diet, as is well known, will produce a condition of acidosis marked by lowered carbon dioxid tension of the alveolar air, increased excretion of ammonia nitrogen in the urine, and the appearance of acetone bodies. The low blood pressure of shock with attendant slight urinary secretion, and the generally depressed state of the patient, render accurate studies of the urine difficult. The possibility of a "starvation" acidosis being present, however, is suggested by the fact that not infrequently men are brought to the casualty clearing station about noon who have been wounded and

shocked in a night raid, and who testify to having eaten nothing since the previous afternoon. On admission to the station, they are often too ill to take nourishment. In consequence they may be without food for a time which might be expected to produce metabolic disturbance. A prime condition for "starvation" acidosis is lack of sufficient carbohydrate in the body to play a necessary rôle in the oxidation of fat, in this instance body fat which is being used as a source of energy. Determinations of blood sugar will show, therefore, whether or not a deficiency of carbohydrate prevails. In Table 5 are presented the results of observations in cases of shock and hemorrhage.

From these observations it is clear, in the first place, that there is no lack of sugar in the blood; indeed, that

TABLE 5.—SUGAR CONTENT OF BLOOD IN CASES OF SHOCK AND HEMORRHAGE

| Initials | Carbon Dioxid Capacity | Blood Sugar | Initials | Carbon Dioxid Capacity | Blood Sugar |
|---|---|---|---|---|---|
| F. W. | 34 | 0.11 | A. H. P. | 47 | 0.10 |
| W. B. | 36 | 0.15 | W. G. | 47 | 0.18 |
| O. C. R. | 40 | 0.15 (6 hours after hit) | O. C. R. | 50 | 0.12 (1 hour after hit) |
| J. B. | 42 | 0.19 | A. S. | 52 | 0.11 (after op'n) |
| H. H. | 42 | 0.22 | A. S. | 60 | 0.12 (before op'n) |

the amount is commonly above normal—0.1 per cent. Furthermore, there appears to be no relation between variations of the carbon dioxid capacity of the blood and the percentage of sugar. In a few cases urine was obtained from shock cases and tested for acetone bodies. The test for diacetic acid was negative. The acidosis of shock cases, according to this evidence, is due to some other alteration of the blood than the development of acetone bodies.

### The Effect of Anesthesia and Operation on Existent Acidosis and Low Blood Pressure

That anesthesia and operation are accompanied by a reduction in the alkali reserve of the blood has been shown by Crile and Menten,[11] Austin and Jonas,[12]

11. Crile and Menten: Ann. Surg., 1915, **62**, 262.
12. Austin and Jonas: Am. Jour. Med. Sc., 1917, **153**, 90.

Morriss,[13] and Caldwell and Cleveland.[10] The largest drop during operation recorded by Austin and Jonas was 10 volumes per cent. carbon dioxid capacity, as determined with the Van Slyke apparatus. The figures reported by Caldwell and Cleveland, based on a study of a large series of cases in which they used the Van Slyke method, show a drop in the carbon dioxid capacity between 4.7 and 7.7 volumes per cent. in operations averaging about fifty minutes in duration. In their series, however, no acidosis was present (except in one case), and the drop barely brought the capacity to the boundary line between normality and

TABLE 6.—TESTS AFTER OPERATION IN CASES WITH ACIDOSIS AND LOW BLOOD PRESSURE

| Initials | Duration of Operation | Carbon Dioxid Capacity | | Blood Pressure | |
|---|---|---|---|---|---|
| | | Before Operation | After Operation | Before Operation | After Operation |
| | Mins. | % | % | | |
| S. R. | 75 | 58 | 46 | 82–58 | 58–36 |
| B. S. | 40 | 58 | 46 | 88–62 | 74–42 |
| F. A. T. | 60 | 56 | 40 | 98–80 | 64–48 |
| G. J. H. H. | 45 | 50 | 44 | 90–62 | 64–46 |
| W. C. | 20* | 49 | 40 | 76–58 | — |
| J. H. C. | 45 | 47 | 40 | 102–80 | 50–28 |
| W. A. T. | 60* | 46 | 38 | 76–36 | 68–28 |
| G. K. | 50* | 46 | 27 | 84–60 | 58–36 |
| C. P. H. | 70 | 41 | 28 | 76–46 | — |
| Averages | 52 | 50 | 38 | 88–62 (7 cases) | 62–31 |

* Anesthetized with nitrous oxid and oxygen.

acidosis (50 per cent.) It is well known in civil surgical practice that patients with a low alkali reserve (as diabetics with acidosis, and children whose reserve is naturally much lower than that of adults) stand operation poorly, and may pass from anesthesia into coma and die. A highly interesting and practical question, therefore, is raised as to the influence of anesthesia and operation on wounded men with an acidosis already existent. In Table 6 are shown the results of blood tests and blood pressure readings before and after operation in cases with a carbon dioxid capacity below 50 per cent. at the start or found below that level at the end of anesthesia.

13. Morriss, W. H.: The Prophylaxis of Anesthesia Acidosis, THE JOURNAL A. M. A., May 12, 1917, p. 1391.

Comparison of the changes in the blood of these wounded men suffering from shock and unstable circulation with those reported by Caldwell and Cleveland reveals that the average drop in carbon dioxid capacity is approximately twice that seen by them in ordinary civil cases. Furthermore, if the three cases are eliminated which at the start had a carbon dioxid capacity well over 50 per cent., the results show that the fall is likely to be greater the lower the original capacity. The encroachment on the reserve of alkali is greater, therefore, as the margin of safety in the reserve is less. In other words, the more marked the existent acidosis the more sensitive is the patient to operative procedures, and the more likely he is to be let down by them into a region of danger. In ten of the series of cases under consideration the carbon dioxid capacity before operation was 40 *per cent. or less.* The closeness of danger in such cases may be realized by the fact that blood taken from the heart at the moment of death from shock has a capacity between 20 and 24 per cent., and in two of the cases reported in Table 6 the capacity fell to 27 and 28 per cent. from an initial 46 and 41 per cent., respectively. Such profound changes may occur in about an hour. The suddenness of this remarkable fall in available alkali is in itself important, for as Caldwell and Cleveland have pointed out, the effects are more damaging when the fall is rapid than when it is slow and gradual. With regard for the baneful effect on internal respiration and on other processes of the body due to an impoverishment of the blood in alkali, it is clear that a rational treatment of shock should include provision against the dangers of this sudden depletion.

Table 6 brings out another important fact of practical importance—the striking fall of blood pressure as a result of operation in these cases. In experience with patients in whom the decrease of the carbon dioxid capacity, as a result of operation, did not extend below 50 per cent. (that is, did not develop an acidosis, in the Van Slyke sense), no noteworthy alteration of arterial pressure occurred. In fact, both the systolic and the diastolic pressure may be higher at the end of operation than at the start, and seldom is there a lowering of the mean pressure. On the other hand, if the carbon dioxid capacity falls below

50 per cent., or being below that level it sinks still lower as a consequence of operation, the blood pressure may suffer an astonishing decline. In the seven complete records in Table 6, the average fall was 88 and 62 to 62 and 36—the systolic pressure at the end of operation was commonly below the diastolic pressure at the start. This ominous sinking of the blood pressure has been repeatedly seen during operation in shock cases in which the alkali reserve was not determined; in all probability these cases should be classified with those in the foregoing list.

Marshall[14] has testified that anesthesia with nitrous oxid and oxygen is specially to be recommended in operating on men in shock, because it leaves the patient in much better condition than do other anesthetics. Crile and Lower[15] also have stated that nitrous oxid-oxygen anesthesia is less likely to increase shock than ether. In two patients in the present series, blood examination before and after operation with nitrous-oxid-oxygen as the anesthetic, there was no change whatever in the carbon oxid capacity; it remained at 58 per cent. in the one, and at 48 per cent. in the other during operations lasting forty and twenty-three minutes, respectively. That this anesthetic does not preclude a fall of blood pressure and an attendant alkali reduction is shown by the three cases in Table 6 distinguished by asterisks—patients anesthetized with nitrous oxid and oxygen. The rate of change in the second of these three, however, was less than in any of the others, and the blood pressure was only slightly reduced. The charted results presented by Caldwell and Cleveland show not quite so great a decrease of the carbon dioxid capacity under "gas" and oxygen as under ether, but they conclude that the differences under different anesthetics are negligible. The possibility that the patient anesthetized with nitrous oxid and oxygen may suffer no appreciable blood change, and also that such changes as have been recorded may be due in part to lack of skill (as in allowing the patient to become cyanosed), gives support to the judgment that it is the anesthetic to be employed when possible in operating in cases of shock.

14. Marshall: Proc. Roy. Soc. Med., 1917, **10**, 27.
15. Crile and Lower: Anoci-Association, Philadelphia, 1914, p. 78.

## Alkaline Treatment of Extreme Acidosis in Shock

The danger zone that shocked men are liable to enter when they are operated on is approached, as already explained, through a precipitous fall of blood pressure and a sharp decrease of the alkali reserve to a degree at which the H-ion concentration of the blood tends to increase rapidly. The harmful effects on tissue respiration and on other bodily functions that occur when the H-ions are increased have already been emphasized. Wright[16] had reported a lessening of the alkalinity of the blood in human beings and in lower animals infected with the gas bacillus, a condition which he designated as "acidemia." The evidence presented above shows that this condition is not peculiar to toxic shock with gas infection,[17] but is general for states of low blood pressure, whether due to wounds or hemorrhage, without notable infection. The toxemia is merely an additive factor. The rational treatment of patients with diminished alkali in the blood, as has long been recognized in dealing with the acidosis of diabetes, is to supply alkali. Wright suggested this therapy also for treatment of gas infection, and reported good results in two instances. Its use in combating the extreme acidosis that may follow operation on men in shock is shown by the following cases:

Private J. F. J. was admitted, July 27, with multiple shell wounds of the leg, knee, right forearm, chest, and right hand. Considerable hemorrhage was probable. Before operation blood pressure was: systolic 80, diastolic 58, pulse 134, respiration 24, and not peculiar. The carbon dioxid capacity was 43 per cent. At operation the left knee was resected, about 6 inches of the right gastrocnemius removed, a long slit was made in the right forearm with excision of the flexor longus digitorum, and the ring and little fingers of the right hand, with their metacarpals, were taken away. Evidence of gas infection was found.

Shortly after recovery from the anesthesia, the blood pressure was 68 and 40, pulse 148, and respiration 34, deep and

16. Wright: Lancet, London, 1917, **1**, 1.

17. The importance of a good circulation for checking the spread of gas infection has been remarked by a number of writers on the subject. Attention has been directed mainly, however, to local tensions in the tissues and to thrombosis as the occasions for a poor blood supply. That a low general blood pressure may lie behind the local impairment should be more generally recognized.

vigorous. An hour later, at 10:30 p. m., the pulse was still as before, but the respiration had risen to 48, and was as energetic as if there had been violent struggle. In spite of his wounds, the patient tried to sit up, crying, "I must have air. I can't breathe." A cannula was slipped into an elbow vein, and 35 ounces of warm 4 per cent. sodium bicarbonate were introduced. A most dramatic change at once occurred. The patient's restlessness and "air hunger" immediately disappeared, and in a few minutes he fell asleep with a pulse of 126, and quiet respirations 26 per minute. A second respiratory crisis during the night required another injection of the sodium bicarbonate solution. Alkaline treatment was continued by mouth—1 dram in 8 ounces of sweetened water every two hours. On the following morning the patient was found smoking a cigaret. In the afternoon the blood pressure was 114 and 56, pulse 132, respiration 28. The second day the blood pressure was 128 and 58, pulse 144, respiration 38, but gas infection was found in the calves of both legs. In the subsequent operation considerable blood was lost. The patient sank rapidly thereafter, and died in about three hours.

Private H. (not in tabulation) was admitted, July 27, with multiple shell wounds. There was a big opening in the left loin with fracture of the pelvis and infection of the muscles; extensive lacerations of the left thigh, the left calf, and the right calf, with gas infection; many superficial wounds of back and chest and left hand. He was operated on at 11 p. m., but records of his blood pressure were not taken. About 6 o'clock the next morning his pulse was 150, and he was suffering from typical "air hunger" with deep respirations approximately 60 a minute. He was given intravenously 2 pints of warm 4 per cent. sodium bicarbonate. He became quiet at once, and fell asleep. Alkaline treatment was continued by mouth. At 12:30 p. m. the blood pressure was 72 and 56, pulse 140, and respiration 32. The carbon dioxid capacity was 51 per cent. In the afternoon the blood pressure was 104 and 68, pulse 132, respiration 27. The condition was the same next morning, except that the blood pressure had risen to 114 and 72. The patient then began to have an oscillating fever temperature; a foot wound was found infected, and the pathologist reported a coccus in the blood. A blood destruction started, and at the end of a week the red count fell to 1,800,000, hemoglobin 30 per cent. Transfusion was done, with consequent improvement. The patient was evacuated, but died of pneumonia in a hospital at the base.

"Air hunger" in such cases is typically a signal of impending death. The results described in the foregoing records demonstrate that the desperate state of the patient suffering from the marked acidosis of traumatic and toxic shock may be so changed by intravenous alkaline therapy that with astonishing suddenness he passes from distress to comfort, and may

later recover normal blood pressure. These cases also indicate, however, that the individual who has passed through such a crisis appears to have little resistance and is exposed to dangers of subsequent infection.

The development of acidosis in shock and its serious aggravation by surgical procedures suggested the desirability of using, if possible, preventive measures—increasing the chances of survival for wounded men by avoidance of critical risks. To throw light on these possibilities, the natural history of the shocked individual should be known. For the purpose of securing this knowledge, Captain Cowell went to the front trenches to study cases which were later traced through the aid post and the dressing station to the casualty clearing station. The results of this study are given in the following paper.

SUMMARY

Cases of low blood pressure due to shock, hemorrhage, or infection with the gas bacillus have a diminished supply of available alkali in the blood, that is, an acidosis. As a general rule, the lower the pressure the more marked the acidosis.

The pulse is rapid in these cases, but does not vary with the degree of acidosis.

The respiratory rate becomes more rapid as the acidosis increases until, shortly before death, a true "air hunger" may prevail.

Blood sugar is usually somewhat increased above the normal in cases of shock and hemorrhage. The acidosis in these cases, therefore, is not due to lack of circulating carbohydrate.

Operation on men suffering from shock and acidosis results in serious and rapid sinking of arterial pressure when it is already low, and in marked and sudden decrease of the alkali reserve of the blood when that reserve likewise is already low. This change may not occur if nitrous oxid-oxygen anesthesia, instead of ether, is employed, but that anesthetic affords no guarantee against the ominous decline.

Shocked men suffering after operation from extreme acidosis with "air hunger" can be quickly relieved of their distress by intravenous injection of a solution of sodium bicarbonate, and their blood pressure restored to normal.

# THE INITIATION OF WOUND SHOCK

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## INTRODUCTION

The name "wound shock" is suggested in order to avoid the confusion which arises, even among medical officers, if the word "shock" alone is used. The term "surgical shock" used in connection with the reaction of the body to wound injury is also to be deprecated.

Wound shock may supervene early on. That is, the man suddenly becomes pale, clammy and pulseless; and a low pressure may be found as soon as it is possible to make a reading, fifteen to twenty minutes after the man has been hit. To this group of symptoms with hypotension the name *primary wound shock* is given. When, on the other hand, as the result of a long carry in the cold or the onset of toxemia, or the presence of continuous slight hemorrhage, or combinations of these conditions, a man previously in good condition develops similar shock symptoms, a condition of *secondary wound shock* may be said to exist.

In order to understand more completely the later phases of fully developed wound shock, the necessity for making clinical studies of the earliest manifestations of the condition became more and more emphasized. Opportunities arose and facilities were granted by the army medical authorities for carrying out the requisite investigations. A series of posts were established along the route of evacuation from the firing line to the casualty clearing station. Suitable cases were chosen and examined, blood pressure readings taken, and notes made by medical officers interested in the work. A spring sphygmomanometer was used at each post. In a few instances the same observer followed the case for several miles, in order to watch the patient and check observations.

It was thus possible to collect a small series of cases and establish the exact time relations in the pressure curves of both primary and secondary wound shock.

Besides a study of the blood pressure, careful clinical notes were kept of all cases. In addition, a few hematocrit readings were made in the line and samples of venous blood taken for examination in the laboratory for acidosis.

A dynamometer was improvised from the spring blood pressure instrument in order to form an idea of the general muscular power and "tone" of the nervous system in these cases of hypotension.

Besides observations in actual wound cases, many pressure readings of officers and men in different parts of the line and under varying conditions were taken The facts observed during a tour of duty in the line in February, 1917, were confirmed and amplified.

### Physiology of the Fighting Soldier

A good deal of light is shed on the pathogenesis of wound shock by a consideration of the conditions of life of the soldier in the zone of fire. In the case of men living in the areas behind the trenches, where they are exposed to occasional shell fire, the conditions of food, drink, sleep, and shelter from the weather are practically normal. One finds an average systolic pressure of from 110 to 120 mm., with a diastolic of from 75 to 80 mm. and a pulse pressure of from 35 to 40 mm.

In the front line trenches, however, the conditions of existence are for the most part unfavorable. The soldier is subjected to long spells of hard physical labor, often accompanied by profuse sweating. Sleep is short, and generally interrupted. The food supply is sufficient, but water is very often available only as a limited ration. In the firing line and during battle all these factors become accentuated.

For a large part of the year, exposure to wet and cold must be taken into account. Thus the man, who may be hit at any time, is likely to be in a state of fatigue, with a tendency to concentration of plasma, sluggish peripheral circulation, and accumulation of waste products of muscular metabolism.

The urine of such men is dark, scanty, and loaded with phosphates.

Evidence has already been obtained which shows that in conditions of this kind at an exposed active

sector of the line the average systolic pressure was a little above the normal, that is, from 120 to 130 mm. Pressures were found without exception to be raised at times of increased activity, with the following average results: systolic pressure, from 140 to 160 mm.; diastolic pressure, from 70 to 100 mm.; pulse pressure, from 60 to 70 mm. Communication of the details of these observations must at present be deferred.

It is not possible to make similar observations in the heat of battle, but there is no doubt that these physiologic conditions are all present, only in a much greater degree, affording important prewound factors in the initiation of wound shock.

## Classification of Wounds with Reference to the Incidence of Wound Shock.

From the point of view of the production of shock, the wounded may be divided into the three following classes:

A. Trivial wounds, which cause only slight damage to the tissues.

B. Moderately severe wounds, in which the anatomic disturbance disables the man, but is not sufficient immediately to endanger life.

C. Serious wounds which, from the nature of the injury, must of necessity prove mortal, or which will cause death unless the man is given surgical attention within a short time.

### CLASS A. TRIVIAL WOUNDS

In these a transient psychic disturbance may occur, and the man become pale and sweat. But in three cases of fainting after being wounded, the pressure, which was taken as soon as possible after the recovery from the faint, was found to be normal. In men who are more "highly strung" an excitement stage may follow a trivial wound. For example, a boy 18 years of age, with very slight multiple superficial shell wounds, exhibited a systolic pressure of 180 mm. In this group of cases the question of temperament plays a prominent part. Examination was made of a party of slightly wounded men, belonging to a county infantry battalion, as they came back into the firing trench

from a raid. The average systolic pressure was 110 mm.; only one man had a reading as high as 130 mm.; he was talkative and excited.

Apropos of this question, the experimental work of Cannon[1] and his co-workers on the physiology of excitement is very interesting and eminently applicable to the soldier in battle. According to this author, a definite series of psychologic events results from the application of suitable psychic stimuli. The main factor is the outpouring of suprarenal secretion, which helps to transform the animal into a fighting mechanism. The circulation is better maintained by the raised blood pressure; the heart and skeletal muscles can do more work with their increased supply of blood sugar, and in the event of a wound being received the coagulation time of the blood is so diminished that clotting occurs more quickly and the bleeding is staunched.

In the excitement cases the pressure subsides rapidly with quiet and rest. In the case of the boy of 18, it had fallen to 160 and 90 in thirty minutes and 126 and 80 in the next hour.

### CLASS B. MODERATELY SEVERE WOUNDS

In this group are included those wounds which cause a certain degree of damage to the tissues, but either a vital organ is not involved at all, or, if such is involved, the damage is slight, as in a small perforating wound of the abdomen, and danger to life does not develop for some hours. In these cases primary wound shock does not as a rule occur. If the patient is examined within a few minutes where he fell, he presents no sign of general systemic disturbance, and the blood pressure remains within normal limits. After the lapse of a short time, however, and as a result of the operation of certain factors which are to a great extent avoidable, the symptoms of shock with hypotension appear, and the patient then shows secondary wound shock (Chart 1).

For example, a man (*a*) was hit by a shrapnel ball which fractured his femur. He was passing along the trench just outside the aid post at the time, and was treated by the medical officer at once. His pulse was 72 and pressure 120 and 80. Again, by a curious

1. Cannon: Bodily Changes in Pain, Hunger, Fear and Rage, New York, 1915.

chance, a shell hit a house in which was a party of men (*b*), four of whom sustained compound fractures of the femur, without developing immediate shock. Another case (*c*) was observed in an outpost in the front line, when a bomb arrived which killed one man on the spot and wounded his chum. The foot of this man was partially shattered and his shoulders and neck sprinkled by little fragments. His blood pressure,

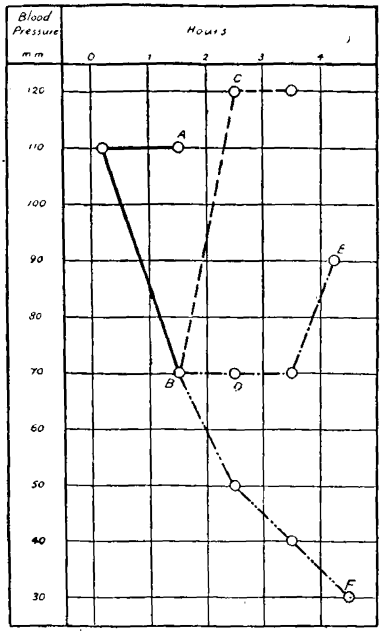


Chart 1.—Secondary shock curves, diagrammatic: In many wound cases the pressure will remain level (*A*). In others, as a result of hemorrhage or exposure to cold, there is a drop of pressure with the establishment of a secondary shock (*B*). If the patient is at this stage well cared for and the wound not too severe, the pressure will rise during the next stage of the journey (*C*) or remain stationary (*D*), improving after admission to the casualty clearing station (*E*). In the absence of favorable circumstances, the pressure goes steadily down and the case terminates fatally, usually in from twelve to twenty-four hours (*F*).

which, as it happened, had been found to be 110 and 70 a short time before, remained unaltered.

One other example (*d*) of this class of case may be quoted, which possibly is exceptional, but which is nevertheless of interest. A strong, burly lance-cor-

poral, a boxer of good reputation, was hit by a shell which carried away his left leg halfway between the knee and the ankle and also shattered his right foot. When seen, soon after, his pulse was 96 and systolic pressure 115 mm. Clinically, one would say none of the usual symptoms of shock were present. As the pain was considerable, he was at once given a hypodermic injection of morphin, ⅓ grain, by the medical officer.

The immediate after history of these wounded men is instructive.

The man (*a*), with a compound fracture of the femur, who was hit outside the aid post, was warm at the time. He was put on a stretcher at once, well wrapped up, and sent down to the next post on the line of evacuation. On arrival here his pulse was 72 and pressure still normal. Since he was quite comfortable in his splint, he was kept several hours before being sent to the casualty clearing station, and developed no signs of shock.[2]

The four men (*b*), hit in the same house, were splinted at once and placed on a motor ambulance, which was driven at a walking pace to a casualty clearing station within half a mile. No shock developed, and all did well.

In the example of the man (*c*) hit by a bomb at the outpost, secondary wound shock developed. He arrived cold and pulseless at the aid post. Before he was hit he was cold, fatigued and thirsty; and during the carry of an hour and a half, exposed to the chill wind preceding dawn, he lost more heat. Fear, too, was probably superimposed as a factor in this case, since on the journey down the stretcher party was exposed to occasional shells and bursts of machine gun fire. A sound man feels moderately secure in the trenches because he can take cover; but a man who is already hit and is being carried along on a stretcher, shoulder high, feels helpless. At the next stage this

2. There is little doubt that since the universal introduction of the Thomas splint for fractures of the femur, which has proved increased comfort to the patient and immobilization of the fragments of the bone, the incidence of secondary shock has been greatly diminished. Such patients used to arrive at the casualty clearing station with well established shock showing systolic pressures of 80 or 90 mm. or less. Similar cases now arrive with systolic and diastolic pressures of 130 and 90, 120 and 80, or even 160 and 100 in one case.

man was still pulseless. Very soon after being admitted to the casualty clearing station, however, he was warmed; his pulse returned, and he did well without the exhibition of any antishock methods of treatment.

The boxer (*d*), with his leg blown off, also developed secondary wound shock. He did not manifest any lowering of tension on the journey from the trenches to the advanced dressing station, but on arrival at the casualty clearing station, about five hours after his being wounded, toxemia was beginning to develop, and the pressure had dropped from 114 and 70 to 88 and 62 mm.

Before leaving the second group of cases, another example of marked wound shock, but with quick recovery, may be mentioned. A young soldier (*e*) in a wiring party sustained two simple bullet wounds of the fleshy part of the thigh. It was a cold night. After being carried two hours, with no hemorrhage, he was found to be cold, thirsty and pale, with a pressure of 70 and 50 mm., and a small thready pulse of 68. He was given a hot drink and, after being warmed and well wrapped up, was sent off. At the next post his arterial pressure had risen to 120 and 80 mm., and remained at that level on arrival at the casualty clearing station.

CLASS C. SERIOUS WOUNDS

In the third group a serious surgical condition has resulted from the wounding. The wound shock comes on early, and apparently in proportion to the gravity of the lesion[3] (Chart 2).

In primary wound shock, very definite clinical symptoms may be observed. For example, the driver of an ambulance car was hit in the abdomen by a shell fragment as he arrived at the dressing station on a warm, sunny morning. He fell down, and within a few minutes, as his wound was being dressed, drew the attention of the medical officer to his profuse sweating. He was wrapped up in blankets, hot bottles were applied, and he was sent off. After thirty minutes' ride over a rough road, scarred by shell holes, he was found to be responsive to questions. He complained of pain, and presented cold, clammy extremities, with the sweating still marked. He was pale, with

3. The only predetermining factor of any importance in the amount of shock which may result is the temperamental stability of the patient. This point is illustrated in some of the following cases.

a pulse of 96 and a pressure of 100 and 70. On arrival at the casualty clearing station, about forty-five minutes later, the pressure had further dropped to 82 and 70 and the pulse had risen to 100. Operation was performed in time to stop further hemorrhage. Ten rents in the bowel were repaired, and after intravenous infusion he eventually made a good recovery.

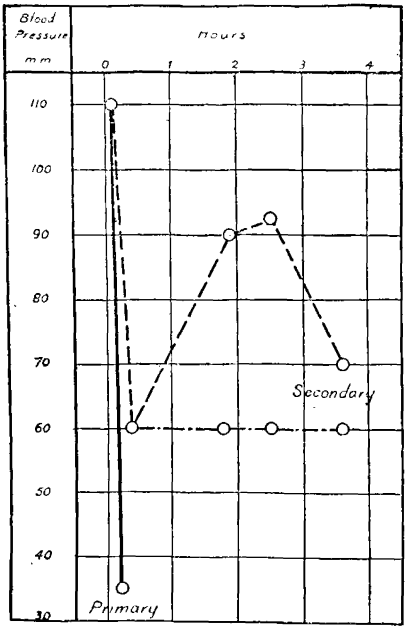


Chart 2.—Primary wound shock curves; diagrammatic: Following the receipt of a severe anatomic injury, where death must ensue or where life can be saved only by prompt surgical interference, instant shock appears accompanied by hypotension. This may be fatal in a short time. If all precautions are taken in the careful transit of the patient, the pressure may rise *en route* or remain level without further drop. After the lapse of a few hours the condition of primary wound shock merges into that of secondary wound shock unless recovery has first occurred.

A few other examples of severe injuries with early wound shock may be quoted, in which circulatory disturbance predominated. In these cases, whenever the systolic pressure was found below from 60 to 70 mm., it was always a distressing symptom.

Case 1.—A man on a working party was seen about forty minutes after being hit by a shell burst. He had sustained penetrating wounds of the chest, buttocks, legs and thigh,

with fracture of the femur. He was cold, with a clammy skin; pulse 80, and blood pressure 60 and 50 mm. He complained loudly of thirst and pain. His sensation was normal, and as the antitoxin needle was inserted he cried out. Respiration was from 20 to 24, with no unusual characteristics. Morphin, ¼ grain, hypodermically, stopped the pain in a few minutes; and after a few hours' warming he was sent on with a pulse of 90 and pressure of 98 and 70.

CASE 2.—A young officer, of a nervous, irritable disposition, was hit in the dorsal region of the spine by a sniper just after dawn. Two hours later his blood pressure was 75 and 50, pulse 84, and all the signs of wound shock were present.

CASE 3.—A large, powerfully built gunner was seen within half an hour after being hit, one hot, sunny morning. The shell fragments had fractured his lower jaw and left femur. He was cold, sweating, pulseless and thirsty. The systolic pressure was about 30 mm. There was considerable hemorrhage. He was treated in the routine way and sent down. He improved for a time at the casualty clearing station, but eventually died of gas gangrene.

CASE 4.—Another man, hit by a shell while on a working party on a cold, wet, muddy night, was brought to the advanced dressing station within fifty minutes. He was found to have multiple severe wounds, including compound fractures of both a femur and a humerus, with laceration of the muscles. When examined, the wounds looked like dead tissues. There was no bleeding, and practically no capillary oozing. His systolic pressure was 40 mm. Mentally he was quite bright and responsive, so that the medical officer in charge of the case remarked on the patient's "wonderful fitness." The man, however, was dead within the hour.

CASE 5.—Another man had a severe wound of the buttock, penetrating the abdomen. He was seen a short time before death, about two hours after he was hit. He was pulseless at the wrist, with a blood pressure of about 30 mm., and was so restless that he had to be held on the stretcher by two orderlies.

CASE 6.—A thin, lightly built sergeant of anxious, worrying disposition was hit as he lay asleep at the foot of his dugout, by a shell which shattered both his feet. His company commander, a medical man, was fetched within two minutes and found him unconscious and pulseless. Both feet were pulped, but not bleeding. He died about an hour later.

The "grip," as recorded by the improvised dynamometer in those cases of profound hypotension in which it was possible to make this test of muscular power, was in all cases practically the same as for a normal man.

## Conclusions

1. The psychology and physiology of the average healthy "veteran" soldier, living in the fighting zone under "peace" conditions are, for practical purposes, normal.

2. In battle and at points of activity, the conditions of excitement, cold, thirst, fatigue and possibly loss of sleep become important predisposing factors in the initiation of wound shock.

3. Wounds may be classified into three groups with regard to the incidence of shock.

*Trivial wounds,* such as slight scalp injuries or small lesions of subcutaneous tissues only, give rise to neither primary nor secondary wound shock. Transient psychic disturbances of either depression or excitement may result, accompanied by a normal or raised blood pressure.

In *moderately severe wounds,* such as uncomplicated compound fracture of the femur, a slight penetrating wound of the abdomen or a lacerating wound of muscle without urgent hemorrhage, as a rule, primary wound shock is absent, that is, if the patient is seen within a few minutes. If circumstances permit of sufficient care being taken, probably no untoward symptoms will arise, and secondary wound shock may be averted (Cases *a* and *b*).

As the factors of cold, toxemia, pain, anxiety or lack of water are allowed to come into play, however, there develops, after a lapse of several hours, secondary wound shock (Cases *c, d,* and *e*).

Into the category of *serious wounds* will fall a large group of cases in which the wound must of necessity prove mortal. Symptoms result immediately and hypotension is found from the earliest moments (Cases 1 to 6). This is primary wound shock. Such shock is likely to be produced to a greater degree in men possessing temperamental instability. After a few hours, unless recovery has first occurred, the condition of primary wound shock merges into that of secondary wound shock, owing to the operation of the same conditions, cold, pain, anxiety. In the slighter cases, recovery from primary symptoms is rapid (Case 1); but then exposure and the factors enumerated in B may come into play, and secondary wound shock become established.

# A CONSIDERATION OF THE NATURE OF WOUND SHOCK

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## INTRODUCTION

The previous papers of this series have consisted mainly of records of observed phenomena in cases of shock, with little discussion of their origin or significance. These observations, however, have suggestive values and are pertinent to theoretical aspects of the shock problem. A consideration of the bearings of our results on previous views of the nature of shock may bring out some new points and may be useful in directing further investigation into fruitful channels.

## THE BEARING OF PRESENT WORK ON PREVIOUS THEORIES OF SHOCK

### A. THE ACAPNIA THEORY

The thesis advanced by Yandell Henderson that shock is due to a reduction of the carbon dioxid of the blood (acapnia) has received much attention. In support of his view, Henderson[1] produced low blood pressure in animals by vigorous artificial respiration, and he assumed that the lowered carbon dioxid content of the blood thus induced was the prime factor in establishing the shocklike state. In all probability, however, the effect was due, not to reduction of carbon dioxid, but to mechanical obstruction to return of blood to the heart and to consequent failure of the circulation. Only by such extreme inflation of the lungs as would hinder the passage of venous blood through the veins of the chest were Janeway and Ewing[2] able to obtain the results described by Henderson, and they succeeded equally well when the normal carbon dioxid content of the blood was maintained.

Further doubt is thrown on Henderson's views by observations on the character of the breathing in wounded men suffering pain. Deep and vigorous ventilation of the lungs is required to produce a marked

1. Henderson, Yandell: Am. Jour. Physiol., a series of papers, 1908-1910.
2. Janeway and Ewing: Ann. Surg., 1914, **59**, 158.

diminution of the carbon dioxid content of the blood. Cowell made a special point of looking for that type of respiration in recently wounded men, but did not see it. And in a man brought under my observation at the clearing station a few moments after he had been hit in the wrist by a bomb splinter, which was giving him much pain, explosive respirations, preceded by holding the breath, were going on at the rate of 12 per minute. From the evidence in hand it appears that painful wounds are not directly associated with a hyperpnea that would produce acapnia.

Recently Henderson, Prince and Haggard[3] have reported finding in animals experimentally shocked a condition of acidosis; and since the blood in this state has a reduced capacity for carbon dioxid, he has given the "acapnia" theory a new interpretation—the carbon dioxid is low because of the acidosis. The observations previously reported[4] confirm in human cases the evidence secured experimentally. Shock is accompanied by acidosis, and the ability of the blood to take up carbon dioxid is correspondingly reduced.

Henderson has raised the question, however, as to whether the alkali of the blood controls the carbon dioxid content, or the carbon dioxid content controls the alkali. Apparently reluctant to abandon his acapnia theory, he suggests that in shock, excessive breathing may greatly lower the carbon dioxid of the blood, and that as a protective compensation acidosis is developed to prevent the fatal apnea that might ensue from lack of stimulating H-ions. Again it is proper to emphasize the observation that such hyperpnea as would result in marked reduction of the circulating carbon dioxid is absent from wounded men. To be sure, the chlorin content of the plasma increases[5] as carbon dioxid leaves the blood, but as any one may easily demonstrate to himself, this is not a process of sufficient magnitude to prevent apnea after vigorous hyperpnea. Furthermore, in shock, acidosis, in the sense of a lessened alkali reserve, occurs before any noteworthy effect on respiration is manifest. These considerations render

3. Henderson, Yandell; Prince, A. L., and Haggard, H. W.: Observations on Surgical Shock, THE JOURNAL A. M. A., Sept. 22, 1917, p. 965.

4. Cannon, W. B.: Acidosis in Cases of Shock, Hemorrhage and Gas Infection, p. 47.

5. Hamburger: Osmostischer Druck und Ionenlehre, Wiesbaden, 1902., I, p. 264. See also Austin and Jonas: Am. Jour. Med. Sc., 1917, cliii, p. 86.

highly questionable Henderson's suggestion of a primary acapnia.

Henderson[3] and also Porter[6] have advocated rebreathing expired air as a means of improving the circulation in shock. Henderson's object is to maintain the carbon dioxid content of the blood in spite of an alkali deficit, with the idea that alkali may thus be mobilized and the circulatory apparatus benefited. Porter's object is to increase the amplitude of the movements of the diaphragm so that the sufferer who has bled "into his own abdominal veins" may pump the blood from this reservoir into the heart. The certain effect of the increase of the carbon dioxid in the blood is to increase the H-ion concentration. That this stimulates not only the respiratory center but also the vasomotor center was shown by Mathison.[7] The ability of the vasomotor center to respond to stimulation, even in profound shock, was proved by Porter[8] in 1908. That the increased arterial pressure obtainable by increasing the H-ions of the blood would be only temporary was to be expected. As one of Porter's cases shows, the raised pressure promptly fell to the former shock level as soon as the carbon dioxid administration stopped. The respiratory mechanism, of course, at once gets rid of the excess of carbon dioxid, the H-ion concentration falls, and the stimulus which raised the pressure is thereby reduced. There are possibilities of harm in this procedure, however, that should not be overlooked. As Milroy[9] has recently demonstrated, when the alkali reserve is reduced, exposure of the plasma to a given concentration of carbon dioxid increases the H-ion content to a much greater degree than is the case when the plasma is normal. Increase of the H-ion concentration interferes with cellular oxidation. If the patient rebreathes his expired air, the oxidative processes are further interfered with through diminished oxygen supply. Thus, nonvolatile acids may arise which fix the alkali and lead to a more permanent increase of the H-ion concentration of the blood. The state of acidosis which already exists in the shocked man may, therefore, be distinctly aggravated by the procedures advocated by Henderson and by Porter.

6. Porter: Boston Med. and Surg. Jour., 1917, **176**, 699; **177**, 326
7. Mathison: Jour. Physiol., 1910, **41**, 430.
8. Porter: Am. Jour. Physiol., 1908, **20**, 404.
9. Milroy: Jour. Physiol., 1917, **51**, 279.

In this connection the testimony of Marshall, who as an expert anesthetist in a casualty clearing station has had large experience, is pertinent. He has declared that the most important consideration in anesthetizing patients suffering from hemorrhage or shock is to avoid anything in the nature of asphyxia; indeed, that if such a patient becomes cyanosed, he loses ground that can hardly be recovered. This warning is in accord with the contention that any action increasing acidosis is to be avoided.

### B. THE IDEA OF SUPRARENAL EXHAUSTION

Since removal of the suprarenal glands results in lowered arterial pressure, and since secretion or injection of the extract of the suprarenal medulla increases the pressure, the idea has been advanced that in shock there is suprarenal exhaustion and consequent hypotension.[10] If the distinction which should be drawn between the effects on blood pressure of the medullary and cortical portions of these glands is for the moment disregarded, it may be pointed out that, according to Short,[11] who used a very delicate test, the epinephrin content of the glands in fatal cases of shock is not notably reduced. Furthermore, Mann[12] has reported that total excision of the suprarenals does not reproduce the phenomena of shock.

There is experimental testimony that painful stimuli and asphyxia increase both the secretion of epinephrin and the percentage of sugar in the blood, and that the sugar percentage does not rise if the suprarenal glands are not cooperative.[13] Persons suffering from wound shock have been severely stimulated, and the low blood pressure which characterizes their condition produces a state which is equivalent to partial asphyxia. Bedford and Jackson[14] and later Bedford reported finding the epinephrin content of the blood increased if the blood pressure is low. The high percentage of sugar found in our series of shock cases likewise indicates

10. Corbett, J. F.: The Suprarenal Gland in Shock, THE JOURNAL A. M. A., July 31, 1915, p. 380.
11. Short: Lancet, London, 1914, **1**, 131.
12. Mann, F. C.: Shock During General Anesthesia, THE JOURNAL A. M. A., Aug. 4, 1917, p. 371.
13. Cannon, W. B.: Bodily Changes in Pain, Hunger, Fear and Rage, New York, 1915, pp. 69-77.
14. Bedford and Jackson: Proc. Soc. Exper. Biol. and Med., 1916, **13**, 85-87. Bedford: Am. Jour. Physiol., 1917, **43**, 235.

that the suprarenal glands are, if anything, overactive rather than exhausted.

### C. THE NERVE EXHAUSTION THEORY

When observers noted that arterial tension is low in shock, the first suggestion offered was that relaxation of the arterioles had occurred, and in consequence there was no support for the head of pressure which the heart might otherwise develop. This view, long ago expressed by Mitchell, Keen and Morehouse, has been elaborated by Crile[15] in extensive investigations on the blood pressure and on the nerve cells in shocked animals. It is his belief that the most vital effect of shock is "the impairment of the vasomotor mechanism." The concept has been gradually developed that shock consists essentially of exhaustion of cells in the brain, the liver, and the suprarenal glands. Evidence that the suprarenals are not exhausted has been presented above. The theory of a primary exhaustion of nerve cells requires examination. It may be considered, first with regard to the vasomotor center, and then with regard to cerebral and motor functionings.

A lowering of arterial pressure is not proof that the vasomotor center is inactive or exhausted, for arterial pressure may be low in consequence of hemorrhage, that is, when only a small volume of blood is delivered to the heart for each contraction. Furthermore, even when an animal is in extreme shock, Porter[16] and his collaborators found that both pressor and depressor reflexes still occur. The occurrence of depressor effects proves that some tonic activity of the vasomotor center is still present, for otherwise its action could not be depressed; and the pressor responses show that the center is still capable of increased action when stimulated. These observations by Porter have been confirmed by Seelig and Lyon[17] and by Mann.[18]

Since the vasomotor center is not exhausted, the question arises as to its actual condition in shock. Recent experimental evidence points to its effective control of peripheral and visceral arterioles in the

15. Crile: Volumes on Surgical Shock; Blood Pressure in Surgery; Anoci-Association.
16. Porter: Am. Jour. Physiol., 1907, **20**, 399.
17. Seelig and Lyon: Surg., Gynec. and Obst., 1910, **11**, 146.
18. Mann: Bull. Johns Hopkins Hosp., 1914, p. 208.

shocked state. Seelig and Lyon[19] found that cutting the nerve of a leg in a shocked animal caused an increased flow of blood from the femoral vein. Guthrie, in confirming this work, observed that whereas the increase of flow in a normal animal was 22 per cent., in a shocked animal it was 76 per cent. Later, Seelig and Joseph[20] noted that if in a shocked rabbit the blood pressure was suddenly raised by clamping the aorta, the blood greatly distended the arteries of one ear whose nerves had previously been cut, but failed to distend the arteries of the other ear whose nerves were still connected with the vasomotor center. In other words, the center was still holding the vessels in effective contraction. Similar observations have been made by Mann[21] on internal organs. And a number of investigators have reported that in an animal with low blood pressure the rate of perfusion flow is less with a given pressure than in a normal animal, and that severance of the nerves to the organ or increase of the blood flow to the vasomotor center increases the rate.[22]

Moreover, the studies of Pike, Guthrie and Stewart[23] have revealed the fact that the vasomotor center is more capable of withstanding the adverse influences of anemia than any other of the vital bulbar centers—the respiratory, the cardio-inhibitory or the swallowing mechanisms. Its capacity to function is the last to disappear in total anemia, and the first to reappear when the blood flow is restored. Obviously, the vasomotor center should be regarded as an agent whose functions are extremely stable and whose capabilities for continued service are its most outstanding feature. Only endangering circumstances, such as lessened blood supply, are required in order to make it become, for a time at least, more than usually active.

In this connection it is interesting to observe that not infrequently in cases of profound shock, when a pulse cannot be felt at the wrist, it can be felt easily if the palpating fingers are moved up the arm where the arteries are larger, or applied over the carotid. Fraser

19. Seelig, M. G., and Lyon, E. P.: The Condition of the Peripheral Blood Vessels in Shock, THE JOURNAL A. M. A., Jan. 2, 1909, p. 45.
20. Seelig and Joseph: Proc. Soc. Exper. Biol. and Med., 1914, **12**, 49.
21. Mann: Bull. Johns Hopkins Hosp., 1914, p. 209.
22. Morrison and Hooker: Am. Jour. Physiol., 1915, **37**, 93. Pilcher and Sollmann: Ibid., 1914, **35**, 59, 70. Bayliss: Proc. Roy. Soc., London, 1916, **89**, 391.
23. Pike, Guthrie and Stewart: Jour. Exper. Med., 1908, **10**, 499.

has repeatedly noted while operating on shocked men such strong contraction of outlying arteries that no bleeding occurred when the vessels were cut.

The evidence from functional disturbance that parts of the nervous system other than the centers controlling the circulation are *exhausted,* in any strict sense of that term, is meager. Even when the blood pressure has been much reduced, the intelligence remains clear, the patient may be restless rather than somnolent, and often exhibits surprising muscular power. Cowell reports no reduction of the strength of grip in shock; and he observed a wounded man, pulseless, with a systolic pressure about 30 mm., who was so vigorous in his movements that two orderlies were required to hold him on the stretcher.

The evidence for exhaustion which has been advanced by Crile[24] and his co-workers is mainly histologic, and is based on examination of nerve cells taken from shocked animals. It is improbable that these changes are due directly to afferent impulses, for Forbes and Miller,[25] by use of the string galvanometer as an indicator, found that anesthesia blocks the passage of impulses to the brain. The suggestion is reasonable that any cell alterations that may occur in shock are the resultant of the low blood pressure rather than its cause. Indeed, Dolley has admitted that hemorrhage produces the same alterations in the cells that are seen in shock. Crile's testimony that, if the blood pressure is kept up by transfusion into the shocked animal much more severe trauma is required to alter the cells than when shock takes its natural course, is further testimony to the same interpretation. It should not be forgotten, however, that histologic evidence regarding the state of nerve cells is subject to grave mischances both in technic and in interpretation. And other observers who have examined nerve cells from shocked animals declare that the changes are within the limits of normal variations.[26]

Differences in the appearance of nerve cells from separate parts of the nervous system might be regarded

24. Crile: Anoci-Association, Philadelphia, 1913, passim. Dolley: Jour. Med. Research, 1909, p. 95; 1910, p. 331.

25. Forbes and Miller: Am. Jour. Physiol., 1916, **40,** 503-513.

26. Allen: Proc. Soc. Exper. Biol. and Med., 1915, **12,** 76. Kocher, R. A.: The Effect of Activity on the Histologic Structure of Nerve Cells, THE JOURNAL A. M. A., July 22, 1916, p. 278.

as indicating a definitely directed agent, such as nerve impulses, rather than a general agent, such as low blood pressure, at work to induce changes. The differences are explicable, however, on the ground that nerve cells are differentially sensitive to anemia, and by exposure to an inadequate circulation they would be differentially affected.[27]

The interpretation of cell changes as the result rather than the cause of shock points to a clear distinction which should be drawn between early and late indications of asthenia in shocked men. Though the vasomotor centers may for a time be normally active or even hyperactive, and the neuromuscular mechanisms may be ready for service, continuance of low blood pressure and the development of an acidosis which interferes with internal respiration will surely have deleterious influences, and ultimately will destroy the hardiest and most resistant structures. It is when these adverse conditions have brought to the point of exhaustion organs which are of vital importance that death occurs.

### The Cardiac Factor

The foregoing discussion of the exhaustion theory has emphasized the evidence that the low pressure of a shocked man is not due to relaxation of the arteries through paralysis or inactivity of the vasomotor center. The other important factor in maintaining the arterial head of pressure is the heart. As has been often observed, the heart characteristically beats rapidly in shock and after hemorrhage.[4] The suggestion has been offered that the rapid beat is due to paralysis of the cardio-inhibitory center, but Mann[28] found that in the shocked animal the center is responsive to reflex stimulation and also to increase of intracranial tension. The nervous check on the heart, therefore, is not impaired. Indeed, the rapid cardiac beat with hypotension is precisely what is to be expected according to the reciprocal relation which commonly prevails between heart rate and arterial pressure.

That the heart muscle is not defective in shock has been shown experimentally. Raising the arterial pressure to a high level by epinephrin does not over-

27. For discussion see Cannon and Burket: Am. Jour. Physiol., 1913, **32**, 347.

28. Mann: Bull. Johns Hopkins Hosp., 1914, p. 210.

whelm the heart; when properly supplied with blood, it meets the situation and contracts with vigor. Low arterial pressure, however, if prolonged, may incapacitate the heart, for Markwald and Starling[29] have found that when the systolic pressure falls below 80 mm. of mercury, the cardiac contraction begins to weaken. And Patterson[30] has shown that when the H-ion concentration of the blood increases (by increased carbonic acid), the heart relaxes more and more and beats less energetically. The low blood pressure and the acidosis of shock, therefore, may in time impair the efficiency of the organ, though no primary defect be present.

## The Problem of the "Lost Blood" in Shock

If the vasomotor center is efficiently at work, and if the heart is capable of assuming any reasonable burden placed on it, why is there a low arterial pressure in shock? The answer to this question lies in the diminished volume of blood which is in active circulation. Henderson, especially, has laid stress on the necessity of a sufficient supply of blood being delivered to the heart, if arterial pressure is to be kept at its normal level. In the absence of this supply, as, for example, after hemorrhage, the arterial pressure falls to a low level and can be raised only by introducing blood or other viscous fluid into the vessels.

A further question now arises, one of the critical questions in the mystery of shock, "Where is the lost blood in the shocked individual?" There are no indications that it is in the heart or lungs; it must be, therefore, in systemic arteries or capillaries or veins.

### IN THE ARTERIES?

The absence of the lost blood from the arteries is sufficiently proved by the facts already discussed. With an efficient vasomotor center and a capable heart, an adequate amount of blood in the arteries would be accompanied by high arterial pressure. That the pressure is low, as already stated, signifies that the heart is not supplied with enough blood to fill the arterial system.

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29. Markwald and Starling: Jour. Physiol., 1913, **47**, 275.
30. Patterson: Proc. Roy. Soc., London, 1915, B, **88**, 394.

IN THE VEINS?

The view commonly held in the past has been that in shock, blood is stagnant in the large venous reservoirs of the chest and abdomen, and especially in the capacious splanchnic area. "In shock," it is said, "the sufferer bleeds into his own abdominal veins." It appears that this view is based largely on evidence from experiments which has been rather uncritically accepted. The most certain way to produce shock in a lower animal is by exposure and manipulation of the intestine. Under these circumstances the mesenteric veins stand out prominently, blood gathers in the intestinal walls, and becomes more concentrated there, and the structures that have been freely handled appear as if inflamed.[31] In other words, blood obviously stagnates in abdominal vessels. Such a condition is not seen in natural shock. According to Keith,[32] the venous cistern, formed by the big veins of the chest and abdomen, has a capacity of 400 or 550 c.c. Mann[33] has found that the amount of blood that can be obtained by bleeding and by emptying the heart of normal animals is 76 per cent., leaving 24 per cent. "in the tissues." When animals are shocked by exposure of the intestine the amount left in the tissue rises to 39 per cent., a difference of 15 per cent. If the blood mass of a man of 70 kg. is taken as 3,500 c.c., the amount thus "lost" would be 525 c.c. If this blood were in the veins of the abdomen, systemic or splanchnic, their capacity would have to be greatly enlarged, and their distention would be clearly visible.

Surgeons of extensive experience at casualty clearing stations in the present war, who have performed many hundreds of abdominal operations on patients in all degrees of wound shock, have testified that on opening the abdomen they have not found any primary splanchnic congestion.[34] The method employed to produce shock in lower animals, which has repeatedly called attention to the abdomen and its peculiar circulation, has given rise to misleading inferences as to what occurs in natural shock brought on by wounding other regions than the abdomen.

31. Morrison and Hooker: Am. Jour. Physiol., 1915, **27**, 93. Mann: Surg., Gynec. and Obst., 1915, **65**, 380.
32. Keith: Jour. Anat. and Physiol., 1908, **62**, 1.
33. Mann: Surg., Gynec. and Obst., 1915, **55**, 380.
34. Statement by Wallace, Fraser and Drummond: Lancet, London, 1917, **2**, 727.

If the lost blood were in the systemic veins, furthermore, it should be possible promptly to remedy the condition of a shocked individual by placing his body in a slanting head-down position, bandaging the limbs, and compressing the abdomen. Such measures have been thoroughly tried in treating shock, and though perhaps in some cases helpful, they do not give results which indicate that the blood which is out of circulation is stagnant in the large venous channels.

The fact should be remembered that veins are to a considerable extent subject to vasoconstrictor impulses; and if conditions are such as to continue the activity or to induce an overactivity of the vasoconstrictor center, the veins as well as the arteries might be contracted. Venomotor nerves have not been demonstrated for all parts of the body, however, and if there are veins free from nervous control, other influences causing relaxation might prevail. Only slight dilation, perhaps too little to be conspicuous, would be needed to increase considerably the venous capacity. But there are no observations that the veins are even slightly dilated in shock.

### IN THE CAPILLARIES?

If in wound shock the lost blood is not in the arteries and probably not to a great amount in the veins, it must be mainly stagnant in the capillaries. Observations reported in a previous paper[35] have shown that in shock a striking discrepancy exists between the corpuscular content of the capillaries and the veins. There is a concentration of the blood and a stagnation of the corpuscles in the capillaries which can be demonstrated in such widely separated parts as the ears, the fingers and the toes. The discrepancy is, to be sure, more marked in superficial areas than in deeper regions; but even in the latter a noteworthy difference is found.

The question immediately occurs, Is the capillary capacity sufficient to contain the lost blood in shock? Unfortunately, the data for estimating the capillary capacity are not definitely established, and it is impossible to state with any assurance what amount of blood these vessels may contain. Ranke inferred from determinations made on freshly killed rabbits that approxi-

35. Cannon, W. B.; Fraser, John, and Hooper, A. N.: Some Alterations in Distribution and Character of Blood in Shock and Hemorrhage, p. 32.

mately one fourth of the blood is in the heart, lungs and great blood vessels, one fourth in the liver, one fourth in the resting muscles, and one fourth in the remaining organs.[36] The large proportion of the blood, about 75 per cent., which is outside the heart, lungs and large veins and arteries, seems to indicate an abundant capacity in the small vessels lying within the tissues. On the other hand, an estimation of the capacity of the capillaries, based on the inverse ratio between the rate of flow and the cross-section, yields a rather small volume for capillary contents. The most favorable ratio of the rate of flow in the aorta and in the capillaries, stated by Tigerstedt,[37] is 2,000:1. The cross-section of the aorta of an adult man is about 4.4 sq.cm.; that of the total capillary bed, on this basis, would be (4.4× 2,000) 8,800 sq.cm. The average length of a capillary is given as 0.05 cm. The total capacity of the capillary system, therefore, would be only about 440 c.c. This calculation does not take into consideration, however, the fact that capillaries are not all full of blood. Heubner[38] observed after injecting sodium gold chlorid the sudden appearance of new capillaries in the frog's web, so that a coarse mesh was quickly changed to a fine one. And Worm-Müller[39] was convinced that the only way to account for the ability of the circulatory system to accommodate itself to injection of large amounts of blood was to assume a utilization of capillaries not ordinarily filled. He cited the difference of appearance of the intestine when at rest and when digesting, the phenomenon of blushing and the redness of the inflamed skin as illustrating the idea that the capillary net may contain much more blood than it usually contains. The *distensibility* of the capillaries also should be considered, for Roy and Brown[40] noted that chloroform could double the diameter of capillaries (thus quadrupling their capacity). Still another consideration which is pertinent to the conditions in shock is the *concentration* of the capillary blood, as shown in a previous paper,[35] which means a retention mainly of

36. Vierordt: Anatomische, Physiologische und Physikalische Daten und Tabellen, Jena, 1893.
37. Tigerstedt: Physiologie des Kreislaufes, Leipzig, 1893, p. 423.
38. Heubner: Arch. f. Exper. Path. u. Pharmakol., 1907, **56**, 375.
39. Worm-Müller: Ber. ü. d. Verhandl. a. k. Sachs. Ges. d. Wissensch., 1873, **25**, 650.
40. Roy and Brown: Jour. Physiol., 1879, **2**, 375.

corpuscles in the capillaries. All these facts appear to warrant the conclusion that the capillary capacity is sufficient to contain the lost blood in shock, and that the chances of its doing so are greater the more concentrated the lost blood becomes.

The observations previously reported[35] indicated that the capillary blood may be concentrated to such an extent that a cubic millimeter contains 8 million instead of 5 or 6 million corpuscles. An equal concentration does not occur in all parts of the body. The observations of Cohnstein and Zuntz,[41] however, suggest that when blood pressure is lowered (by cutting the spinal cord) a capillary stagnation occurs to such an extent as to pack the vessels closely, while the venous blood quickly falls in corpuscular content (a drop of about a million corpuscles per cubic millimeter in ten minutes). Lowering of blood pressure by spinal section will in itself, on this evidence, induce a stasis which can be clearly seen under the microscope, and is observable in both surface and internal capillaries.[42]

## The Viscosity Factor

There are other conditions besides a low blood pressure that are favorable to capillary stagnation of the corpuscles. These are concerned with alterations in viscosity. The viscosity of the blood is complex, consisting, as it does, of the internal friction of the plasma, the friction of the corpuscles with the plasma and with each other, and the frictional contacts of the corpuscles with the vessel walls, especially in the capillaries.

A prime factor affecting the viscosity of blood is the number of corpuscles per unit volume. The polycythemia of cholera, for example, may cause the viscosity of the blood to rise from 4.8 to over 20.[43] Cohnheim[44] attributed the low blood pressure in cholera to a failure of the blood to return to the heart, owing to the enormous increase of frictional resistance that is caused by concentration. The concentration of the corpuscles in the capillaries would, in itself, render the friction

41. Cohnstein and Zuntz: Arch. f. d. ges. Physiol., 1888, **42**, 326.
42. These observers did not consider possible dilution of the blood by tissue fluids, and they transferred the observations on capillary stagnation in the frog to explain the conditions they found in the rabbit. The importance of the results, in relation to the nature of shock, warrants a careful repetition of the experiments under more critical conditions.
43. Bence: Ztschr. f. klin. Med., 1906, **58**, 203.
44. Cohnheim: Lectures in Pathology, London, 1889, **1**, 466.

greater, and increase the resistance to an onward movement.

Another agent affecting the blood's viscosity to a notable degree is temperature. The increase of internal friction is related directly to a fall of temperature. Denning and Watson[45] found that viscosity of blood was increased 3 per cent. with a fall of 1 degree Centigrade, and that the temperature factor was more effective the larger the number of corpuscles present. Even in normal persons, application of cold increases the red count in the cooled capillary areas. Cowell's observations on recently wounded men have shown that a prompt and striking reaction to the injury is profuse sweating.[46] One of the most effective modes of lowering body temperature is through evaporation of sweat; indeed, it is the only way of losing heat when the surrounding temperature is equal to or exceeds that of the body. Normally, as the surrounding temperature falls, sweating ceases and heat is lost by radiation and conduction. In the shocked man exposed to cold all three processes are going on; and the clothing, wet with sweat or rain, permits the loss by conduction to be much augmented. Thus the surface is liable to be speedily cooled, and soon the whole body is affected. It is common for the body temperature (buccal) to be below 95 F., and readings as low as 87 and 88 have been noted in shocked men. Of course, the skin and extremities are much colder.[47] According to Cowell's observations shivering is rarely seen under these conditions. The heat loss, therefore, is not compensated for by heat production.

The increase of viscosity due to cold is not to be regarded as the only factor leading to capillary stagnation; it is probably not sufficient by itself to have that effect. Possibly cold affects capillary walls in a way leading to greater friction. Hough and Ballantyne[48] have reported a rise of capillary pressure in cooled parts of the body, together with lessened conspicuousness of the veins, and they suggest that contraction of muscles in the venules may check the outflow from cooled capillary areas. However loss

45. Denning and Watson: Proc. Roy. Soc., 1906, **78**, 318.
46. Cowell, E. M.: The Initiation of Wound Shock, p. 61.
47. Weil: München. med. Wchnschr., Sept. 11, 1917.
48. Hough and Ballantyne: Jour. Boston Soc. Med. Sc., 1899, **3**, 330.

of heat may operate, the evidence is clear that it results in concentration of blood in capillary areas; and this factor, added to the effect of low blood pressure, would favor segregation of the corpuscles in the capillaries. Significant in this connection is the commonly observed greater incidence of shock in cold weather, and especially when the cold is accompanied by rain, so that clothing is wet through. Likewise significant is the fact that as a wounded man becomes chilled his blood pressure falls, and as he is warmed his blood pressure may rise again.[46]

In addition to concentration of corpuscles and lowered temperature as conditions increasing the viscosity of the blood, there is the influence of an increase of H-ions. I have already[4] dealt with the existence of acidosis in cases of shock. Since there is evidence that acidosis has other effects than merely on the corpuscles, its influence will be considered in some detail.

### The Effects of Acidosis on the Circulation

In all probability, the lowering of the alkali reserve in cases of shock and hemorrhage is due to a fixed union of the alkali with acids, which, unlike carbonic acid, do not pass off in the lungs. The production of such acids is known to occur when oxidation is interfered with. A low blood pressure with slow circulation, cold, and corpuscular stagnation would all cooperate to check the normal oxidative processes of the body, and to increase the production of intermediary acid metabolites. Emphasis should be kept on the fact that these metabolites would be more concentrated in the tissues, and in the perivascular fluids than they would be in the blood, for they must diffuse from their source in the cells into the circulating stream. Thus, though the alkali reserve in the blood may not be reduced to a degree which would indicate a considerable increase in the H-ion concentration, the H-ion concentration in stagnant capillary regions must be still higher. And if the reserve in the blood is greatly reduced and the H-ion concentration is much raised, still greater concentration must exist in the tissues. In addition, if the tissue fluids are cooled, they will be still more acid; for, as L. J. Hen-

derson[49] has pointed out, the alkalinity of the body fluids decreases as the temperature falls. The acids thus developed in the tissues and affecting first the capillaries and small veins, with their corpuscular contents, might have effects on the circulation which would augment the action of low pressure, concentration and cold as considered above.

1. There is evidence that acid or change in the blood in the direction of acidity may have depressive effects on the *blood pressure.* Thus Hooker[50] observed that carbonic acid in minimal effective amounts always causes relaxation of vascular muscle. And Gaskell,[51] and also Bayliss,[52] proved that other acid, for example, lactic, which results from inadequate oxidation, has the same effect as carbonic acid. According to Severini's[53] studies, both microscopic and physiologic, the capillaries also are dilated by carbonic acid. Barcroft[54] has measured the increase of acid in blood coming away from the submaxillary gland after it had been stimulated to secretion by epinephrin, and accounts for the greater flow of blood through the gland, when thus made active, by the local dilator effects of the acid metabolites. As acid develops in tissues poorly supplied with oxygen, the blood vessels locally affected by these acids might reasonably be expected, on the basis of the foregoing evidence, to undergo relaxation.

An antagonistic factor is found, however, in the action of H-ions on the vasomotor centers. Oxygen lack, increase of carbonic acid in the blood, or injection of weak organic acids, all of which increase the H-ion content, stimulate the vasomoter center and cause a rise of blood pressure.[55] Thus, in acidosis with increased H-ions in the blood, vessels under nervous control would be subjected to impulses which would cause them to contract. This evidence harmonizes with that presented earlier, that in shock the arterioles are in constriction. Probably most veins are likewise involved in the subjection to nervous discharges. The capillaries, however, would be affected by the local

49. Henderson, L. J.: Am. Jour. Physiol., 1908, **21,** 441.
50. Hooker: Am. Jour. Physiol., 1912, **31,** 58.
51. Gaskell: Jour. Physiol., 1880, **3,** 66.
52. Bayliss: Jour. Physiol., 1901, **26,** xxxii.
53. Severini: Ricerche sulla Innervazione dei Vasi Sanguini, Perugia, 1878, p. 96; La Contractilita dei Vasi Capillari, Perugia, 1881.
54. Barcroft: The Respiratory Function of the Blood, Cambridge, 1914, p. 154.
55. Mathison: Jour. Physiol., 1910, **42,** 283.

increase of H-ions, and being dilated would be capable of holding an extra amount of blood. Some veins, likewise, might be relaxed. Thus capacity for the lost blood would be provided in the small vessels of the tissues rather than in the large venous trunks.

2. Increase of the carbonic acid of the blood affects *cardiac contraction*. As previously noted, Patterson[30] has shown that when the H-ions are increased by this means, cardiac muscle relaxes to a greater extent during diastole and contracts less forcibly in systole, so that the output is diminished. This condition again would tend to result in further impairment of the circulation.

3. Increase of carbonic acid increases *the viscosity of the blood*. Bence[43] reports that by breathing an atmosphere rich in carbon dioxid, the viscosity of the blood may be increased by from 25 to 52 per cent., and according to Ferrai's[56] experiments, asphyxiated blood has a viscosity approximately double that of oxygenated blood, the increase rising as the carbonic acid content rises. This factor would cooperate with low arterial pressure, concentration of corpuscles, and cold, as agencies operating to produce stagnation of corpuscles in capillaries.

4. Hamburger[57] has noted that the *size of corpuscles* is increased by the action of carbonic and other acids. The change need be only a slight one to produce demonstrable results, as, for example, when the arterial blood becomes venous. If the arterial blood contains an excess of H-ions, it would deliver to the systemic capillaries enlarged corpuscles. Corpuscles stagnant in regions ill supplied with circulating blood, where acid metabolites are most concentrated, would be especially subject to enlargement. In some of our hematocrit readings we found that the capillary corpuscular volume was greater compared with the venous corpuscular volume than it should have been according to the ratio of corpuscular and venous counts. The discrepancy was greatest in severe acidosis. The larger corpuscles might find obstruction to their progress where smaller corpuscles would not, and in any case might raise the viscosity of the blood.

56. Ferrai: Arch. di fisiol., 1904, **1**, 385.
57. Hamburger: Osmotischer Druck und Ionenlehre, Wiesbaden, 1902, **1**, 296.

All these effects of increasing the H-ions of the blood—relaxation of vessel walls (especially the capillaries), weakening of cardiac contraction and increase of blood viscosity—would be favorable to a continuance of low blood pressure. It seems probable, therefore, that when acidosis is once established, it would tend to continue the disturbances of the circulation which have been produced by other conditions.

### Vicious Circles in Shock

In relation to the observations and inferences discussed above, it is of interest to consider some of their interrelations. In all probability a number of vicious circles would be started which, if not interrupted, would lead to an aggravation of the already existent abnormal state, and which would account for the progressive nature of fatal shock. The following possibilities may have suggestive value:

1. The retarded blood flow in cooled capillaries would result in a lessened supply of heat to the regions of stasis; the parts would thus become still cooler; the cooling would increase still further the viscosity of the blood, and thereby the blood flow in the capillaries would be still further retarded.

2. Increase in the *number* of corpuscles per cubic millimeter increases the viscosity; thus the more the blood concentrates in some of the capillaries the more friction would there be in driving the corpuscles through them, and consequently a still greater accumulation in these capillaries might be expected.

3. As more blood accumulates in capillary areas, less is returned to the heart; the arterial pressure in consequence continues to fall, the force driving the blood through the capillaries is thereby progressively lessened, and the tendency for blood to gather in the capillaries where the passages are narrowest and the friction greatest is continuously augmented.

4. As the blood pressure falls, the "head" normally in the arteries is largely lost and becomes insufficient to maintain the circulation equally in all parts of the body; the blood would be forced through capillaries in which resistance is slight rather than through those in which it is increased; the blood flow in cooled, clogged capillaries, as those of the limbs, would thus

tend to be gradually diminished, with resultant greater stasis.

5. An increased H-ion content of the blood lessens the rate and the amount of union of oxygen with hemoglobin,[58] and a lowered temperature (in cooled tissues) lessens the rate of dissociation of the oxygen; a diminished oxygen supply would interfere with cellular oxidation, and thus increase the amount of fixed acid in the blood; this in turn would check the oxidative processes in the cells and interfere with heat production; in consequence, through cooling and greater acidosis, the abnormal state would be made worse.

6. As arterial pressure falls and acid accumulates in the blood, the kidneys, because of the low pressure, become less and less active; fixed acid consequently accumulates still further in the blood, and the disturbing condition is thus augmented.

Doubtless there are still other ways by which the abnormal factors may interact so as gradually to lessen the chances of recovery for the shocked individual. All these deranging processes, however, require appreciable time for their operation. It is important, therefore, that the treatment of shock be prompt, and directed toward preventing an increase of the unfavorable conditions.

## Shock as "Exemia"

The view developed in the foregoing discussion that in shock the circulatory difficulty is due to loss of blood from circulation, though not from the body, is one which has been growing in recent years, as experimental data have accumulated. The general condition was first reasoned out by Malcolm,[59] and later the idea was greatly elaborated by Henderson,[60] both of whom supposed that the lost blood was gathered in the veins. Still later, Mann[33] expressed the same idea, declaring that in shock there is a loss of circulatory fluid at a point beyond vasomotor control. And more recently the idea has been generalized for various shocklike states, traumatic and toxic, and the sugges-

58. Barcroft: The Respiratory Function of the Blood, Cambridge, 1914, pp. 27 and 53.
59. Malcolm: Tr. Med. Soc., London, 1909, **32**, 274.
60. Henderson: Am. Jour. Physiol., 1910, **27**, 152.

tion offered that capillary concentration might occur.[61] In all these states the essential feature appears to be a draining or holding back of blood from normal currency.

This view of "shock" is so different from that which probably gave reason for the original use of the term—a sudden collapse due to a severe wound—that a more descriptive name seems needed. The general employment of the word "shock" for a variety of meanings, as, for example, emotional shock, shell shock, concussion shock, besides traumatic or surgical and toxic shock, points likewise to the need of a new designation. The word *exemia* was employed by Hippocrates and signifies "drained of blood." As the discussion presented above has indicated, such is the condition in the shocked man who has not bled—his blood pressure is low because essential parts of the circulatory system have been drained of blood. The term *exemia* may properly be used to describe this condition.

## A Concept of the Development of Shock or Exemia

Although an addition to the already numerous theories of shock seems uncalled for, the facts presented in the present series of papers may be worthy of an attempt at correlation in a general statement. These facts may be listed as follows: There are primary wound shock with rapid lowering of arterial pressure, and secondary wound shock with toxemia and hemorrhage, and later lowering of the pressure. Sweating occurs, leading to loss of fluid and loss of heat from the body. The blood becomes stagnant and concentrated in the capillaries, and as the blood pressure falls there is loss of the alkali reserve of the blood (acidosis) roughly corresponding to the drop in pressure. After the discussion in the foregoing pages, how may these facts be set together consistently?

Primary wound shock—dusky pallor; rapid, thready, low tension pulse; hypotension; sweating; thirst, and restlessness—may come on so soon after injury as to be accounted for only as the result of nervous action.

61. Medical Research Committee, London: Memorandum upon Surgical Shock, Brit. Med. Jour., March 24, 1917. Janeway and Jackson: Proc. Soc. Exper. Biol. and Med., 1915, **12**, 193.

The organization of the individual (for example, a "high strung" temperament), fear and fatigue probably provide favorable conditions for the nervous response. Cowell's observation of fainting after slight wounds may perhaps be regarded as a transient state which in true shock is more persistent. Sweating and exposure lead to rapid loss of heat from the body; previous sweating, wetness of the clothing, and low external temperature favor the process. Inactivity of the wounded man and absence of shivering lessen heat protection. Thus the body becomes cold, especially the surface and extremities. In consequence of the low blood pressure, aided by the chilled tissues, there is a stagnation of corpuscles in the capillaries. The onward flow here checked undergoes concentration, so that the capillary red count is high. Prolonged lack of fluid and sweating may favor the stagnation and further concentration of the blood. The low arterial pressure can continue a flow through easy channels, but is insufficient to maintain the normal flow where resistance is high. Thus cooled regions receive less heat from the interior of the body and tend to become cooler, and thus in turn more blood accumulates. By accumulation in capillaries the return of blood to the heart is lessened until a persistent low blood pressure becomes established. The blood lost from currency produces a state equivalent to hemorrhage. Any true hemorrhage therefore exaggerates the existent shock (exemia).

When a wound has not caused a primary fall of blood pressure, but has rendered the control of the circulation unstable, unfavorable conditions, such as cold, hemorrhage and toxemia, will bring about the same sequence of events that is seen in primary shock.

As the low blood pressure continues, the alkali reserve of the blood is reduced (acidosis). Previous starvation and fatigue would favor the development of acidosis. This state, by locally relaxing vessels which are not under nervous control, by weakening cardiac contraction, and by increasing the viscosity of the blood, tends to make worse the dangerous condition which has been established. And, as pointed out in an earlier paper, the individual with acidosis is sensitized so that operation, because still further

increasing the acidosis and still further lowering blood pressure, becomes hazardous.

This conception of the events that take place in a wounded man who passes into shock gives a reasonable account of the primary effect of wounds, the influence of cold in continuing the low blood pressure or inducing it when the circulatory apparatus is unstable, the influence of warmth in restoring him in part to a fit condition, and the slowness of a full recovery. It leaves unsettled the occasion for the primary fall of pressure, though the suggestion is offered that it may be of reflex character, similar to fainting. The conception offers a hopeful outlook for the care of the shocked man, because two of the most potent factors making his chances unfavorable, cold and acidosis, can be controlled.

# THE PREVENTIVE TREATMENT OF WOUND SHOCK

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## INTRODUCTION

Whatever the nature of the bodily changes which underlie the state of shock, it is evident that the circulatory functions are in a precarious condition, and that the heart, nervous system and other organs are suffering from an insufficient blood supply. Everything should be done to promote the factors favorable to restoration of a normal and stable blood flow, and anything unfavorable to such restoration should be scrupulously avoided. There are certain practices, such as the prompt arrest of hemorrhage, the lessening of sepsis by appropriate dressings, and the reduction of pain by suitable splints, by the judicious use of morphin, and by careful transport, that are generally recognized as important measures in the care of a wounded man who is shocked or liable to shock. Besides these there are other precautions which are suggested by observations reported in the foregoing papers.

In previous papers of this series, evidence has been afforded, among others, on the following points:

1. Cooling of a person in shock is attended by a further lowering of an already low blood pressure or by continuance of the pressure at a low level.

2. Surgical operation performed on a person in shock is accompanied by a rapid and large increase of an acidosis which is already present, and by a correspondingly sudden and extensive fall in an existent low arterial pressure.

These two sets of changes are both harmful, and may turn the slender chances of a shocked man defi-

nitely away from safety and into disaster. The dangers of both cooling and surgical operation, however, can be dealt with in ways which will lead to their avoidance. The protection of the wounded man against conditions which would develop or increase shock offers, we believe, the greatest hope for his recovery.

In the following account are presented suggestions, based on preceding papers,[1] for the prevention and early treatment of wound shock and for the preparation of the shocked man for surgical operation.

## The Prevention and Early Treatment of Wound Shock

The reader will recall that of the three classes of wounds, Class B, those of moderate severity, may, through the effects of cold, hemorrhage and toxemia, develop into secondary wound shock; and that Class C, those of such severity as to endanger life unless early surgical treatment is given, show primary wound shock. Without proper care the case of secondary shock may be unnecessarily fatal, and with care the patient with primary shock may be tided over a critical period and brought to a casualty clearing station in a still operable condition.

The actual carry along the line of evacuation will vary greatly, according to the military situation and to uncontrollable circumstances, such as the weather, for example. The average wounded man on the Western front passes through the hands of several relays of bearers and at least three or four medical officers before he reaches a clearing station. Stretcher carrying along narrow trenches in the dark is laborious and slow work, even under good weather conditions. Bad weather doubles or quadruples the time required for the first stages of the journey.

If a man is hit in the front line trenches he may be at least one or two hours' journey away from the regimental aid post. And a man in a working party, a little farther back, may be as far away from the advanced dressing station. The observations recorded in the article on the initiation of wound shock have shown that it is in this stage of the journey that sec-

1. Cannon, W. B.: Acidosis in Cases of Shock, Hemorrhage and Gas Infection, p. 47. Cowell, E. M.: The Initiation of Wound Shock, p. 61.

ondary wound shock develops, and that one of the chief factors in its initiation is loss of body heat.

To send blankets to all parts of the line is impossible, but by the adoption of a waterproof sheet-blanket "packet" system a stetcher prepared for use is provided with means for preventing excessive loss of body heat. Reference to Figure 1 will make clear this simple method of carrying a dry blanket. This

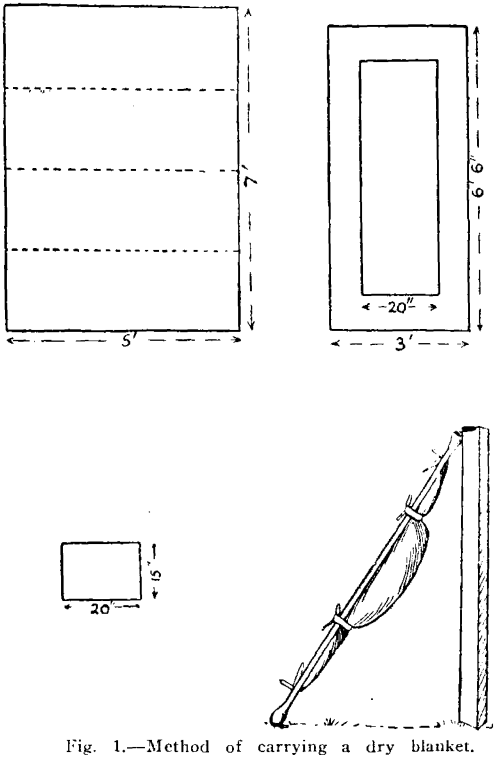


Fig. 1.—Method of carrying a dry blanket.

method has already been put into practice in a large part of the line.

All regimental stretchers at advanced bearer posts in the front line, and stretchers carried by working parties, should be equipped with this packet. The regimental bearers should be insistently instructed by their medical officer as to the importance of doing everything to prevent wound shock. The wounded

man should be guarded as much as possible against loss of heat. Efficient first-aid should be given rapidly without unduly exposing the patient to the cold for a prolonged period. A hot drink should be given at the earliest moment. Then, having been carefully wrapped up, the patient should be carried down with all speed to the regimental aid post. A well trained, intelligent orderly might be entrusted to give a tablet of morphin (one-fourth grain) by mouth in cases of severe pain. A note of this treatment should be made in the usual way.

At the *regimental aid post* it is wise to consider the general condition of the patient first and his wound second. A dry stretcher with three blankets should always be in readiness for a possible case at any time of the day or night. In the properly equipped aid post there will be a constant source of heat, such as can be supplied by any good type of small closed stove or a small open brazier with a flue to carry off coke fumes. Space will be limited; but an open stretcher, together with three blankets folded lengthwise three times, may be kept supported horizontally against the wall of the dugout behind the stove. A dry stretcher and a supply of warm blankets will thus always be at hand. A tin of water may be kept standing on the fire to provide for hot drinks and for filling hot-water bottles.

As soon as a patient arrives he should be given a few ounces of hot drink, and his wet boots and puttees removed, along with any other clothing which may cover wounds. Meanwhile the dry stretcher is prepared by arranging the first two blankets so that four folds will come underneath the patient (Fig. 2). The blankets are covered temporarily with a waterproof sheet to prevent soiling while wounds are being dressed. The man is now transferred to this prepared stretcher, which is supported on trestles and stands well over the stove. The third or free fold of each of the lower two blankets hangs down on either side and helps to form an enclosed warming chamber. If there is no constant source of heat, a hot air chamber may be made in a few minutes by use of a Primus or Beatrice stove.[2]

2. A Primus stove of ordnance pattern will burn a gallon of paraffin in twenty-four hours if operating continuously.

The patient is now becoming warmed, while the medical officer is attending to the surgical cleansing of the wounds and neighboring parts, and is applying proper dressings and splints. As soon as the dressings are finished, well guarded hot water bottles are placed in each axilla and a third across the loins or between the legs; and the third blanket, which is doubled lengthwise, is laid over the patient. The two warmed blankets which have been hanging to form the sides of the hot air chamber are lifted, carried over the patient, and tucked in. He now has four folds of blanket over him as well as underneath.

Finally, just before the patient is sent off, he is given a hot drink of sweetened tea in which a dram of sodium bicarbonate is dissolved.

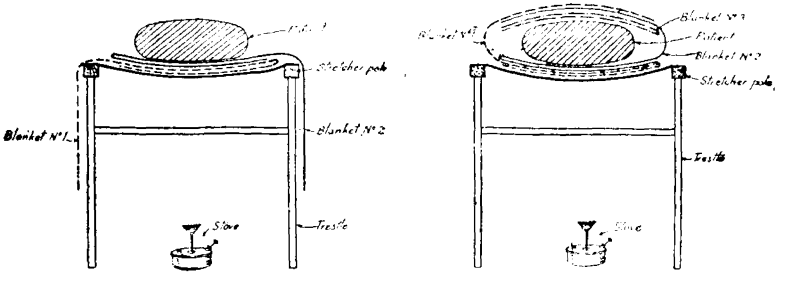


Fig. 2.—Method of folding three blankets to give four folds above and below the patient; also the formation of a hot air chamber.

At the *advanced dressing station* the warming process with the Primus stove may be repeated, but usually without changing the stretcher and blankets. Meanwhile, any necessary treatment is carried out. Before the patient is sent on, a hot sweetened alkaline drink is administered as described above, and a fresh set of hot water bottles is put in place.

The next stage of the journey is usually undertaken by means of some mechanical transport, such as a narrow gage railway or a motor ambulance. The cars are at present warmed, so that there is a lessened chance of loss of body heat on the final stages of the journey.

At the *clearing station*, application of warmth should be emphasized as the most important part of the treatment in all serious cases. While the patient is being

undressed and made ready for operation, he should be put over the same hot air chamber as has already been described. Electric warming apparatus, to be set over the patient, is at hand to apply as soon as he is ready, but it will not permit handling and treatment as will the heating from below.

While operating, again, every care must be taken to prevent loss of heat, even in summer. An electrically heated operating table is invaluable.

### Modifications of Treatment in Battle Conditions

The outlines of early treatment, sketched in the preceding paragraphs, apply to such conditions on the Western front as are "normal" for the greater part of the year. In battle, the early exposure to cold will not be so important in most cases. But, as a result of previous fatigue and loss of body fluid by sweating and hemorrhage, together with the establishment of infection and the onset of toxemia, secondary wound shock develops. Whereas the appearance and degree of primary wound shock depend on the degree of damage done to a vital organ, secondary wound shock is proportionate to the length of time the pernicious factors are allowed to work — in other words, to the period during which the wounded man is "lying out."

In the rush of dealing with large numbers, it will be impossible in the line to attend to all the details described above. Large drafts of hot sweet tea made alkaline with sodium bicarbonate can, however, be easily provided, and should be given whenever the shocked patient complains of thirst at the dressing station or at any other suitable point on the way down to the clearing station.

### Protection Against the Effects of Surgical Operation

Evidence presented in an earlier paper showed that the acidosis which prevails in cases of low blood pressure is associated with such sensitization of the body that surgical operation may result in a serious increase of the acidosis and a perilous sinking of the blood pressure. The question as to the causal relation between low pressure and acidosis has been discussed, and it appears that the two conditions may interact,

each contributing to the development of the other. Under these circumstances, advantage would be gained by protection against each of the conditions—first, against the development of the sensitizing acidosis, and, secondly, against the increase of acidosis and the further fall of blood pressure which occur at operation.

PREOPERATIVE PROPHYLAXIS

In the acidosis of diabetes, Asiatic cholera, nephritis and other pathologic states, the urine can be rendered alkaline by administration of sodium bicarbonate by mouth, as has been shown by Sellards,[3] Palmer and Henderson,[4] and other observers. The change in the reaction of the urine implies an abundance of available alkali in the blood. The possibility of fortifying the body against a reduction of the alkaline reserves in surgical cases was demonstrated by Caldwell and Cleveland.[5] They gave sodium bicarbonate by mouth and found that the normal reserves can be so increased as to avoid wholly the drop in carbon dioxid capacity that occurs in the period preparatory to operation, and also to reduce by half the rate of drop that takes place during operation. The degree to which available alkali can be provided by giving sodium bicarbonate by mouth in cases of shock and gas infection is shown in the following case:

German *Unteroffizier* H.—Admitted, August 15, with wounds of both buttocks, but in good condition. The next day the pressure was low (78 and 56), and the carbon dioxid capacity 48 per cent. Alkaline drink had been started and was continued at two-hour intervals. On the second day the wounds were evidently extensively infected with gas, but the blood pressure was 102 and 66, and the blood had a carbon dioxid capacity of 61 per cent. In spite of operation, the infection spread and death resulted.

The results obtained in the foregoing case confirm the results obtained by others in showing that by administration of sodium bicarbonate the alkaline reserves of the body can be greatly increased even in unfavorable circumstances. Since acidosis develops in shock and involves a definite risk when operation

3. Sellards: Philippine Jour. Sc., 1910, **5**, 313; Bull. Johns Hopkins Hosp., 1912, **23**, 289.
4. Palmer, W. W., and Henderson, L. J.: Clinical Studies on Acid Base Equilibrium and the Nature of Acidosis, Arch. Int. Med., August, 1913, p. 153.
5. Caldwell and Cleveland: Surg., Gynec. and Obst., 1917, **25**, 22.

is undertaken, its avoidance should be sought. The recommendation is offered that wounded men be provided with a warm drink containing a dram, or 4 grams, of sodium bicarbonate at suitable relay posts on their way from the front to casualty clearing stations, as indicated in the first section of this paper.

### OPERATIVE PROPHYLAXIS

When a wounded man, badly shocked, is brought to a clearing station, he commonly must be operated on in spite of a low blood pressure and its attendant acidosis. The lapse of time when a man is in this condition gives opportunity for the extension of infective processes which gravely menace his chances. The surgeon has to choose, therefore, between an operation when the risk is serious, and a delay during which toxemia may develop to a menacing degree.

The low blood pressure of shock has been met hitherto by the injection of normal or hypertonic salt solution, or by combinations of salt solutions and gum. There is no doubt that in some cases such injections have had definitely beneficial effects. As shown in the previous papers, part of the pathology of shock is due to a loss of fluid from the circulation and consequent concentration of the blood. The injection of salt solution adds fluid to the body and improves the circulation, so that the concentration is soon abolished,[6] and the viscosity of the stagnant blood lessened. In such cases, however, the degree of acidosis had not been determined, and injection of physiologic sodium chlorid solution or Ringer's solution makes no provision against such critical turns as have been encountered during operation or shortly thereafter in consequence of increased acidosis.[7] Not only does ordinary salt solution fail to combat acidosis, it actually *increases* an already existent acidosis. Milroy[8] has recently shown that hemorrhage results in a loss of reserve alkali, and that then injection of sodium chlorid solution alone causes a *greater* H-ion concentration of the blood exposed to a given pressure of carbon dioxid than was present before the injection.

6. Cannon, W. B.; Fraser, John, and Hooper, A. N.: Some Alterations in Distribution and Character of Blood in Shock and Hemorrhage, p. 32, Cases 65 A and 60 A.

7. Cannon, W. B.: Acidosis in Cases of Shock, Hemorrhage and Gas Infection, p. 47.

8. Milroy: Jour. Physiol., 1917, **51**, 277-279.

What is wanted is a fluid that will have the advantages already demonstrated for salt solutions — thinning of concentrated blood, lessening of viscosity and increase of blood flow with restoration of arterial pressure — together with antagonism to the state of acidosis. Such a fluid is found in sodium bicarbonate solution. Howell[9] observed years ago that alkaline injections (sodium bicarbonate) into a vein or into the rectum raised a systolic pressure of 60 or 70 mm. permanently to the normal level, and caused a marked increase in the force of the heart beats. If the pressure was lower (from 20 to 30 mm.) it was raised to 60 or 70 mm. Also the effects were relatively permanent, lasting one or more hours. These observations were confirmed by Dawson.[10] Either intravenous or intrarectal injections of sodium bicarbonate can restore the alkali reserve and abolish an existent acidosis.[11]

Injection of a fluid that will increase blood pressure has dangers in itself. Hemorrhage in a case of shock may not have occurred to a marked degree because blood pressure has been too low and the flow too scant to overcome the obstacle offered by a clot. If the pressure is raised before the surgeon is ready to check any bleeding that may take place, blood that is sorely needed may be lost. Fortunately, the injection may be made at the start of operation, just after the patient has been prepared and when the surgeon is ready to stop any hemorrhage, and it may continue as the operation proceeds.

The fluid that was injected in the cases recorded below was a 4 per cent. solution of sodium bicarbonate. Since 1.5 per cent. sodium bicarbonate is approximately isotonic with the blood plasma, the 4 per cent. solution is rather strongly hypertonic. It had previously been employed, however, in treating cases of diabetic coma,[12] and if introduced slowly (at the rate of an ounce a minute), it causes no noteworthy alteration of the blood. The fluid should, of course, be delivered to the body at approximately body temperature — it may be a few degrees warmer, but should not be colder, than that. If the solution is passed

9. Howell: Am. Jour. Physiol., 1900, **4**, 14.
10. Dawson: Am. Jour. Physiol., 1904, **10**, 35.
11. Milroy: Jour. Physiol., 1917, **51**, 278-281.
12. Peabody: Am. Jour. Med. Sc., 1916, **151**, 198.

through a tube of considerable length before it enters the vein, it should have a temperature maintained between 110 and 115 F.

The boiling of sodium bicarbonate in solution changes it to sodium carbonate. Since the bicarbonate should be injected, it should not be boiled. The solution should be made just before it is to be used, by the addition to warm sterile water the proper amount of the sterile salt.[13]

To inject the solution it is usually not necessary to lay bare a vein and introduce a cannula. If a small rubber tube is drawn around the upper arm as a tourniquet and held by a looped twist (not so tightly as to obstruct the arterial flow, which, it will be noted, has a low pressure), the veins in the elbow will in most cases become sufficiently prominent to permit a hollow needle to be introduced into one of them. The lumen of the needle should be large enough to permit a pint of the bicarbonate solution to pass in fifteen or twenty minutes when the head of pressure is only 2 or 3 feet. A glass reservoir marked in ounces permits a judgment as to the proper rate of flow. As soon as the needle enters the vein, an outflow of blood through the lumen gives evidence. When the blood thus appears, the tourniquet should be pulled loose, the rubber tubing full of the solution should be connected with the needle, and the flow allowed to start. In some few cases the veins are so obscure as to make this procedure difficult or impossible; in that case the needle or a small cannula must be inserted into the bared vessel.

The following cases illustrate results which have been obtained when the solution of sodium bicarbonate has been injected in the manner described above:

Private R. M., wounded 9:30 p. m., September 24, sustaining a compound fracture of the left tibia and fibula, and a wound of the hand, was admitted, 11:30, September 25, with

13. An ideal injection fluid for shock cases would be a solution with colloid added, as Bayliss (Proc. Roy. Soc., 1916, **89**, 380) has suggested, for example, one containing sodium bicarbonate in proper amount and 6 per cent. gum acacia. (See further Bayliss: Injections to Replace Blood, Memorandum 1, of Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions, Nov. 25, 1917.) Unfortunately, the gum contains calcium, which precipitates as calcium carbonate as sodium bicarbonate is added. If this precipitate is filtered out, the solution has the advantages of being both alkaline and viscous. The Medical Research Committee is preparing to provide in bottles sterile solutions of 6 per cent. gum, to which 2 per cent. sodium bicarbonate has been added, and the deposit filtered out.

severe gas infection of the leg; temperature 96; blood pressure, 96 and 50. Operation was begun at 12:40; the carbon dioxid capacity, 40 per cent. One pint of sodium bicarbonate solution was injected. Operation ended at 1:25; blood pressure, 100 and 60; carbon dioxid capacity, 63 per cent. The pressure was lower in the afternoon (4:30), 74 and 46, but the next morning it was 112 and 66, and it did not again fall below normal limits.

Private J. B. was admitted, September 24, with a gunshot wound of the head (three deep lacerations), contusion of left eye, fracture of both bones of the left leg, and the left foot blown off. Unconscious from cerebral injury. Blood pressure at 11:35, 54 and 20. Operation begun at 11:47; carbon dioxid capacity, 42 per cent. One pint of sodium bicarbonate solution was injected. Operation ended 12:22; blood pressure 112 and 40; carbon dioxid capacity, 58 per cent. The patient lived until the evening of the next day, and died without regaining consciousness.

Private F. B. was admitted, September 9, with one foot mangled and wounds of the right thigh and right shoulder. Wounded at 3:30 a. m.; at 11:30 a. m., just after admission, temperature 98.4, pulse 144, blood pressure 75 and 46; sweating, thirsty, face and lips pale. Operation begun at 2 p. m.; pulse 144, blood pressure 72 and 42, carbon dioxid capacity, 36 per cent. One pint of sodium bicarbonate was injected. Operation ended at 2:40; pulse 120, blood pressure 104 and 50. At 11:30 the pulse was 110, but as the patient was asleep the pressure was not taken. The next day (9 a. m.), pulse 98, blood pressure 128 and 70. Gas infection required removal of leg in the afternoon. The patient stood the operation well, and made a smooth recovery.

Private H. H. was admitted, October 3, with the left foot blown off; he had lost much blood. The pulse was thready and rapid. Operation was started at 5:10; systolic pressure 62; diastolic pressure unreadable; carbon dioxid capacity, 42 per cent. One pint of sodium bicarbonate solution was injected. Operation ended at 5:40; blood pressure 62 and 46; pulse slow and easily palpable. The next morning (9:30), pulse 96, blood pressure 104 and 72. The patient recovered.

Private F. W. was admitted, September 26, with a compound fracture of the left thigh and a wound of the left hand. Wounded 11 p. m., September 25. At 4 a. m., September 26, just after admission, temperature was 96.6, pulse 132, blood pressure 98 and 70. Operation was begun at 4:50; carbon dioxid capacity, 34 per cent. Pressure fell to 82 and 50, pulse 160. One pint of sodium bicarbonate solution was injected. At 5:35, blood pressure was 110 and 85, carbon dioxid capacity, 68 per cent. At 9:15 a. m., blood pressure 124 and 90, and at 4:30 p. m., pulse 120, blood pressure 128 and 98. A smooth recovery followed.

Sometimes a patient apparently in a condition fit for operation becomes badly shocked as the operation proceeds. The following case illustrates the use of the bicarbonate solution in such circumstances:

Private D. was wounded by shrapnel at 10:30 a. m., September 19. He complained of sweating within less than five minutes of being hit. Seen by Cowell at 11 p. m.; pulse 96, blood pressure 104 and 70; cold, pallid, sweating. Admitted to the clearing station at 11:45; blood pressure 82 and 70, pulse 96, temperature 96. At operation ten large tears of the small intestine were found, and much blood in the abdominal cavity. At the end of operation the blood pressure was unreadable. One pint of sodium bicarbonate solution was injected; blood pressure rose to 85 and 60. In the evening the pressure was 86 and 60. The following morning it was 82 and 60, and the patient was in excellent condition. That evening the pressure was 102 and 80. On the next morning, September 21, the pressure was 112 and 90. The recovery was uneventful.

The foregoing cases show that an alkaline injection at the start of anesthesia prevents the dangerous depressive effects which the anesthetic and operative procedures have in cases of shock with acidosis. The operation ends, not with an increase of the existent acidosis, but with the acidosis overcome and a normal alkaline reserve provided. And the blood pressure, instead of being perilously lowered, is actually raised during the critical period. The blood pressure may fall to some extent later, but the improved state of the patient during operation is unmistakable, and the subsequent course of shock cases in which operation has been performed with the precautions described above has been highly gratifying.

In concluding this series of papers, we take pleasure in expressing our thanks to Col. Cuthbert Wallace, C. M. C., A. M. S., and to Col. T. R. Elliott for facilitating arrangements to carry on the work and for counsel; to Lieut.-Col. Winder, R. A. M. C., for cooperative interest; and to the Medical Research Committee and the American Red Cross in France for instruments used in the investigations.