Gee (1888), describing the coeliac affection, wrote (*italics mine*): "There is a kind of chronic indigestion which is met with in persons of *all ages*, yet is especially apt to affect children between 1 and 5 years old"; and again: "The coeliac disease is commonest in patients between 1 and 5 years old; it often begins during the second year of life. Sometimes from India Englishmen return sick with the coeliac affection; seldom is it met with in *adults* who have never left our island." Gee therefore was almost certainly aware of the existence of adult cases of "non-tropical sprue" as well as of the infantile cases of coeliac disease and of tropical sprue. Tropical sprue was described before Gee's time. Gee's original paper was apparently forgotten until in 1906 Herter, in the U.S.A., gave a good account of the disease in children. Thaysen gave excellent descriptions of adult cases of idiopathic steatorrhoea in 1929 and 1932. With these facts in mind my co-workers and I (Moore *et al.*, 1936) wrote as follows:

"In the interest of clarity, and in order to give credit to those observers who have been chiefly instrumental in the recognition, description, or separation of these three interesting disease entities, we suggest that coeliac disease of children be called the Gee-Herter disease, that idiopathic steatorrhoea as it occurs in adults and adolescents of non-tropical countries be called the Gee-Thaysen disease, and that the name sprue be retained for the disease as it occurs in adult residents of tropical countries. It may be that a better nomenclature will become apparent as the obscurity at present surrounding the pathogeny and aetiology of the idiopathic steatorrhoeas becomes more clear. The name idiopathic steatorrhoea is not a suitable one, for steatorrhoea is common to all three and the term idiopathic is simply a teleological confession of ignorance: steatorrhoea, also, occurs under other conditions."

Bennett and Hardwick would probably argue that it is wrong to use the name Gee-Thaysen disease with reference to an adult case in which the disease began many years back in childhood and persisted into adult life, as well as for the case in which the malady first appeared in adult life or middle age; on the other hand, the symptomatology may not be quite the same in infancy as in adult life, even in those cases in which the disease originated in infancy and with persistence of symptoms into adult life, although not infrequently the resemblance may be close. In the latter case it is admittedly not wholly desirable to call a disease of infancy by one name and to use another when it persists to a later age. While we await the further information necessary for a final nomenclature Gee's name can be attached to both; in the infantile cases the name of Herter and in the adult cases that of Thaysen could be added to denote the changing symptomatology coincident with increasing age.

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I. S. Cormanschi (*Thèse de Paris*, 1940, No. 252), who records three cases in patients aged from 19 to 29, one of which is original, describes a disease characterized by the triad of iritis, genital ulceration, and buccal aphthae. The aetiology is still obscure. Recurrent iritis with hypopyon is found in both sexes. The genital ulcers and buccal lesions are slight. The ocular lesions as a rule consist not only of iritis or iridocyclitis but also of uveitis.

BLOOD AND PLASMA TRANSFUSION IN ALIMENTARY HAEMORRHAGE

BY

D. A. K. BLACK, M.D., M.R.C.P.

Beit Memorial Research Fellow

AND

A. FORD SMITH, B.M., M.R.C.P.

(From the Nuffield Department of Clinical Medicine, Oxford)

The infusion of serum or plasma has been shown to exert a favourable effect not only in experimental shock in animals (Bond and Wright, 1938; Mahoney, 1938; Best and Solandt, 1940) but also in shock from burns (Trusler *et al.*, 1939) and wound shock (Edwards *et al.*, 1940) in the human subject. It has been argued that even in acute haemorrhage the absolute deficiency of red cells is of less importance than the vascular and circulatory derangement which prevents the effective access of blood to the tissues, and that the improvement of circulation following plasma infusion would outweigh the reduction in the number of red cells per unit volume of blood. If this were the whole story the ready availability of stored plasma and the absence of delay required to group the recipient would render it a valuable preliminary treatment of haemorrhage, even if it were necessary to give whole blood at some later time.

The object of this paper is to compare the effects of citrated whole blood with those of plasma in treating patients with massive bleeding from the stomach or duodenum. For a study of this type patients with haematemesis and melaena offer the advantages that the lesion causing haemorrhage is uniform in type, enabling a fair comparison to be made; reasonably good quantitative indications of the severity and progress of the case are given by measurements of the blood volume (Bennett *et al.*, 1938) and blood urea; and the indications for transfusion are comparatively definite. These last have been summarized by Witts (1937) as a pulse rate above 140, systolic blood pressure below 90 mm., blood urea above 100 mg. per 100 c.cm., or haemoglobin lower than 40%.

Clinical Material and Methods

The nine patients studied were all suffering from severe bleeding from the stomach or duodenum, and were kept at rest in bed on an adequate intake of food and fluid in the form of two-hourly feeds of milk and cream, with added vitamins. The first three were treated with massive transfusion of citrated blood by slow drip, and in one of them three transfusions of a litre or more were given. The fourth patient was treated with reconstituted dried serum in fourfold strength, but showed so severe an immediate reaction that this experiment was not repeated. In Cases 5 and 6 plasma infusion was started, but had to be supplemented straight away by blood transfusion; in Cases 7, 8, and 9 it was possible initially to give plasma alone, by slow drip, although Case 8 required whole-blood transfusion five days later. The plasma used was obtained by allowing blood of Group AB to stand in M.R.C. citrate bottles for forty-eight to seventy-two hours at 4° C., and aspirating the plasma into clean bottles with sterile precautions; it may be of importance that plasma so obtained is diluted bulk for bulk with citrate solution, and it may perhaps be best described as half-strength plasma.

In addition to observations on the general state of the patients, estimations of the blood urea were carried out

at intervals of a few hours on finger-prick blood, using the micro-method of Archer and Robb (1925); blood volume was estimated by the vital red method.

Haematological Findings

Whole-blood Transfusion.—The giving of whole blood is followed by an increase in the total amount of circulating red cells, as calculated from the plasma volume, directly determined by the dye method, and from the haematocrit on venous blood. The increase in red cell volume may correspond closely to the mass of red cells given in the transfused blood, as in Case 1, in which the cell volume increased by 0.4 litre after a transfusion of 0.8 litre of blood containing 0.35 litre of red cells. On the other hand, deviations from this conformity are sometimes greatly in excess of the limits of error of the method used—as carried out in normal subjects—where estimations on several consecutive days have agreed within ± 0.1 litre. It is possible to account for instances in which the observed increase in red cell volume is less than the amount of red cells supplied by postulating continued haemorrhage, other signs of which may be present, such as rising pulse rate or fall in blood pressure. In Case 3, however, a transfusion containing 0.4 litre of red cells was followed by an apparent rise in red cell volume to 0.7 litre, a discrepancy which may be accounted for in the following way. The accuracy of the calculation of red cell volume depends on the extent to which the venous haematocrit reading is representative of the cell:plasma ratio in the whole mass of circulating blood: in conditions of shock, haemoconcentration may be present in the capillaries (McDowall, 1940), so that the plasma:cell ratio in the veins is higher than in the blood stream as a whole. In these circumstances the value for total blood volume, derived from the expression

$$\text{Blood volume} = \frac{\text{plasma volume}}{\text{venous plasma : whole blood ratio}},$$

is low, so that the first estimation of red cell volume is probably too low; while the second, made after the shock has been treated by transfusion, is more accurate, and the difference between the two estimations is thus accentuated in patients in whom capillary haemoconcentration has been prominent.

The changes in plasma volume after transfusion of whole blood are largely dependent on the extent to which the restoration of blood volume by plasma dilution has progressed by the time transfusion is carried out. When the plasma volume before transfusion has already risen to a normal amount, as in Case 1, little further increase is observed; but when dilution is incomplete before transfusion, as in Case 3, increase in plasma volume is observed even in excess of the volume of added plasma, since the improvement wrought in the circulation by transfusion enables the normal process of dilution to proceed at a more rapid rate. These findings are in accordance with the observations of Bock (1921) on the tendency to maintain or restore a normal plasma volume under widely differing conditions. The haematocrit and haemoglobin percentages and the red cell count were found to be raised in all cases when blood was given.

Serum or Plasma.—In the four patients to whom serum or plasma alone was given increments in plasma volume occurred. In No. 7, who had pronounced ill effects after the plasma infusion, the increase in plasma volume was 900 c.cm.—almost equal to the litre of plasma given; in Cases 8 and 9 the increase in plasma volume was only about half the amount of plasma given, and in them the ill effects were less severe. In No. 4, who received 140 c.cm. of four-times-normal serum, the increase in plasma volume was 300 c.cm., showing that some dilution

had occurred: as it was not possible to estimate the blood volume immediately after the injection it is probable that dilution was complete but that some fluid was lost thereafter. In all four patients the estimated red cell volume after plasma infusion was within 100 c.cm. of the value obtained before infusion; there is therefore no evidence that plasma infusion in these patients effected any mobilization of red cells from the tissues, as it has been claimed to do (Brennan, 1940). Reduction of haematocrit percentage, haemoglobin concentration, and red cell count was found in the patients treated with plasma, and regeneration to the normal count from this lowered level appeared to be slower.

Blood Urea

The causes of the rise in blood urea observed after severe alimentary haemorrhage have been discussed at some length in another paper (Black and Leese, 1940); for

FIG. 1.—Blood volume, haemoglobin, and urea before and after blood transfusion in Case 1. The shaded area represents the total red cell volume.

FIG. 2.—Blood volume, haemoglobin, and urea in relation to repeated blood transfusions in Case 2.

our present purpose it is sufficient to state that the degree of elevation of blood urea bears a fairly close relation to the severity of illness, and that clinical improvement is accompanied by decline in the blood urea. A fall in blood urea after massive transfusion in haematemesis was observed by Wood (1936); but the changes in blood volume over the same period were not studied.

Whole-blood Transfusion.—The results in our patients, including those to whom blood had to be given after plasma, show that a fall in blood urea occurred after each of eight large transfusions of whole blood, carried out on six subjects; two other patients have shown a definite fall in blood urea after transfusion with citrated blood, but

are not included in the present series, since evidence of renal damage persisted after the acute haemorrhagic episode. In four of the eight transfusions here reported the blood urea was rising when transfusion was started, so the subsequent decline is not likely to have been spontaneous; moreover, it occurred with much greater rapidity than the decline in blood urea which is usually observed a few days after the haemorrhage. The time relation between transfusion, blood-volume increase, and decline in blood urea can be best seen in Figs. 1 and 2. A small and transient decline in blood urea was sometimes seen during the actual course of the transfusion, as in Cases 1 and 3 and in the second transfusion of those given in Case 2; but the major and lasting decrease in blood urea followed rather than accompanied the transfusion. The initial slight fall in blood urea may perhaps be attributed to dilution of azotaemic blood with blood of normal urea content; its transient nature can be accounted for by the rapidity with which urea diffuses from the tissues into the blood stream. The important fall in blood urea which occurs after the transfusion cannot be ascribed to dilution, and we believe that it depends on the increase in blood volume, which improves renal function, facilitating the removal of urea from the blood stream.

Serum or Plasma.—Four patients were treated with plasma or serum without any subsequent blood transfusion; in No. 4, who was given serum, the blood urea rose just after the transfusion, but fell slowly to normal in the next few days. In Case 9 the blood urea had fallen almost to normal before plasma was infused, and no further rise occurred. Cases 7 (see Fig. 3) and 8 each

FIG. 3.—Blood volume, haemoglobin, and urea before and after plasma infusion in Case 7.

had azotaemia for more than five days after the infusion, in contrast with patients who had been given whole blood, and even with patients left to recover spontaneously from haematemesis. Plasma and serum therefore did not produce the same rapid fall in blood urea as did transfusion of whole blood.

General Effects

The beneficial effects of whole-blood transfusion in patients who have lost blood are well known. In the six patients who were given plasma immediate subjective or objective clinical improvement was absent; all showed some harmful effect, and in two of them the reaction was so severe that massive blood transfusion had to be used at once. The chief complaints during plasma infusion were of headache, faintness, and a vague sense of irritability. Objectively, increase in pallor, quickening pulse, and drowsiness leading to actual unconsciousness were noted; in the first patient, who had a low haemoglobin at the outset, unconsciousness persisted for over half an hour. Even after recovery from the immediate effects

of plasma transfusion clinical improvement, and especially blood regeneration, were notably slower than in the patients to whom whole blood had been given; No. 4, who had serum, and whose plasma volume increased only by 300 c.cm., was an exception to this.

Discussion

Our experience has been that in patients suffering from alimentary haemorrhage plasma has not only been of no value as a substitute for whole blood in therapy, but has given rise to untoward and even dramatic ill effects. We have used plasma prepared in the same way in the treatment of burn shock, with good results and with no adverse reaction; it is therefore unlikely that any inherent defect of the plasma is responsible for the unsatisfactory response of patients with haemorrhage. Such patients are distinguished from patients with burn shock, who respond well to plasma, by the low content of haemoglobin in the blood. The most obvious effect of giving plasma is to increase the dilution of the formed elements in the blood, so that blood volume is increased at the expense of haemoglobin concentration. In this connexion the observations of Robertson and Bock (1918) in the war of 1914–18 are of great significance. They found that when the haemoglobin was below 40 to 50% spontaneous blood dilution did not occur; it seemed, in fact, that maintenance of haemoglobin at such a level was of more importance to the organism than restoration of the blood volume to full normal limits. It is reasonable to ascribe the ill effects of plasma infusion in patients with severe haemorrhage to the rapid and forced dilution of the blood to a greater extent than would naturally occur in response to haemorrhage. This interpretation is supported by the very rapid appearance of ill effects in patient No. 4, who received fourfold serum, and in whom blood dilution was likely to have occurred even more rapidly than with ordinary plasma.

It must be emphasized that plasma is of great value in the treatment of shock from such causes as burns and intestinal obstruction; when, however, shock is associated with a degree of haemorrhage sufficient to lower the haemoglobin percentage by a half or more, our results suggest that the giving of plasma is contraindicated, even as a temporary measure pending whole-blood transfusion. This conclusion does not conflict with the favourable results reported by Brennan (1940), since the lowest red cell count in his series was 2.99 millions: there is a stage of post-haemorrhagic shock, before blood dilution has occurred, when plasma infusion seems to be of value. When, however, the haemoglobin has fallen to 50% or less, as happens in many cases before the patient comes under observation, the time for plasma is past, and benefit will be obtained only from transfusion of whole blood. The practical recommendation we would wish to make is that any patient who is known to have bled should only be given plasma if the haemoglobin percentage is over 50; otherwise there is a risk of serious collapse, which may demand for its treatment an amount of whole blood larger than would have been required in the first instance. On the other hand, in recent haemorrhage, when dilution is slow on account of shock and the haemoglobin percentage remains high, plasma infusion is beneficial, as shown by the results of Brennan (1940) in patients and of Buttle *et al.* (1940) in animals.

Case Protocols

Case 1: Bleeding Peptic Ulcer.—This patient, a man aged 48, had had vomiting and dyspepsia for three weeks before admission for haematemesis on August 14, 1939. He was pale and shocked, with a blood pressure of 100/50, pulse rate 110, and temperature 98.4°. After transfusion of citrated blood, carried out on the day of admission, his blood pressure

became 110/70, and remained at this level. His pulse rate dropped to 80. Although he had a pyrexia of 102° F. on the day after transfusion his general condition was much improved. He made a good recovery, but had to be evacuated from hospital before full investigation had been carried out. The results of blood examination are as follows, and are also represented in Fig. 1.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Blood Urea (mg./100 c.cm.)	Haemoglobin % (Haldane)	R.B.C. (millions per c.mm.)	W.B.C. per c.mm.
14/8/39 3 p.m. 5 p.m. 6 p.m. 8 p.m.	3.17	26	4.3	57 68	60	2.75	13,600
15/8/39 2 a.m. 8 a.m. 10 a.m.	3.3	31	4.8	62 69 42	76	3.44	8,800
18/8/39 21/8/39 26/8/39 28/8/39				56 60 32 28	82		

Case 2: Bleeding from Gastric Ulcer.—The second patient, a woman aged 43, had had dyspepsia since 1926; gastro-enterostomy was done in 1929 for duodenal ulcer. She was admitted on September 25, 1939, having vomited blood repeatedly on that and the previous day. She continued to vomit in hospital, and her blood pressure, which had been 180/100 on admission, fell to 95/30 on the 29th, while her pulse rate rose from 90 to 130. She was given repeated transfusions of citrated blood, as detailed below, and showed temporary improvement after each, though she did not stop vomiting until October 4. A barium meal showed an ulcer high up on the lesser curvature, and this appeared at gastroscopy on November 1 to be in process of healing. The effect of the transfusions is indicated by the results given below and in Fig. 2; it is of interest that the small infusion of 400 c.cm. of blood produced no lowering of the blood urea.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Blood Urea (mg./100 c.cm.)	Haemoglobin % (Haldane)	R.B.C. (millions)	W.B.C.
26/9/39 27/9/39 28/9/39 29/9/39	2.7	20	3.4	38 58	50	3.14	9,800
11 a.m. 3 p.m. 6 p.m. 10 p.m.	2.1	14	2.45	96 120 88	28	1.93	—
30/9/39 9.30 a.m. 12 noon 6 p.m. 11 p.m.				82 52 120 82 100	36		
1/10/39 10 a.m. 2/10/39 10 a.m. 2 p.m. 6 p.m.	2.8	9	3.1	64 76	23		
3/10/39 10 a.m. 6 p.m.				76 88	24		
4/10/39 5/10/39 6/10/39 7/10/39 10/10/39 14/10/39 20/10/39 3/11/39 4/11/39	2.9	20	3.6	45 42 44 44 50 80	— 34 42 48 50 80	2.47	4,400

Case 3: Bleeding from Gastric Carcinoma.—Although dyspepsia had been present for seven years in this patient, a man aged 60, his pain had become more constant and severe in the month preceding his admission on November 6, 1939. He had small haematemesis while in hospital, but the first massive bleeding occurred on December 4, when he had three haematemesis of upwards of a pint each, and became pale, with a feeble pulse, the rate being 120 per minute. He was given a transfusion of a litre of citrated blood, with great improvement in his general state and a fall in the pulse rate to 80. A barium meal on December 19 showed the presence of a large mass involving the body of the stomach.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Blood Urea (mg./100 c.cm.)	Haemoglobin % (Haldane)
4/12/39 4 p.m.	2.1	25	2.8	67	60
5/12/39 9 a.m. 11.30 a.m. 12.30 p.m. 4 p.m. 7 p.m. 10 p.m.	Transfusion started			100 146 114 114 128	60
6/12/39 9 a.m. 10 a.m. 12.30 p.m. 4.30 p.m.	3.5	28	4.9	102 84 70	64
7/12/39 8/12/39 9/12/39				63 62 60	66 62 62

Case 4: Gastric Erosion.—This patient, a man aged 44, was admitted on May 10, 1940, after repeated small haematemesis which began the previous day. He was pale and shocked, with a pulse of 144 and a blood pressure of 82/60; epigastric tenderness was present. Two hours after admission reconstituted dried serum in fourfold concentration was given intravenously by syringe at a rate of 10 c.cm. a minute. After five minutes respiration quickened and deepened, the patient became restless, and he vomited twice. After 140 c.cm. had been given a convulsion occurred and the injection was stopped. For the next half-hour he was collapsed and semi-conscious, but could be roused. He was well on the following morning, apart from slight backache. The residue in the bottles used was found to be sterile. On May 11 his blood pressure was 114/65 and the pulse rate 98. Subsequent recovery was uneventful.

Date and Time	Haemoglobin (%)	R.B.C. (millions)	Plasma Volume (litres)	Blood Volume (litres)	Haematocrit (%)	Blood Urea (mg./100 c.cm.)	Plasma Protein (gm./100 c.cm.)	Plasma Albumin (gm./100 c.cm.)	Plasma Bicarb. (vol./100 c.cm.)	Plasma Chloride (mg./100 c.cm.)
10/5/40 3.30 p.m. 5 p.m. 5.15 p.m. 9 p.m.	62 62 50	3.0	2.8	3.9	29	132 130 152	6.0	4.5	46	560
11/5/40 10 a.m. 9 p.m.	58	2.6	3.1	4.1	25	90 62 44	5.0	4.0	54	578
12/5/40 13/5/40 14/5/40 18/5/40 23/5/40 4/6/40	58 44 50 52 60 74	3.0 4.0				38 35 38 34				

Case 5: Bleeding from Gastric Ulcer.—This patient, a man aged 38, was transferred from a cottage hospital, where he had been under treatment for haematemesis for six weeks. On admission to the Radcliffe Infirmary his pulse rate was 120, blood pressure 60/40, and haemoglobin 18%, and he appeared to be in shock. In spite of the low haemoglobin it was decided to give him plasma, since a good supply of compatible blood was available if his response to plasma were poor. After 300 c.cm. of plasma had been given at the rate of 200 c.cm. an hour he became unconscious, following some preliminary restlessness, and a change-over to blood was made. He then improved rapidly, and his blood urea fell in the usual fashion, as shown below. He was later transferred to a surgical ward, where gastro-enterostomy was done for relief of pyloric obstruction caused by a chronic gastric ulcer.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Blood Urea (mg./100 c.cm.)	Haemoglobin % (Haldane)
4/2/40 2.30 p.m. 3.30 p.m. 5 p.m. 5 p.m.	4.25	9	4.67	76	18
5/2/40 10 a.m. 6.2/40 7.2/40 10.2/40 20.2/40	4.0	21	5.1	45 37 35 28 30	17 38 36 38 40 50

Case 6: Duodenal Ulcer.—This man, aged 58, had several haematemeses on August 6, 1940, and was admitted the next day at noon. He was pale and slightly cyanosed, with blood pressure 125/75 and pulse 146. A plasma infusion was begun, but the patient became restless and irritable, and looked much worse. Oxygen was administered with the B.L.B. mask, but did not appear to arrest the deterioration in his condition. His blood pressure fell to 100/46, and after a pint of plasma had been given a change-over to blood had to be made, and this was followed by the usual improvement. On August 8 his pulse was 100 and blood pressure 122/56. Recovery was normal, and he was discharged on September 20.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Haemoglobin %	R.B.C. (millions)	Blood Urea (mg./100 c.cm.)
7/8/40						
2 p.m.				30		120
4 p.m.	4.0	12	4.6	28	1.5	118
6 p.m.	Plasma drip begun					
8.30 p.m.				28		120
9.30 p.m.	Plasma replaced by blood after 500 c.cm. had been given					
12 p.m.				30		120
8.8.40						
11 a.m.						126
4 p.m.		16		38	1.73	72
10/8/40 ..				40		
30/8/40 ..				50	2.9	
14/9/40 ..				70	3.8	
2/10/40 ..				88		32

Case 7: Bleeding from Gastric Ulcer.—This man, aged 59, had had dyspepsia of gastric ulcer type since 1917; he had had slight haematemesis and considerable melaena for five days before admission to hospital on February 25, 1940. On admission he was pale and shocked, with blood pressure 90/50 and pulse rate 140. As his haemoglobin was 75% it was decided to give him plasma; so between 12 noon and 5 p.m. on the 26th a litre of plasma was infused. The patient became confused and apathetic during the plasma administration, but consciousness was not lost. Following the plasma infusion there was no subjective improvement such as is normally brought about by blood transfusion in these cases. On the day after the infusion he felt better than he did immediately after, and blood-volume estimation showed that he had by that time lost the greater part of the increase in plasma volume brought about by the infusion. A barium meal on March 11 showed a chronic gastric ulcer with hour-glass deformity. The results of blood examination are as follows, and are shown also in Fig. 3.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Blood Urea (mg./100 c.cm.)	Plasma Protein (gm./100 c.cm.)	Haemoglobin % (Haldane)	R.B.C. (millions)
25/2/40				90			
9 p.m.							
26/2/40				68	—	{ 70 (venous)	3.29
11 a.m.	2.9	34.5	4.5	68		{ 72 (capillary)	3.51
12 noon	Plasma infusion started						
2 p.m.				64		68	
4 p.m.				70		64	
5 p.m.	Plasma infusion stopped (1,000 c.cm.)						
6 p.m.	3.8	29	5.4	50	5.0	{ 68 (venous)	3.25
27/2/40						{ 66 (capillary)	3.14
3 p.m.	3.1	27	4.25	74	5.0	{ 64 (venous)	3.0
28/2/40				76		{ 62 (capillary)	2.9
29/2/40				62		64	3.1
1/3/40				68		68	
2/3/40				68		68	
5/3/40				71		68	
8/3/40	Plasma bicarb. 55 vol. %						
	Plasma NaCl 592 mg./100 c.cm.						
12/3/40				50		64	
16/3/40				48		66	3.3
19/3/40				46		66	

Case 8: Bleeding from Duodenal Ulcer.—This patient, a man aged 56, had a seven-years history of dyspepsia, with occasional attacks of melaena. He had been passing dark stools for two days before his admission to hospital on February 26, 1940, and was very pale, with pulse rate of 100, blood pressure 90/45, and haemoglobin 56%. The next day he was given a litre of plasma over a period of three hours; there was a complete absence of improvement, and the pulse, which had been running at a level of 100, rose during the course of the infusion to 120, and remained at that level until

the following day. The systolic blood pressure fluctuated between 90 and 120, and was 97 at the close of the infusion. Half an hour after beginning the infusion the patient complained of headache, but this was not severe. On March 4, since the haemoglobin was not rising, a blood transfusion was given, with definite improvement in the patient's subjective and objective condition, and he made a good recovery. A barium meal on March 12 showed deformity of the duodenal cap, although an ulcer crater was not actually demonstrated. The numerical values obtained are given below.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Blood Urea (mg./100 c.cm.)	Plasma Protein (gm./100 c.cm.)	Haemoglobin % (Haldane)	R.B.C. (millions)
27/2/40							
10 a.m.				74		56	
12 noon	2.65	24	3.5	82	4.5	{ 56 (venous)	2.71
						{ 54 (capillary)	2.60
1.20 p.m.	Plasma infusion begun						
3 p.m.				106		40	2.33
4.20 p.m.	End of plasma infusion (1,000 c.cm.)						
6 p.m.	3.1	19	3.85	60		{ 46 (venous)	2.3
10 p.m.				74		{ 40 (capillary)	2.5
8/2/40						36	
12 noon	3.34	19	4.1	68	4.8	{ 42 (venous)	2.08
						{ 42 (capillary)	2.17
29/2/40 ..				64		42	
1/3/40 ..				58		40	
2/3/40 ..				64		40	
4/3/40							
10.30 a.m.	3.57	21	4.52			36	2.41
11 a.m.	Blood transfusion begun						
2 p.m.				52		42	
4.30 p.m.	End of blood transfusion (1,000 c.cm.)						
				53		50	
5/3/40 ..	3.4	28	4.72	38		48	
8/3/40 ..	Plasma bicarb. 61 vol. %						
	Plasma NaCl 555 mg./100 c.cm.						
12/3/40 ..				49		50	
16/3/40 ..				40		54	
19/3/40 ..				41		62	

Case 9: Duodenal Ulcer.—After melaena for two days and one attack of haematemesis this patient, a man aged 47, was admitted to hospital on September 16, 1940. His blood pressure was 124/76 and pulse rate 90; he was pale but did not appear to be suffering from shock. A plasma infusion was started at 7 p.m. on the day of admission. There was no severe reaction, but headache began three hours after the infusion started, and lasted for over twenty-four hours. On the day after plasma infusion the pulse was 85 and blood pressure 122/64. He made a good recovery, and was discharged on October 14.

Date and Time	Plasma Volume (litres)	Haematocrit %	Blood Volume (litres)	Haemoglobin %	R.B.C. (millions)	Blood Urea (mg./100 c.cm.)
16/9/40						
3 p.m.				50		88
5 p.m.	3.5	18	4.3	46	2.1	
7 p.m.	Plasma infusion started					
10 p.m.				38		52
17/9/40						74
12.50 p.m.	Plasma infusion stopped after 1,000 c.cm. had been given					
10 a.m.				36		54
2 p.m.	4.0	14	4.7	38	1.6	39
18/9/40 ..				36		42
24/9/40 ..				46	2.3	39
5/10/40 ..				54	3.1	
5/11/40 ..	3.4	43	6.0	90	4.8	35

Summary

In nine patients with severe haemorrhage from the stomach or duodenum plasma compared unfavourably with whole blood in its effects on the blood volume and haemoglobin concentration. It did not lower the degree of azotaemia to the same extent as whole blood, and the general condition of the patients was adversely affected. It is suggested that the ill effects of plasma in these cases are due to forced dilution of the blood in excess of the limits within which blood dilution is favourable to recovery from haemorrhage. The circumstances which determine the usefulness of plasma in post-haemorrhagic shock are discussed; in general terms, plasma is contraindicated when the haemoglobin is less than 50%.

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AMPUTATIONS IN BED

BY

H. A. BRITTAIN, M.A., M.Ch., F.R.C.S.

Orthopaedic Surgeon, Norfolk and Norwich Hospital

Orthopaedic surgeons are proud, and justly so, of their aseptic operative technique. Consequently, when one of them advocates the performance of an operation in the patient's bed one can visualize the hackles rising. My apology and excuse is, "C'est la guerre," and the emergencies of war have forced us to modify the technique which in peacetime is rightly regarded as essential.

Air raids, the mechanization of the Army, and the intensified industrial output have caused a great increase in the numbers of injuries to the limbs. Motor-cycle accidents to dispatch riders have resulted in as many as sixty-one serious fractures in six months in one provincial hospital alone. These patients are often suffering from severe shock, and multiple injuries may be present. Prompt amputation of mangled limbs before adequate resuscitation is to be condemned. Nevertheless the patient may recover to a certain extent from the primary shock, and then his condition may degenerate into secondary shock as a result of extensive damage to the limb.

Indications for Amputation

The indications are: (1) severe compound fractures or crushed limbs that cannot survive owing to interference with the circulation; (2) infection by anaerobic organisms, with spreading gangrene; (3) laceration or crushing of soft parts to such an extent that (a) the presence of the limb is retarding recovery from or accentuating shock, or (b) the subsequent scarring will leave a useless limb.

Choice of the right moment to amputate is of paramount importance, and equally so is the execution of the operation with the least possible disturbance to the patient. Temporary suspension of continuous-drip transfusion, and transport to the theatre necessitating lifting the patient from the bed to the stretcher, from the stretcher to the theatre table, from the theatre table to the stretcher and back to bed again, often have a deleterious effect. There may also be difficulty in restarting the blood transfusion, which will certainly be necessary. Multiple injuries may render transport difficult or impossible. It may, of course, be possible to wheel the bed accompanied by the drip transfusion into the theatre, and in some hospitals, and in the absence of an overwhelming rush of casualties, this may be considered preferable. A mobile surgical team might have to perform an amputation in the patient's house. For these patients amputation in bed in the ward may be a life-saving measure, and it has been found that it can be quickly, smoothly, and expeditiously carried out if carefully planned beforehand and a formal technique is faithfully observed.

Technique

Essential Materials.—Samways' tourniquet, or Callandar's modification of same; large amputation knife; dissecting forceps; amputation saw; artery forceps; catgut or silk; sulphanilamide powder; vaselined gauze arranged as a dressing and laid on wool 8 in. by 8 in. and 1 in. thick; bandages; three strips of 2-in. strapping two feet long.

Anaesthesia.—Intravenous pentothal or cyclopropane. The former is to be preferred for ease of administration.

Assistants.—Three are required, apart from the anaesthetist, if available. Two is the minimum.

Operation.—Speed is desirable and often essential. An amputation through the lower third of the thigh is described, although the same technique can be adopted for any site. The guillotine operation is of course the method of choice in these severely shocked patients. The tourniquet is applied and kept in position by one assistant, who twists it round and holds it with his hands. Another assistant lifts the limb by the foot. Mackintoshes and towels are applied, and the incision is begun on the postero-medial aspect of the limb, continued over the front of the circumference of the limb, and carried down to the bone. The bone is quickly divided with a saw, the skin being pulled up as far as possible by pressure with the left hand. The femoral vessels are clamped and tied with silk or No. 2 catgut. An assistant then releases the tourniquet slightly, and any other strongly bleeding vessel is clamped and ligatured. The generalized ooze is ignored. The tourniquet is now held tight again, and sulphanilamide powder is dusted on to the raw area. Vaselined gauze and wool are then applied to the stump and firmly bandaged on. The dressing is further anchored in place by strapping, which passes over the end of the stump from the abdomen to the buttocks and from the inner to the outer aspect of the thigh. Below are given two illustrative cases.

Case I

A munition worker aged 18 became an air-raid casualty at 2 p.m. in August, 1940. The further history of his case is as follows:

3.15 p.m.—Admitted to hospital in fairly shocked condition. Blood pressure 120/80. Pulse 80, good volume. Talking clearly and well. Compound comminuted fracture of head of tibia. Morphine given and anti-shock treatment instituted.

4.15 p.m.—Plasma transfusion given preparatory to operation.

4.30 p.m.—Condition degenerating. Cold, perspiring, and cyanosed. Blood pressure 95/60. 6 litres of oxygen given with B.L.B. mask.

9 p.m.—Condition much worse. Blood pressure 85/50. Pulse rate risen from 100 to 160. Blood transfusion administered.

11 p.m.—Amputation through lower third of thigh performed in bed.

11.10 p.m.—Immediately after operation blood pressure 70/40.

Next day, 12.30 a.m.—Blood pressure 80/60. Pulse 140.

8.30 a.m.—Blood pressure 120/80. Pulse 120. Steady gradual improvement followed.

Case II

An R.A.F. dispatch rider aged 24 had a motor-cycle accident at 8.30 a.m. on November 15, 1940. His case proceeded as set out below.

November 15, 11.30 a.m.—Admitted to hospital with severe shock. Compound comminuted fracture of tibia and fibula plus fractured pelvis. Large wound with extensive laceration of soft parts. Pulse 100—irregular and of poor volume. Resuscitation started at once.