Effect of severe haemorrhage and shock on the condition of the blood

Dukes, Cuthbert Esquire

MD

1918

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THE EFFECT OF

SEVERE HAEMORRHAGE

AND

SHOCK.

ON THE CONDITION OF

THE BLOOD.

Cuthbert Esquire Dukes,

M. B. Ch. B.

Captain R. A. M. C.

B. E. F. France.

MARCH. 1918.
INTRODUCTION.

The loss of a large quantity of blood from the human body is followed quickly by a reaction, in which all the blood-forming tissues play a part. Body fluids pass into the plasma so that the blood is diluted. (1)

This transfer of fluid from tissues to blood is extremely rapid. It has been shown that during the course of an experimental bleeding, the later samples of blood are more diluted than those taken earlier. (2)

The proteins and blood corpuscles, however, are more slowly restored, the former directly from the food, the latter by an increased activity of the blood-forming cells of the bone marrow. At the same time, the other systems of the body adapt themselves to the altered conditions. The vaso centre responds to the needs of the organism by inducing a general vascular constriction which diminishes the total capacity of the circulating system. The heart beats more frequently, and so aids the emptying of the venous
arterial system. Respirations become more rapid and the patient suffers from great thirst. In the case of a patient who, at the time of his haemorrhage, sustains also severe bodily injury, such as a compound fracture of the femur, the normal recuperative adaptations are hindered by the development of the condition known as Surgical Shock. Crile and Lower (3) have made numerous investigations of the microscopic appearances of the brain, liver, and other organs, after severe injury. Emotional, Toxic, Foreign Proteid and Anaphylactic shock, and find that, as the result of painful stimuli, profound changes are produced, especially in the brain cells. These show first a hyperchromatic stage, followed by a hypochromatic stage, leading—in severe cases—to disintegration of the nerve cells. The development of this condition complicates the normal quick response to loss of blood. In the cases subsequently to be described loss of blood and severe both contributed to produce the grave condition in which these patients were found. The results of these
investigations make no contribution to the disputed question as to the concentration of the blood in pure surgical shock. It has been maintained by Bainbridge (4) and others that, in pure surgical shock, the blood is more concentrated than usual. On the other hand Short (5) has made experiments which tend to show that the specific gravity of the blood is lowered by shock and the blood is more dilute. In all the cases subsequently to be described low specific gravity readings were obtained, but all these patients had lost a large quantity of blood, and the low specific gravity was due chiefly to the normal recuperative adaptations to loss of blood, namely the passage of fluids from the tissues into the blood.

The object for which the following experiments were undertaken was to determine the actual condition of the blood in patients who had received severe injuries and were suffering from the combined effects of loss of blood and traumatic shock.
CLASS OF CASES EXAMINED.

The patients were admitted to a forward casualty clearing station from 4 to 12 hours after having been wounded. They had all sustained severe injuries, such as compound fracture of the femur or tibia, gunshot wounds of the abdomen with perforation of a hollow viscus, shell wounds with severe laceration of muscles, and, in some cases, multiple wounds. All were suffering from the effects of haemorrhage and traumatic shock, in some cases the loss of blood being the predominating element, in other cases the shock. The nature of the injuries which each patient sustained is described, and, at the time of examination, an effort was made to decide in each case whether the patient was suffering chiefly from haemorrhage or from shock. All were badly infected wounds, and many of these patients subsequently succumbed to Sepsis. Sepsis was present from the first, but at the early stage at which these observations were made, the virulent organisms with which all wounds were infected were still in the incubation period, and whatever effect
they may have had at this time was completely masked by the results of loss of blood and traumatic shock.

**CLINICAL PICTURE OF PATIENTS EXAMINED.**

All these wounded men had just escaped from the noise and excitement of a battle to the comparative peace and quietness of a field hospital. Before being wounded most of them were fatigued by the strenuous experiences of the preceding days, so that, in spite of their wounds, many of them were brought into hospital asleep on the stretcher. They had nearly all received an injection of morphia as they passed through the Field Ambulance, and fractures had been splinted and wounds redressed. They were all pale from loss of blood, and some of them were so severely wounded as to be only just conscious of their surroundings. Their respirations were quiet and regular. Some were cold as the result of exposure; all were very thirsty. Their pain had been relieved by morphia and they asked only for water, warmth and sleep.
The pulse and blood pressure were taken immediately on admission, and at the same time a drop of blood was obtained from the ear for examination, after which the patient was sent to the operating theatre for surgical treatment, or to the resuscitation tent, according as his condition demanded.

**METHODS EMPLOYED IN EXAMINATION.**

**SECTION I.**

An examination of the radial PULSE is the simplest and one of the most valuable methods of deriving information as to the state of the circulatory system. Accordingly this was first examined, as to frequency, regularity, and quality.

The SYSTOLIC BLOOD PRESSURE was measured by a Riva Rocei Sphygmanometer with a band round the upper arm, the bag being distended with air till the pulse was obliterated and then the air slowly let out till the pulse was just perceptible.

The SPECIFIC GRAVITY of the blood was examined by
Hammerschlag's method of placing a drop of blood in a mixture of chloroform and Xylol, and then adding Chloroform or Xylol until the drop neither rose nor fell. The specific gravity of this mixture was then taken.

The HAEMOGLOBIN PERCENTAGE was determined by Sahli's method, a measured quantity of blood being mixed with a dilute solution of hydrochloric acid and compared with a standard solution.

The enumeration of the RED BLOOD CORPUSCLES was made with the ordinary blood count apparatus and films were made to detect any abnormalities in the cells.

The COAGULATION TIME of the blood was estimated by receiving a drop of the patient's blood into a long capillary tube. A $\frac{1}{4}$ inch of the tube was broken off every half minute until coagulation had taken place, which was shown by the presence of a thread of fibrin between the broken ends of the tube. At the same time a drop of blood from an unwounded man was examined in
exactly the same way and the time of coagulation compared. This series of experiments comprises Section I in the tables of cases.

SECTION II. deals with examinations of the urine. The function of the urine is to eliminate unchanged such bodies as are brought to it by the blood. With the exception of hippuric acid, all chemical substances found in the urine have been excreted from the blood, though their relative proportions may have been changed in transit through the kidneys. (6) A systematic examination of the urine was therefore made in order to discover the possible presence of any abnormal substances circulating in the blood of these severely injured men. The urine was examined for blood, albumen, pus, bile, \(\text{\textit{B}-oxy\textit{By}tyric\ acid,\ diacetic acid,\ acetone,\ and\ lactic\ acid.}\)

At the same time the total nitrogen of the urine was estimated by KJELDAHL'S method of boiling a measured quantity of urine with concentrated sulphuric acid until
all the nitrogen was converted into ammonia, and then neutralising with alkali to set free the ammonia from its combination with sulphuric acid, and, by prolonged boiling, distilling the ammonia into a known amount of decinormal acid.

The Ammonia Nitrogen of the urine was estimated by MALFATTI'S method of treating neutralised urine with formaldehyde with the formation of hexa-methylene-tetramine and the liberation of an acid. The acid formed was estimated by titrating with decinormal alkali, phenolphthalein being used as the indicator.

\[ 6 \text{CH}_2\text{O} + 2\left(\text{NH}_3\right)_2\text{SO}_4 = 6\text{H}_2\text{O} + \text{N}\left(\text{CH}_2\right)_{12} + 2\text{H}_2\text{SO}_4 \]

A comparison was made between the amount of nitrogen excreted as ammonia and the total nitrogen of the urine, this giving an indication of the excess of acids over bases to be excreted from the blood.

SECTION III. The third section deals with the question of the alkalinity of the blood in these severely wounded men. As a result of the urine
examinations described in Section II., I came to the conclusion that there was a definite tendency to acidosis of the blood as revealed by the relationship of the ammonia nitrogen to the total nitrogen of the urine. Acting on the suggestion of Lieut: Oswald Robertson, of the American Army Medical Service, I made use of the Van Slyke Blood Gas Apparatus to determine the degree of the alkaline reserve of the blood. ( 7 ) Three cubic centimeters of blood were withdrawn by a syringe, without suction, from the median basilic vein and received into a centrifuge tube containing powdered potassium oxalate. This was gently moved from side to side and then centrifuged at once. When the deposit of cells had settled, the supernatant plasma was removed by a pipette and placed in a 300 C.Cs. funnel with an entrance and exit stop-cock as shown in diagram I. This plasma was completely saturated with $\text{CO}_2$ by one complete expiration from the lungs. After a normal inspiration, a complete expiration was made, the air passing over the glass beads to absorb moisture. The Funnel was
now revolved for two minutes and then allowed to stand for five minutes. One C.C. of this plasma was withdraw by pipette and examined.

The specimen of plasma was examined in the Van Slyke Blood Gas Apparatus represented in Diagram II.

The 50 C.C. pipette of the apparatus was first filled with mercury by raising the mercury levelling bulb, the enclosed air being allowed to escape by opening the stop-cock above. This was now sealed and the apparatus tested by lowering the levelling bulb, thereby producing in the pipette a Torricellian vacuum.

The fact that no leakage of air was taking place was proved by the sharp "click" with which the mercury hit the stop-cock at the top of the pipette when the leveller was raised again.

The apparatus having been proved to be efficient, the 50 C.C. pipette was filled with mercury and the one C.C. of plasma to be analysed was lowered into water in the receiver "A" in Diagram II. The end of the pipette was dipped below the level of the fluid
in the cup to prevent loss of $\text{CO}_2$. The fluid was slowly let in through the two way stop cock and the cup twice washed with 0.5 C C of water, the total amount of water admitted being about 2.5 C C. One drop of octyl alcohol was added to prevent foaming and finally the plasma was acidified with 0.5 C C of 5% sulphuric acid and the stop cock sealed with mercury. The admission of the plasma, water and acid was a very delicate proceeding.

See diagrams.
Funnel for saturating plasma with CO₂

Oxalate plasma from centrifuged specimen of blood.

Glass beads for absorbing moisture.

Diagram I

Van Slyke apparatus.
FOR REMOVAL OF SOLUTIONS FROM APPARATUS

A

B

FOR ADMISSION OF FLUIDS.

1 MM. OF LENGTH CORRESPONDS TO 0.01 C.C.

LEVELLING BULB FILLED WITH MERCURY CONNECTED BY HEAVY-WALLED RUBBER TUBE.

FOR SUBSEQUENT RELEASE OF VACUUM BY Entrance OF MERCURY TO DRAW OFF SOLUTIONS AFTER CO2 HAS BEEN EXTRACTED FROM THEM.

50 C.C. PIPETTE

DIAGRAM II

VAN SLYKE BLOOD GAS APPARATUS
Great care had to be exercised to avoid the entrance of air and to avoid the possibility of the acid acting on the plasma before they were mixed in the main pipette. It was found necessary to have an intelligent assistant holding the levelling bulb all the time, and to raise it or lower it as the circumstances required. The plasma and acid having been admitted, the levelling bulb was lowered and a Torricellian Vacuum obtained in the pipette. As a result of half a minute's shaking in the evacuated chamber, the $C_2O_2$ was extracted from the fluid in pipette and the solution was drawn out into D. Mercury was then re-admitted through C and the volume of gas evolved read off at atmospheric pressure in the finely graduated upper stem of the pipette. The observed volume was corrected by subtraction of the amount of air (0.04 to 0.05 C.C. according to temperature) which entered the apparatus dissolved in the water, and by addition of the 4 - 5% of the total $C_2O_2$ which remained unextracted because of its solubility in water. The Gas volume was
measured after a single extraction and the result calculated by means of a table (8) and converted into terms of volume percentage of \( \text{CO}_2 \) gas, measured at 760 m. m. mercury and \( 0^\circ \text{C.} \), which is bound as bicarbonate in plasma.

The results obtained from the cases examined will be found in the tables in Section III.
The condition of the circulation as revealed by

A. The Pulse.

B. Blood Pressure readings.

C. Specific Gravity of the Blood.

D. Haemoglobin Percentage.

E. Blood counts of Red Corpuscles.
<table>
<thead>
<tr>
<th>Date of Admission</th>
<th>Name and Rank</th>
<th>Nature of injuries with notes of history before admission</th>
<th>Pulse</th>
<th>Blood Pressure</th>
<th>Blood Specific Gravity</th>
<th>Haemo- Blood globin Count Percentage</th>
<th>RESULT</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-7-1917</td>
<td>Pte: Watson 12 W. Yorks.</td>
<td>Shell Wound of R. Thigh with Comp: Fracture of R. Femur. History of Haemorrhage. Wounded at 2 a.m. 1st Examination at 10 a.m. 120</td>
<td>80</td>
<td>1052</td>
<td>-</td>
<td>-</td>
<td>Died at 1.30 a.m. 29-7-1917.</td>
</tr>
<tr>
<td>26-7-1917</td>
<td>2nd. Lieut. Edwards 2/7 West Yorks.</td>
<td>Bomb wounds of both legs with comp: fracture R. Tibia and fibula, and severe laceration of L. leg. History of Haemorrhage. Wounded at 1 a.m. 1st. examination at 11.30 a.m. 132</td>
<td>70</td>
<td>1043</td>
<td>-</td>
<td>-</td>
<td>Evacuated to base. Doing very well.</td>
</tr>
<tr>
<td>5-8-1917</td>
<td>Pte: Chester 10/Royal Welsh Fusiliers</td>
<td>Comp: Fracture Tibia and fibula of left leg. Comp fracture patella of R. leg involving knee joint; Wound of left arm. Severe shock. Wounded 12 a.m. Examined 11 a.m. 96</td>
<td>110</td>
<td>1056</td>
<td>-</td>
<td>-</td>
<td>Died 10-8-1917.</td>
</tr>
<tr>
<td>Date of Admission</td>
<td>Name and Rank</td>
<td>Nature of injuries with notes of history before admission.</td>
<td>Pulse</td>
<td>Blood Pressure</td>
<td>Blood Specific Gravity</td>
<td>Haemoglobin Count</td>
<td>Result</td>
</tr>
<tr>
<td>-------------------</td>
<td>---------------</td>
<td>----------------------------------------------------------</td>
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<td>--------</td>
</tr>
<tr>
<td>12-8-17</td>
<td>Pte: Burton 20/K.R.R.C.</td>
<td>Wounds of both legs with compound fracture of both thighs; Haemorrhage and shock. Examined 2 p.m.</td>
<td>130</td>
<td>55</td>
<td>1048</td>
<td>-</td>
<td>Died 5 p.m.</td>
</tr>
<tr>
<td>3-9-17</td>
<td>Pte. Britton 2/7 W.Yorks</td>
<td>Shattered right leg with severe laceration of muscles</td>
<td>140</td>
<td>90</td>
<td>1048</td>
<td>68%</td>
<td>Evacuated to base.</td>
</tr>
<tr>
<td>5-9-17</td>
<td>Pte: Dewar 8 Black Watch</td>
<td>G.S.W. Abdomen with several holes in small intestine. Chiefly shock.</td>
<td>130</td>
<td>80</td>
<td>1052</td>
<td>88%</td>
<td>Died.</td>
</tr>
<tr>
<td>15-8-17</td>
<td>Pte: Moorhouse 2/6 W. Yorks</td>
<td>Multiple shrapnel wounds. No great element of shock or haemorrhage</td>
<td>160</td>
<td>105</td>
<td>1057</td>
<td>-</td>
<td>Evacuated to base.</td>
</tr>
<tr>
<td>24-8-17</td>
<td>Pte: Dearden 13/Kings Liverpool</td>
<td>Comp: Fracture of both femurs. Very severe shock</td>
<td>124</td>
<td>60</td>
<td>1054</td>
<td>70% Capillary Died of Venous Sepsis 3,900,000. 1-9-17.</td>
<td></td>
</tr>
</tbody>
</table>

EXAMINATION continued

Pulse: 130, 140, 130, 160
Blood Pressure: 55, 90, 80, 105
Blood Specific Gravity: 1048, 1048, 1052, 1057
Haemoglobin Count: -
Result: Died 5 p.m., Evacuated to base, Died, Evacuated to base, Died of Sepsis.
<table>
<thead>
<tr>
<th>Date of Admission</th>
<th>Name Rank &amp; Regiment</th>
<th>Nature of injuries with notes of history before admission</th>
<th>Pulse</th>
<th>Blood Pressure</th>
<th>Specific Gravity</th>
<th>Haemoglobin percentage</th>
<th>Blood Count</th>
<th>End Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>26-8-17</td>
<td>Pte: Esslemont 1st Gordons</td>
<td>G.S.W. Both thighs with very extensive muscle damage, but no fracture. Both Haemorrhage and shock.</td>
<td>120</td>
<td>115</td>
<td>1052</td>
<td>60%</td>
<td>4,600,000</td>
<td>Evacuated to Base</td>
</tr>
<tr>
<td>31-8-17</td>
<td>Pte: Notton</td>
<td>G.S.W. abdomen with several holes in large and small intestine; moderate degree of shock on admission.</td>
<td>84</td>
<td>105</td>
<td>1056</td>
<td>--</td>
<td>--</td>
<td>Died</td>
</tr>
<tr>
<td>6-9-17</td>
<td>Gnr. Hames R.F.A.</td>
<td>Shell Wounds of both legs and arms. Had lost considerable amount of blood.</td>
<td>90</td>
<td>95</td>
<td>1054</td>
<td>65%</td>
<td>--</td>
<td>Evacuated to Base</td>
</tr>
<tr>
<td>8-9-17</td>
<td>L/Cpl: Tupp 1/12 London</td>
<td>Compound Fracture of left Femur and shell wound of R. leg. Severe shock.</td>
<td>130</td>
<td>85</td>
<td>1051</td>
<td>65%</td>
<td>Capillaries 3,800,000 Veins 4,000,000</td>
<td>Died 10-9-17</td>
</tr>
<tr>
<td>9-9-17</td>
<td>Rflm: Pickles 2/1 W.Yorks</td>
<td>Compound Fracture of left femur with severe wounds of both legs. Both Haemorrhage and shock.</td>
<td>104</td>
<td>105</td>
<td>1050</td>
<td>70%</td>
<td>--</td>
<td>Died Four hours after admission.</td>
</tr>
<tr>
<td>Date of Admission</td>
<td>Name</td>
<td>Rank and Regiment</td>
<td>Nature of Injuries</td>
<td>Pulse</td>
<td>Blood Pressure</td>
<td>Specific Gravity</td>
<td>Haemoglobin Count</td>
<td>Blood End</td>
</tr>
<tr>
<td>-------------------</td>
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<td>-----------------------------------------------------------------------------------</td>
<td>-------</td>
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<td>-----------</td>
</tr>
<tr>
<td>28-9-17</td>
<td>Pte: Cameron</td>
<td>109 Battery R.F.A.</td>
<td>Patient was run over by a limber waggon and was afterwards found to be suffering from fractured ribs, crushed lung, and ruptured spleen. Accident occurred at 4 p.m. 28-9-17</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1st Examination 8 a.m. 29-9-17</td>
<td>120</td>
<td>160</td>
<td>1058</td>
<td>100%</td>
<td>5,112,000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2nd. Examination 3 p.m. 29-9-17</td>
<td>130</td>
<td>80</td>
<td>1053</td>
<td>100%</td>
<td>5,184,000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3rd. Examination 7 p.m. 29-9-17</td>
<td>Very weak</td>
<td>Very low</td>
<td>1044</td>
<td>--</td>
<td>4,880,000</td>
</tr>
<tr>
<td>3-10-17</td>
<td>Pte: Palmer</td>
<td>2/4 K.O. Y.L.I.</td>
<td>G.S.W. Abdomen. Several holes in intestine. No signs of severe shock or haemorrhage</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1st Examination 8 a.m. 3-10-17</td>
<td>110</td>
<td>140</td>
<td>1057</td>
<td>90%</td>
<td>5,972,000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2nd. Examination 3 p.m. 3-10-17</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5,456,000</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3rd. Examination 7 p.m. 3-10-17</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Note: This last reading was obtained from a vein. The others from capillaries.
<table>
<thead>
<tr>
<th>Date of Admission</th>
<th>Name Rank and Regiment</th>
<th>Nature of Injuries</th>
<th>Pulse</th>
<th>Blood Pressure</th>
<th>Specific Gravity</th>
<th>Haemoglobin %</th>
<th>Blood Count</th>
<th>End Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-10-17</td>
<td>L./Cpl: Brownbill, 2/5 W. Riding</td>
<td>S.W. abdomen with missile retained and several holes in Pelvic Colon</td>
<td>84</td>
<td>115</td>
<td>--</td>
<td>85%</td>
<td>6,232,000</td>
<td>Evacuated to Base</td>
</tr>
<tr>
<td>22-10-17</td>
<td>Rfml: Rutt, 9th. London</td>
<td>G.S.W. Chest and Side. Plural cavity penetrated and full of blood. Wound of left arm</td>
<td>120</td>
<td>100</td>
<td>1057</td>
<td>80%</td>
<td>4,976,000</td>
<td>Died 24-10-17</td>
</tr>
<tr>
<td>22-10-17</td>
<td>Rfml: Slade, 9th. London</td>
<td>G.S.W. abdomen and shell wound R. thigh. Severe haemorrhage and shock.</td>
<td>115</td>
<td>55</td>
<td>1052</td>
<td>--</td>
<td>5,672,000</td>
<td>Died Same day.</td>
</tr>
<tr>
<td>31-10-17</td>
<td>Pte: Smith, 49/Battery R.F.A.</td>
<td>S.W. Both legs with comp: fractures, both bones of each leg, necessitating immediate double amputation. Both haemorrhage and shock.</td>
<td>160</td>
<td>82</td>
<td>1049</td>
<td>65%</td>
<td>3,600,000</td>
<td>Died 8 hours later.</td>
</tr>
<tr>
<td>Date of Admission</td>
<td>Name &amp; Rank &amp; Regiment</td>
<td>Nature of Injuries</td>
<td>Pulse</td>
<td>Blood Pressure</td>
<td>Specific Gravity</td>
<td>Haemoglobin</td>
<td>Blood Count</td>
<td>End RESULT</td>
</tr>
<tr>
<td>-------------------</td>
<td>------------------------</td>
<td>--------------------</td>
<td>-------</td>
<td>----------------</td>
<td>-----------------</td>
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<td>-------------</td>
<td>------------</td>
</tr>
<tr>
<td>2-11-17 Pte: Turner 13/Kings Liverpool</td>
<td>G.S.W. Abdomen, penetrating intestines. Condition very good, without much haemorrhage or shock</td>
<td>120</td>
<td>150</td>
<td>1065</td>
<td>--</td>
<td>Veins: 5,910,000.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-11-17 Pte: Jones 1/16 London</td>
<td>Comp: fracture of R. Femur, with history of Haemorrhage</td>
<td>140</td>
<td>75</td>
<td>1056</td>
<td>70%</td>
<td>Veins: 4,488,000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Evacuated to Base. Died 3-11-17 from Peritonitis.
SECTION II. Pages 26 - 27.

URINE EXAMINATION.

An examination of the relationship between the nitrogen combined with ammonia and the total nitrogen of the urine.
### SECTION II. URINE EXAMINATION.

<table>
<thead>
<tr>
<th>Date of Admission</th>
<th>Name</th>
<th>Rank &amp; Regiment</th>
<th>Nature of Injuries</th>
<th>Ammonia Nitrogen</th>
<th>Total Nitrogen</th>
<th>Percentage of Ammonia Nitrogen</th>
<th>End Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>16-9-17</td>
<td>Sgt: Greenwood</td>
<td>2/6 W.Riding</td>
<td>Severe shell wound of Buttock and both arms. Large element of Haemorrhage as well as shock.</td>
<td>.081 grms.</td>
<td>.504 grms</td>
<td>16%</td>
<td>Died</td>
</tr>
<tr>
<td>13-9-17</td>
<td>Sgt: Parkins</td>
<td>2/6 West Yorks</td>
<td>Multiple shell wounds of both legs and both arms. Very severe shock.</td>
<td>.141 grms.</td>
<td>.952 grms</td>
<td>13%</td>
<td>Died</td>
</tr>
<tr>
<td>18-9-17</td>
<td>Pte: Speed</td>
<td>2/6 W. Yorks</td>
<td>Large shell wound of buttock with history of haemorrhage.</td>
<td>.096 grms.</td>
<td>.616 grms</td>
<td>15.5%</td>
<td>Died</td>
</tr>
<tr>
<td>13-9-17</td>
<td>Pte: Broadbent</td>
<td>2/6 West Yorks</td>
<td>Shell wound left foot and thigh. Comp: fracture R. leg.</td>
<td>.165 grms.</td>
<td>.941 grms</td>
<td>16.8%</td>
<td>Evacuated to Base.</td>
</tr>
<tr>
<td>14-9-17</td>
<td>Pte: Brooke</td>
<td>2/6 West Yorks</td>
<td>Left leg blown off by shell below knee; large element of shock as well as haemorrhage.</td>
<td>.172 grms.</td>
<td>1.12 grms</td>
<td>18.7%</td>
<td>Evacuated to Base.</td>
</tr>
<tr>
<td>Date of Admission</td>
<td>Name Rank &amp; Regiment</td>
<td>Nature of Injuries</td>
<td>Ammonia Nitrogen</td>
<td>Total Nitrogen</td>
<td>Percentage of Ammonia Nitrogen</td>
<td>End Result</td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>---------------------</td>
<td>-------------------</td>
<td>------------------</td>
<td>---------------</td>
<td>-------------------------------</td>
<td>------------</td>
<td></td>
</tr>
<tr>
<td>29-9-17</td>
<td>Pte: Cameron 109 Battery R.F.A.</td>
<td>(See &quot;Blood Examinations&quot;) (Section I. Page 22.)</td>
<td>1st. Specimen 11.30 p.m. 28-9-17</td>
<td>.079 grms.</td>
<td>.527 grms 14.6%</td>
<td>Died 29-9-17</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2nd. Specimen 11. a.m. 29-9-17</td>
<td>.104 grms.</td>
<td>.448 grms 23 %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-1017</td>
<td>L/Cpl: Brownbill 2/5 West Riding</td>
<td>(See &quot;Blood Examinations&quot;) (Section I. Page 23)</td>
<td></td>
<td>.025 grms.</td>
<td>---</td>
<td>Evacuated to base.</td>
<td></td>
</tr>
<tr>
<td>2-11-17</td>
<td>Pte: Turner 13/Kings Liverpool</td>
<td>(See &quot;Blood Examinations&quot;) (Section I. Page 24)</td>
<td></td>
<td>.03472 grms.</td>
<td>---</td>
<td>Died 3-11-17.</td>
<td></td>
</tr>
<tr>
<td>31-8-17</td>
<td>Pte: Notton</td>
<td>(See &quot;Blood Examinations&quot;) (Section I. Page 21)</td>
<td></td>
<td>.088 grms.</td>
<td>.896 grms 9.8%</td>
<td>Died</td>
<td></td>
</tr>
</tbody>
</table>
The degree of alkaline reserve of the Blood as revealed by readings with the Van Slyke Blood Gas Apparatus, showing volume of $\text{CO}_2$ Gas bound as Bicarbonate. The blood plasma of the normal adult contains 55 - 75% of its volume of $\text{CO}_2$ gas bound as Bicarbonate.
### BICARBONATE CONTENT AS DETERMINED BY VAN SLYKE'S METHOD. BLOOD READINGS.

<table>
<thead>
<tr>
<th>Date of Admission</th>
<th>Name, Rank &amp; Regiment</th>
<th>Nature of Injuries</th>
<th>Bicarbonate Content of Plasma</th>
<th>Remarks</th>
<th>End Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-10-17</td>
<td>L/Cpl: Brownbill 2/5 West Yorks. (See Blood Examination)</td>
<td>Multiple Wounds of both legs and abdomen.</td>
<td>64%</td>
<td>No definite acidosis. (See also under urine examination)</td>
<td>Evacuated to Base.</td>
</tr>
<tr>
<td>11-10-17</td>
<td>Rfim. Grange 2/8 W. Yorks.</td>
<td>Multiple Wounds of both legs and abdomen.</td>
<td>50%</td>
<td>A mild degree of acidosis present 24 hours after being wounded.</td>
<td>Died 12-10-17.</td>
</tr>
<tr>
<td>3-10-17</td>
<td>Pte: Palmer 2/4 K.O.Y.L.I. (See under &quot;Blood Examinations&quot;)</td>
<td></td>
<td>58%</td>
<td>No definite acidosis; Death due to Peritonitis.</td>
<td>Died 5-10-17.</td>
</tr>
<tr>
<td>22-10-17</td>
<td>Rfim: Rutt 9th. London. (See under Blood Examinations)</td>
<td></td>
<td>65%</td>
<td>No definite acidosis 24 hours after being wounded.</td>
<td>Died 24-10-17.</td>
</tr>
<tr>
<td>31-10-17</td>
<td>Pte: Smith 49 Battery R.F.A. (See under Blood Examinations.)</td>
<td></td>
<td>45%</td>
<td>A marked degree of acidosis within ten hours of being wounded.</td>
<td>Died Eight hours after admission</td>
</tr>
<tr>
<td>Date of Admission</td>
<td>Name, Rank &amp; Regiment</td>
<td>Nature of Injuries</td>
<td>Bicarbonate Content of Plasma</td>
<td>Remarks</td>
<td>End RESULT</td>
</tr>
<tr>
<td>-------------------</td>
<td>-----------------------</td>
<td>-------------------</td>
<td>-------------------------------</td>
<td>---------</td>
<td>------------</td>
</tr>
<tr>
<td>2-11-17</td>
<td>Pte: Turner 13 Kings Liverpool</td>
<td>(See under Blood Examinations) Section 1. Page 24. (Also under Urine Examination Page 27.)</td>
<td>64%</td>
<td>No definite acidosis. Death was due to Peritonitis.</td>
<td>Died 3-11-17.</td>
</tr>
<tr>
<td>1-11-17</td>
<td>Pte: Jones 1/16 London</td>
<td>(See under Blood Examinations) Section I Page 24.</td>
<td>59%</td>
<td>No definite acidosis.</td>
<td>Evacuated to base.</td>
</tr>
</tbody>
</table>
DISCUSSION OF RESULTS.

SECTION I. BLOOD EXAMINATIONS.

THE PULSE. With regard to the quality, regularity, and frequency of the pulse of these patients suffering from the effects of haemorrhage and shock, certain deductions may be drawn. In nearly all, the pulse was soft and easily compressible, the amplitude of its excursion was small, and the only word to describe its condition is weak. In the more severely wounded men, the pulse was imperceptible and the patient was described as "pulseless", although the heart sounds could still be heard by auscultation. Many patients admitted as "pulseless" responded to treatment by warmth and rest and after a few hours the pulse was once more perceptible at the wrist. Although not an invariable rule, it was generally found that above 130 beats per minute, the pulse became weaker as its frequency increased.

In every case examined the pulse was regular in rate and rhythm. Even in the cases in which the
element of shock was more pronounced than that of loss of blood, no trace of irregularity was ever found in the action of the heart.

The **frequency** of the pulse was always increased to a greater or lesser extent. About 120 beats per minute was the commonest figure recorded. If an analysis be made of 21 typical cases of the haemorrhage shock complex, it is found that in 14 the pulse was 120 and over. Of those whose pulse was 120 or over on admission, 9 succumbed to their injuries and 5 recovered. In those below 120, 5 died and 2 recovered. One fact is absolutely certain, and has been verified by numerous observations; that is, if a patient be admitted with a pulse between 110 and 130, too ill for immediate surgical treatment, and, if the pulse be recorded hourly, a diminution in frequency may be taken as a good prognostic sign and an increase in frequency as a bad prognostic sign. A pulse that is becoming more rapid than 130, in spite of warmth and rest, may be taken to indicate that the patient
is not responding to treatment.

**THE BLOOD PRESSURE.**

The record of the systolic blood pressure of these patients suffering from haemorrhage and shock yields results which are not easy to interpret. One fact is obvious, and that is, that a very low blood pressure is a bad prognostic sign. In the series of patients examined, no case recovered whose systolic blood pressure on admission was below 60 m.m. of mercury. But the converse does not hold true; a high blood pressure cannot be regarded as a good sign taken by itself. Some of the highest readings were obtained from patients who succumbed to their injuries within 24 hours. In attempting to interpret blood pressure readings, one has to bear in mind the known factors which influence blood pressure. These are (1) Alteration of capacity of the total system either by contraction of walls of the vessels or by pressure in them from without. (2) Alteration of the total volume of the circulating fluid. Either of these
factors would affect in the first place the mean systemic pressure. The distribution of the pressure, that is, the relative pressure in the arteries and veins would be determined by -

(3) Alteration in the output of the heart,
(4) Alteration in the peripheral resistance and therefore in the ease with which the blood can escape from the arterial to the venous side.

Practically speaking, any change in arterial blood pressure depends on the action of the heart and the condition of the arterioles, though the capacity of the circulation and the total content of the blood may have some slight effect. (9)

In the series of 21 cases recorded in Section I, no less than 18 showed blood pressure readings below 120, and 11 below 100. Of those whose blood pressure readings were below 100 on admission, 7 died and four recovered. But blood pressure readings taken by themselves do not give information of much value as regards the prospects of the patient. To estimate
To estimate the degree of the recuperative adaptation which the patient is attempting to make, one must first of all consider whether the loss of blood has been sufficient to diminish appreciably the total content of his blood. Such a diminution would, by itself, tend to lower the blood pressure. Then one must consider whether the increased action of the heart, as shown by the frequency of the pulse, is assisting to raise the blood pressure. But the main factor deciding the arterial blood pressure is the peripheral resistance of the arterioles, because increased frequency of the heart need not by itself have the effect of raising the blood pressure. The essential point is not the frequency but the output of the heart. If the heart is beating with optimum rate and force, it will keep the venous system nearly empty, (at any rate that part nearest the heart) and it is not possible for the heart to obtain more blood, however frequently it may beat (9). The factors governing blood pressure are, therefore, exceedingly
Certain facts, however, were observed in the course of these blood pressure examinations. The blood pressure was found to vary from time to time. The administration of an anaesthetic caused a fall of blood pressure in every case. Nitrous oxide produced less effect in this way than did chloroform or ether. Taken in conjunction with the frequency of the pulse, it was found by experience that a patient was getting worse whose blood pressure was falling and whose pulse was becoming more frequent. Because of the necessity of providing for these patients absolute rest and quietness, it was impossible to make frequent blood pressure examinations, although such a chart of repeated blood pressure records would have been very interesting.

**BLOOD SPECIFIC GRAVITY.**

The normal specific gravity of blood in man is usually given as from 1057 - 1066 and for a woman as from 1054 - 1061. In healthy soldiers, as the result of six readings with the hydrometer used, it
was found to be between 1057 and 1060.

In the series of patients examined and recorded in tables of Section I, it was found to be considerably lowered in nearly every case. This is of interest because the theory of shock already referred to (Page 3) maintains that in patients suffering from shock the blood is more concentrated than usual, and the specific gravity of the blood should be raised. This series of cases cannot be said to contribute much to this interesting question, because all of them were complicated by haemorrhage to a greater or less extent, and this alone would have the effect of lowering the specific gravity, because of the fluid which so quickly passes into the circulation to replace the blood that has been lost. There are two cases, however, in which the element of loss of blood was reduced to a minimum, and these both showed relatively high specific gravity readings. One, an abdominal wound, is reported on (Turner) page 24 in which the specific gravity was 1065. Another case of special interest is the one reported
on Page 22 (Cameron). This patient was found to be suffering from a ruptured spleen and crushed lung, the result of an accident. The first reading taken 16 hours after the accident showed a specific gravity of 1058 and haemoglobin percentage of 100. Seven hours later his specific gravity was 1053, and four hours later (four hours before he died) the blood taken from a vein showed a specific gravity of 1044. At this time his pulse was only just perceptible and the blood pressure could not be estimated with the ordinary mercury manometer. The falling specific gravity was due to continued haemorrhage into the peritoneal cavity. On the other hand, a rise of specific gravity accompanies improvement. In the case of the patient Edwards whose record is given on page 19, the specific gravity rose in 8 days from 1043 to 1052. This patient received a direct transfusion of blood from artery of donor to arm of recipient, as reported in notes.

The main deduction from the low specific gravity readings obtained from these patients suffering from
the effects of loss of blood and shock, is the imperative need of the system for fluids. This point is further emphasised in the note on treatment in the summary.

**HAEMOGLOBIN PERCENTAGE.**

The haemoglobin percentage was lowered in every case but one examined. This was due to the loss of blood at the time of the injury, and it was found that cases with a history of much haemorrhage had a correspondingly low haemoglobin percentage. Only in one patient was the haemoglobin percentage up to normal standard (that of Cameron on page 22.) Those who believe the blood is concentrated in Shock would expect to find a high haemoglobin percentage after severe injuries. The complicating factor of haemorrhage which was present in all the patients now being reported on was responsible for the low haemoglobin percentage.

**BLOOD COUNTS.**

Eighteen blood counts were made of the red cells in this series of cases. In two cases, the figure
was found to be above the average for a healthy soldier and both these patients had penetrating wounds of the abdomen, and, though severely wounded, had not lost much blood, and did not suffer much from shock.

In three other patients, all suffering from abdominal injuries, the blood count was approximately normal, being from 5 to 5½ million. In the remaining cases in which there was a definite history of haemorrhage the blood count was considerably reduced, in three cases being as low as 3,800,000. An attempt was made in five cases to compare the blood count from the capillaries with the blood drawn direct from a vein. In two cases, the figure for the vein was slightly higher; in two cases the figure for the capillaries was slightly higher; and in one they were approximately equal. The fact that after severe haemorrhage the red cell count and haemoglobin percentage are so much lowered, whereas the blood is much diluted (as revealed by the specific gravity) shews that the first re-action of the
organism is concerned with the supply of fluid to the blood, and that the re-establishment of the normal number of corpuscles does not take place till a later date.

THE COLOUR INDEX, which is obtained by dividing the percentage of haemoglobin by the number of red blood corpuscles, expresses the amount of haemoglobin content per corpuscle. The colour index was found to be lowered in every case and was generally represented as .9. In one or two cases, however, it was reduced still further.

No change in the COAGULATION TIME of the blood was noticed in any of the patients examined. The blood clotted in the capillary tubes in from $3\frac{1}{2}$ to $5\frac{1}{2}$ minutes according to the temperature of the room and size of the tubes chosen, but the results were always approximately the same as those obtained on a control under the same conditions.
The nitrogen of the urine is contained in the urea, ammonia, creatinine, and uric acid excreted from the blood by the kidneys. But, whilst all these substances contain nitrogen, the greatest quantity is excreted in the form of urea. The average distribution of nitrogen amongst these constituents of the urine is -

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea</td>
<td>85 - 90%</td>
</tr>
<tr>
<td>Ammonia</td>
<td>2 - 4%</td>
</tr>
<tr>
<td>Creatinine</td>
<td>3%</td>
</tr>
<tr>
<td>Uric Acid</td>
<td>1 - 3%</td>
</tr>
<tr>
<td>Hippuric acid, pigments, etc.</td>
<td>6%</td>
</tr>
</tbody>
</table>

The UREA is partly exogenous and partly endogenous in origin. The greater part is derived from the proteins of the food as a result of deamination of the amino-acids, which occurs shortly after their absorption. That part of it which is of endogenous origin is derived from a breaking down of the tissues of the body.

The nitrogen excreted as ammonia is a variable quantity and its significance will be discussed later.

CREATININE, like urea is partly exogenous and partly endogenous in origin. That part of it which is derived from the tissues of the body is remarkably
constant in starvation.

The **URIC ACID** of the urine is derived from nucleus either endogenous or exogenous, and is the result of purine metabolism.

**HIPPURIC ACID**, derived from benzoic acid, is an inconstant constituent of the urine and very little nitrogen is excreted in this form.

By means of the **KJELDAHL** apparatus already described, the total nitrogen of the urine was estimated. 5 C.C. of urine were examined and the total nitrogen present multiplied by 20 to bring it up to 100 C.CS. The results of the total nitrogen are of no interest in themselves, as it is obvious that the total nitrogen present in a given specimen of urine depends upon the concentration of the urine. But at the same time the nitrogen present in the urine as ammonia was also estimated by **MALFATTI'S** method and the results worked out for 0.0 of urine. It will be noticed at once that the figures for the ammonia nitrogen are considerably higher than those normally obtained,
and when the percentage of total nitrogen excreted as ammonia is worked out, it is found that, in place of the 4% found normally in the urine of these patients suffering from haemorrhage and shock, the nitrogen excreted as ammonia represented from 13 to 20 per cent of the total nitrogen. In the human body, the ammonia is derived from the proteins of the food, as a result of their disintegration, and the greater part is transferred to the liver where it is converted into urea and excreted as such. Only a small portion is excreted as ammonia. After the administration of mineral acids, the amount of nitrogen excreted as ammonia is increased. The same condition is observed in bad cases of diabetes, when, as a result of disordered metabolism abnormal acids, such as \( \text{B - oxy Butyric acid} \), have to be neutralised. In both these conditions, there is a marked rise in the percentage of nitrogen excreted as ammonia. It is essential to life that there should not be an excess of Hydrogen ions circulating in the body fluid. Any tendency to this acidosis is at once neutralised by the available
alkalies of the body. If the alkalies of the body do not suffice for this purpose, advantage is taken of the ammonia which would otherwise have been taken to the liver to be converted into urea. Thus, in all conditions in which there are excess of acid products to be neutralised by the circulating fluid, the nitrogen excreted as ammonia in the urine is increased. This increase in the ammonia nitrogen of the urine is an index of the degree of acidosis. The high percentage of ammonia nitrogen in the urine of these patients suffering from loss of blood and shock proves that there is a tendency to acidosis which is being combatted by the available ammonia. One case recorded is of special interest (Cameron - page 27. Urine Examinations, and page 22 Blood Examinations). This patient who was suffering from a ruptured spleen and crushed lung, showed an ammonia nitrogen percentage of 14.6 on the day before he died, and on the day on which he succumbed to his injuries, he showed an ammonia nitrogen of 23%. This last was a catheter
specimen, as the patient was unable to pass his urine. Very little urine was found in the bladder although he had passed none for twelve hours. An examination of the urine of less severely wounded patients shewed an ammonia nitrogen percentage of 4 - 6%. This is higher than the figure usually given for a healthy man and may be accounted for by the presence of lactic acid formed in muscle fatigue. This was well exemplified in the case of a healthy R. A. M. C. orderly who showed an ammonia nitrogen of 4 per cent. After a long and fatiguing bicycle ride, his urine was examined a second time and the ammonia nitrogen was found to be 6 - 8%.

The urine of these severely injured patients was examined repeatedly for abnormal constituents, more especially B-oxy Butyric acid, diacetic acid and acetone. Only in one case was even a trace of these acid products found. This sole exception was a patient who had already had an anaesthetic,
in whose urine there was found a trace of diacetic acid. The urine was tested for lactic acid also, but no trace of this was ever found. It may be concluded therefore that in these patients who suffered from loss of blood and traumatic shock, there was present a tendency to acidosis represented by the increase of the nitrogen of the urine excreted as ammonia. The cause of this tendency to acidosis is not altogether clear. Certain factors tending to produce an acidosis must be considered first of all. These patients before being wounded were all severely fatigued, both by the excitement and physical strain of the preceding days. The products of muscular fatigue must have been present in excess of the normal. To this must be added a certain degree of starvation because it is necessary for the subsequent surgical treatment of these patients that they should not be encouraged to take much food before operation. Many of them in their desperate condition could have assimilated nothing
but fluids. But these two factors, fatigue and lack of food, are insufficient to account for the relatively high ammonia nitrogen of the urine. There must be other agencies at fault in the metabolic processes, but the exact nature of these must be left for further investigation.

SECTION III.

READING S WITH THE VAN SLYKE BLOOD GAS APPARATUS

The Blood is usually described as an alkaline fluid. As measured by the common acid and alkaline indicators, however, the blood gives a neutral reaction. For instance, the blood plasma is alkaline to litmus and acid to phenolphthalein. Even with electrical methods, both blood and blood plasma reveal little or no greater concentration of $H$ or $OH$ ions than does distilled water. But it is possible to replace the carbon-di-oxide in combination with the sodium of the blood by stronger acid radicals with no change in the reaction. Thus the blood is a potentially alkaline solution, and is capable of
neutralising a certain amount of acid. This potential alkalinity of the blood is extremely important because it represents the capacity of the blood to deal with dangerous acids and to preserve the neutrality which is necessary for the existence of the organism. If fixed acids circulate in the blood, this alkaline reserve is gradually exhausted. The sodium bicarbonate present, therefore, represents the degree of alkalinity of the blood.

The normal potential alkalinity is equivalent to 0.3 per cent $\text{Na}_2\text{CO}_3$. It is increased during digestion and diminished after muscular exercise. It is greater in the early morning than later in the day. (10). Van Slyke has found that in conditions of acidosis the normal alkalinity of the blood is reduced (7). He has pointed out that after all the non-volatile acids have been neutralised, the bicarbonate which is left represents the available resources for neutralising further acids and therefore indicates the alkaline reserve of the body. The blood
plasma of the normal adult contains $55 - 75\%$ of its volume of CO$_2$ bound as bicarbonate. The urine examinations already described suggested that there was a tendency to acidosis present in the blood of patients suffering from the effects of haemorrhage and traumatic shock. The results of the estimation of carbon-dioxide bound as bicarbonate in seven cases is given under Section III. Pages 29 and 30. It will be seen that all the readings obtained are low, but all except two come within the limits of $55 - 75\%$ which Van Slyke describes as normal for a healthy man. One of these (Grange) shows $50\%$, twenty four hours after being wounded. The other (Smith) shows a definite acidosis within ten hours of being wounded. This patient was suffering from a compound fracture of both bones of each leg necessitating immediate double amputation, as the main vessels of each leg were severed. He had lost a great quantity of blood and was suffering severely from shock. He died eighteen hours after having been wounded. The bicarbonate content of a
sample of his plasma is represented as 45% which is certainly suggestive of a definite acidosis.

All that can be said of the other cases is that in them the alkalinity of the blood is slightly reduced but not to such an extent as to be labelled pathological. It may be that the tendency to acidosis is neutralised in the first place by the available ammonia resources of the body, and that it is not until a later stage that the bicarbonate content of the plasma is reduced. The low readings suggest that there is a tendency to acidosis but the formation of acid substances is not excessive.
SUMMARY.

SECTION I.

In patients suffering from loss of blood and shock -

The PULSE is regular, rapid and weak,

The BLOOD PRESSURE is usually low,

The SPECIFIC GRAVITY of the blood is considerably reduced owing to the passage of fluids from the tissues into the circulation to replace the blood that has been lost.

The Haemoglobin Percentage is reduced in proportion to the quantity of blood lost. In wounds of the abdomen, it may be only slightly below normal.

The BLOOD COUNT OF RED CORPUSCLES is low, due to the cells which have been lost.

The COLOUR INDEX is slightly reduced, being represented as about 0.9.

The COAGULATION TIME is not affected within twenty four hours of the reception of the injury.
SUMMARY.  

SECTION II.

An examination of the urine shows the percentage of nitrogen excreted as ammonia increased from the normal 4% to as much as 13% to 20%. No abnormal acids, such as lactic, B-oxymyric, diacetic acid or acetone, were found in the urine. The high percentage of ammonia nitrogen represents a tendency to acidosis, which is being neutralised by ammonia.

SECTION III.

The blood is a potentially alkaline fluid. The blood plasma of the normal adult contains from 55-75% of its volume of carbon-dioxide bound as bicarbonate. The bicarbonate represents the alkaline reserve of the blood and is an important mechanism for combatting acidosis. In the cases examined, all the readings obtained were low, but only two sufficiently low as to be described as a definite acidosis. This shows that the increased formation of acid products is not excessive.
INDICATIONS FOR TREATMENT.

Rest, warmth and quietness are the essential principles of the medical treatment of patients suffering from the effects of loss of blood and shock. Pain should be relieved by morphia and the ears should be plugged with cotton wool to ensure that the patient is not disturbed by noises. Sleep should be encouraged. The condition of the blood shows the imperative need for fluids. If these cannot be assimilated by the mouth, they should be given per rectum, subcutaneously, or intravenously. To assist the passage of fluids from the tissues into the blood, hypertonic saline may be given intravenously, the following prescription being used -

Sodium Chloride 2 grms.
Potassium Chloride 0.05 grms.
Calcium Chloride 0.05 grms.
Distilled Water 100 C. Cms.

The ideal fluid to be given is human blood. This should be given by the direct method, from artery of donor to vein of recipient, or citrate blood may be given by the Syringe method, or the preserved washed
red cells suspended in normal saline.

The tendency to acidosis must be neutralised by the administration of alkalies. These may be added to the intravenous injections, or sodium citrate may be given in doses of thirty grains by the mouth repeatedly. A convenient and palatable form for the administration of alkalies by the mouth is Raisin tea, with the addition of ten grains of Sodium Citrate to the ounce.
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(4) Bainbridge. "Surgical Shock and allied conditions"


(7) Van Slyke. "Journal of Biological Chemistry"

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(8) " " "Journal of Biological Chemistry."


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(10) Gulland & Goodall

"The Blood, a guide to its examination etc" p. 41.