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TREATMENT OF BURN SHOCK WITH PLASMA AND SERUM

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The use of plasma or serum protein in the treatment of shock from extensive burns has frequently been advocated, and there is ample theoretical justification for protein-containing infusions, in view of the constant presence of haemoconcentration and reduced plasma volume in any severe burn. Nevertheless, published accounts of the effects of such treatment are few, and the data given are somewhat incomplete, particularly with regard to blood volume and serum electrolyte concentration. Trusler *et al.* (1940) and Elkinton (1939) have each reported two patients who were treated by plasma infusion with benefit.

This paper deals with eight patients with burns or scalds, of whom seven were treated by plasma or serum, or both. The plasma used was obtained by separation from blood taken into standard M.R.C. bottles containing 180 c.cm. of citrate solution, so that the final product may best be described as "half-strength plasma." The solution of serum protein was made up by the addition of sterile water to dried serum, and the solution used was four times the concentration of the original serum. The schedule of investigations which we proposed to ourselves included the determination of blood and plasma volume, haematocrit, haemoglobin and red cells, protein and albumin, chloride, bicarbonate and urea, sodium and potassium. These were to be determined before and after the infusion of plasma or serum and after complete recovery from the burn. It was not possible to carry out the full programme in all cases, since one or both arms were often involved in the burn; but a complete record was obtained in Cases 3 and 5; and, with the exception of blood volume, all the proposed data were obtained in the other cases. Besides the blood examination, records were kept of the pulse and blood pressure, and frequent notes of the patient's general condition were taken.

The following were the technical methods used: Blood and plasma volume: vital red method (Keith *et al.*, 1915). Haemoglobin: Haldane. Blood urea: urease and Nesslerization (Archer and Robb, 1925). Plasma protein: biuret colorimetric (Harrison, 1937). Plasma chloride: Whitehorn titration (Harrison, 1937). Plasma bicarbonate: volumetric (van Slyke, 1917). Serum sodium: triple acetate (Peters and van Slyke, 1931). Serum potassium: cobaltinitrite (Peters and van Slyke, 1931).

Results

The numerical findings, together with brief clinical notes on the cases, are given in an appendix. The data obtained may be discussed in their relation to the pathology of burn shock and the effect of treatment with plasma proteins.

General Clinical Observations

With the exception of the sixth case, in which injury was slight in extent and degree, the burn was followed in less than an hour by the appearance of severe and increasing shock. Pallor, with slight cyanosis, of the lips and skin, shallow frequent breathing, a mental state bordering on apathy, a supine position with occasional restless alteration, were all in conformity with classical descriptions of the shock syndrome. The superficial veins were collapsed and contained dark inspissated blood, which flowed very slowly into the syringe. Spontaneous vomiting was not common, but even small amounts of water provoked retching. The pulse was rapid and small in volume, and the systolic blood pressure was often below 100 mm., although fall in pressure was relatively late in onset. There was no remission after the onset of shock symptoms such as would justify a distinction between primary and secondary shock in these patients.

Three infusions of fourfold serum, from 150 to 300 c.cm. in amount, and six infusions of plasma, from 500 to 2,000 c.cm., were given to seven patients. The fourth patient, who was burnt over more than half the body surface, did not respond to concentrated serum infusion. The other eight infusions were all followed by definite, in some cases dramatic, improvement. Peripheral cyanosis disappeared, the veins filled out, and mental apathy gave place to alertness; this mental quickening was sometimes evidenced by a new consciousness of pain in the burnt area. Patients were better able to take fluids by mouth, and to retain them. The blood pressure rose in cases in which it was much diminished before the infusion; increase in the volume of the pulse was more notable than decrease in rate, although in the third case a pulse rate of 156 fell to 100 within an hour, during which 300 c.cm. of fourfold serum had been given. No post-transfusion reaction occurred after either serum or plasma.

Haematology

Moon (1939) has emphasized the importance of haemoconcentration in the early recognition of shock. The results on our patients give full support to his conclusions, which were mainly based on work with animals. In Case 6 the

haemoglobin and haematocrit percentages were decidedly raised, although blood pressure and pulse were normal and signs of shock were very slight. Moreover, the rise in red cell count, haematocrit, and haemoglobin persisted in all the cases for many hours after other signs of shock had disappeared. Haemoconcentration is in itself important in the pathology of burn shock as increase in red cells increases the viscosity of the blood (Bence, 1906), adding to the circulatory embarrassment; in Case 4, the only patient of the series who died from shock, the haemoglobin rose from 106 to 132 and the haematocrit from 52 to 59 before death. Blood-volume estimations carried out on three of the patients showed that the total volume of red cells was always within normal limits and that diminution of plasma volume accounted both for the haemoconcentration and for the fall in total blood volume. The presence of haemoconcentration differentiates burn shock very clearly from shock secondary to haemorrhage; and it is hoped to publish evidence that haemorrhagic shock, where the deficient blood volume is primarily due to loss of red cells, does not respond to plasma infusion in the same way as shock due to burns.

It was found that, while infusion of half-strength plasma was always followed by dilution of circulating blood, fourfold serum failed to produce haemodilution in two cases out of three. Even with plasma the haemodilution was smaller than might have been anticipated from the greatly improved general state of the patient; it would seem, in fact, that moderate degrees of haemoconcentration are compatible with the absence of any clinical shock. The repeated blood-volume estimations in the fifth case illustrate the mechanism of haemodilution after plasma infusion. The total plasma volume rose from 1.5 to 3 litres, while the red cell volume remained practically unchanged, the increase in total blood volume being 1.4 litres; at the same time the haematocrit fell from 66 to 51.

Biochemical Findings

(i) *Plasma Proteins.*—For convenience of reference the initial values for protein, chloride, bicarbonate, and urea in six severely burnt patients have been grouped in Table I.

TABLE I.—*Blood Chemistry after Burns*

Case	Plasma Chloride mg. per 100 c.cm.	Plasma Bicarbonate mg. per 100 c.cm.	Blood Urea mg. per 100 c.cm.	Plasma Protein gm. per 100 c.cm.	Albumin : Globulin Ratio
1	726	44	26	4.2	2 : 1
2	580	56	36	8.0	1.5 : 1
3	560	53	30	7.3	5 : 1
4	641	34	32	5.8	2.2 : 1
5	569	50	65	5.0	2.3 : 1
6	—	—	30	5.5	1.4 : 1

Less reduction was encountered in the concentration of plasma protein than might have been anticipated from a knowledge of the large quantities of protein-containing exudate from and into burnt areas. In fact, the average plasma-protein value in six cases before any form of treatment had been given was 6 grammes per 100 c.cm. It would appear that plasma-protein concentration is quite well maintained, either by rapid mobilization of tissue-protein stores, whose labile character has been emphasized by Whipple (1938), or by loss of fluid in amounts comparable to the loss of protein, leading to reduction in plasma volume. The figures for the third case show that very large amounts of protein may be lost: at 5.45 a.m. on June 10, 1940, the plasma-protein concentration was 7.3 grammes per 100 c.cm. and the plasma volume 2.5 litres, so that the total plasma protein was 182 grammes; at 11.30 a.m. on the same day the corresponding figures were 7.5 grammes per 100 c.cm. and 2.6 litres, giving a total plasma protein of 195 grammes, an increase of 13 grammes. In the interval, however, an infusion of 300 c.cm. of fourfold serum, containing 60 grammes of protein, had been given, so that 47 grammes must have been lost in less than six hours, even if no tissue protein were mobilized in that period. By the next day the total plasma protein had fallen to 120 grammes, indicating the loss of another 75 grammes of protein at least. These figures may be compared with the normal value for this patient of 210 grammes of total plasma protein obtained after recovery. No significant decrease in the ratio of albumin to globulin was observed in these patients.

The giving of plasma or serum did not produce any great rise in plasma-protein concentration; in the cases in which blood-volume figures are available and total plasma protein can be calculated it was found that a rise in total plasma protein occurred. The extent of this increase could not, however, be predicted from the quantity of protein administered, presumably since it was modified on the one hand by continuing exudation and on the other by the influx of protein from the liver and other tissues into the blood.

(ii) *Bicarbonate, Chloride, and Urea.*—The bicarbonate content of the plasma is diminished in burns, as in other forms of shock, and the lowering of alkali reserve is greater in the more severely shocked patients. Previous chloride estimations in patients with burn shock have been done as a rule on whole blood, and Underhill *et al.* (1923) found reduction in blood chlorides; since the chloride content of red cells is less than that of plasma by almost 100 mg. per 100 c.cm. this reduction in whole-blood chloride can be largely accounted for by haemoconcentration; and in fact Underhill found that the chloride reduction was roughly proportional to the degree of haemoconcentration present. Our patients showed rather high values for plasma chloride, a finding which is in good accord with the reduction in plasma bicarbonate.

The urea content of the blood was normal in five out of six cases on the initial estimation, although a moderate rise in blood urea was sometimes encountered a day or two after the burn; this finding, which has been confirmed in a number of cases of shock from other causes, is a surprising one when it is remembered how frequently azotaemia is associated with even mild degrees of acidosis or dehydration, both of which may be present in burns.

(iii) *Sodium and Potassium.*—Serum sodium and potassium were determined in four cases. The changes in serum sodium after burns have been intensively studied by Wilson and Stewart (1939); in agreement with these authors we found a fall in serum sodium during the stage of shock; the sodium value rose after serum infusion. Our values for serum potassium are of interest in relation to the importance attached by Scudder (1940) to an elevation of serum potassium in all forms of shock. Serum was obtained in our cases by centrifugation within five minutes of venepuncture, and under these conditions the rise in potassium was slight, being never greater than 10 mg. per 100 c.cm. above the normal value. In two cases in which it was possible to observe the serum potassium before and after protein infusion a fall of 3 to 4 mg. in the serum potassium was found.

The Nature of Burn Shock

The haematological and biochemical investigations which have been carried out in cases of burns make it clear that loss of protein and fluid from the circulating blood stream is a prime cause of the shock syndrome in these patients. Controversy has centred on the question whether local loss of fluid and protein in the burnt area is sufficient to account for diminution in circulating blood volume or whether there is also a generalized increase in capillary permeability, with a tendency to widespread loss of fluid into the tissues. While oedema of the limbs is uncommon in shocked patients, perhaps owing to peripheral vasoconstriction, visceral oedema is a common finding, both in experimental animals (Moon, 1938) and in man (Robb-Smith, 1940). Moon postulates that a capillary dilating substance is formed by damaged tissues, although such a substance has never been isolated or demonstrated in the blood of shocked animals. It seems unnecessary to assume the existence of such a factor, since anoxia in itself causes an increase in capillary permeability (Landis, 1928), and a failure of circulation initiated by fluid loss from the burned areas may be increased progressively by leakage of fluid from the capillaries in other parts of the body which are suffering from stagnation anoxia; in conformity with

this hypothesis is the very striking improvement which may be produced in shocked patients by administering oxygen in high concentration with the B.L.B. mask.

Therapeutic Considerations

The general management and surgical treatment of burns have been reviewed by Cohen (1940). With the exception of the use of routine prophylactic chemotherapy we are in substantial agreement with the scheme of treatment there proposed; and our therapeutic suggestions may be limited to the indications for the use and dosage of plasma or serum.

The period during which patients with burns are in jeopardy from shock extends for some forty-eight or seventy-two hours after the incident. There is at present no proved method of diminishing the pathologically increased capillary permeability, which is the fundamental lesion in shock, although pituitrin and the administration of high concentrations of oxygen are indicated for controlled trial. In the absence of a direct method of improving capillary tone the object of treatment is to maintain an adequate blood volume by replacing the protein and fluid which are lost; in this way circulation can be maintained during the forty-eight- or seventy-two-hour danger period, in which exudation from the burnt area is at its height; the maintenance of an adequate circulation prevents the onset of irreversible tissue changes such as are seen when fluid therapy is inadequate or when the burn is so extensive that the loss of fluid outruns any possible replacement. Since shock is an ingravescent condition plasma should be given early, before general circulatory changes have had time to occur.

The translation of the indications for plasma or serum therapy into clinical idiom is rendered difficult by the inconstant relation between fall in blood volume (which constitutes the fundamental indication for plasma) and the usual clinical data such as pulse rate and blood pressure. Rise in pulse rate above 100 and any fall in blood pressure are indeed indications for plasma transfusion, but cases occur which show no alteration in blood pressure and no great rise in pulse rate, yet are in the early stages of progressive shock: by the time alterations have occurred in blood pressure such cases may be unable to recover, or may require very large doses of plasma where a smaller dose given an hour or two earlier would have sufficed. Routine blood-volume estimations are impracticable, but in patients who have not bled the determination of haemoglobin, one of the simplest laboratory procedures, offers a very sensitive measure of the amount of plasma lost. For approximate calculation of the deficit in plasma volume one is justified in assuming a haemoglobin percentage of 100 and a blood volume of 5 litres, of which 3 litres are plasma. The increased haemoglobin value observed bears the same ratio to the initial value as the initial value for blood volume does to the new blood volume, since the red cell volume remains unchanged. This may be expressed in the formula $\frac{Hb_2}{Hb_1}=\frac{BV_1}{BV_2}$ or, substituting the assumed values, $\frac{Hb_2}{100}=\frac{5}{(5-x)}$, where Hb_2 is the observed haemoglobin value after the burn, and x is the amount of plasma lost. It will be seen that x can easily be calculated if Hb_2 is known. For convenience Table II gives the approximate deficit of plasma volume corresponding to a series of haemoglobin readings. This method can of course be applied only to patients who were in good health before the burn and in whom the injury causing the burn did not also cause significant haemorrhage; but this category comprises the majority of patients with burns. In addition to the deficit of plasma volume as calculated from the haemoglobin one must consider the extent and severity of the burn before deciding to give plasma; this is necessary because a similar degree of haemoconcentration may be present in a comparatively trivial injury (e.g., Case 6), as in the earlier stages of a dangerous burn. Patients who show a haemoconcentration of over 10%, and in whom the burn involves an area equal to one limb or greater, should be given plasma or serum.

TABLE II.—*Estimation of Plasma Volume Reduction. (The figures in the last three columns are derived from the haemoglobin reading by the method described in the text.)*

Haemoglobin % (Haldane)	Blood Volume (litres)	Plasma Volume (litres)	Estimated Deficit in Plasma Volume (c.cm.)
100	5.0	3.0	—
105	4.75	2.75	250
110	4.55	2.55	450
115	4.35	2.35	650
120	4.15	2.15	850
125	4.0	2.0	1,000
130	3.85	1.85	1,150
135	3.7	1.7	1,300
140	3.55	1.55	1,450
145	3.45	1.45	1,550
150	3.35	1.35	1,650

As Best and Solandt (1940) have emphasized, there is no essential difference between plasma and serum protein in therapeutics; the difference in our series was one of concentration, since we used serum in fourfold strength and plasma at half-strength. Fourfold serum was introduced into the treatment of shock in the hope that it would bring about a return of fluid from the tissues into the blood stream; we have found no evidence that this occurs in burn shock, and on two occasions injections of concentrated serum had to be supplemented after an interval of some hours by citrated plasma before blood volume was restored; the third patient to whom concentrated serum was given died.

In patients with shock an overdose of fluid is as dangerous as an underdose, though the danger is less with protein-containing infusions than with saline solutions, which should not be used. To avoid this danger it is recommended that the approximate deficit in the plasma volume should be calculated on admission by the method described above, and that the same amount of citrated plasma should be given intravenously while preparations are made for cleansing the burned area; there is little risk in giving fluid rapidly to a patient whose blood volume is acutely depleted. After the cleansing of the burns the haemoglobin estimation should be repeated, and if haemoconcentration exceeds 10% a further appropriate amount of plasma should be given. In the average case of extensive burns this means that a litre of plasma should be given pre-operatively or during the cleansing of the burns, while after operation as much as two or three litres may be required; such an amount is best given by cannula at a drip speed of about 100 per minute. Even if the plasma deficit has been calculated it is necessary to examine the patient every few hours during the second infusion to prevent overdosage with fluid. Engorgement of the neck veins is a valuable sign that too much fluid is being given; the appearance of peripheral oedema is of less value as it is a late sign, and visceral oedema may be extensive in the complete absence of "clinical oedema." In any event, the intravenous drip should not be maintained for more than one or at most two days after the burn, since by then it should be possible to give enough fluid by mouth and also the danger of death from shock is diminishing, while the risk of pneumonia, which is aggravated by pulmonary oedema, is becoming more imminent. Patients with burns, like other patients with distorted water metabolism, should have records kept of the fluid

balance, and the amount of fluid given by mouth should be regulated on this basis.

Protocols of Patients

Case 1.—This patient, a man aged 24, was admitted after being caught in the flame of a burning aeroplane. Tannafax had already been applied to the burns, which were of third degree and involved both legs from ankle to thigh, both hands and wrists, and the scalp from the lower jaw level upwards. The burns were cleaned and coagulated with 20% tannic acid. Shock was severe, the veins being very collapsed. Plasma infusion in this case was combined with the prophylactic use of sulphapyridine, 1 ampoule four-hourly. Recovery from the shock syndrome was good, but jaundice became very pronounced, and death occurred on March 17, with a definite terminal pneumonia. This experience confirmed some previous scruples as to the value of sulphapyridine when used prophylactically in cases of this type, and it was decided that in future chemotherapy should not be begun until there were definite signs of pneumonia.

Date and Time	Pulse	B.P.	Haematocrit	Haemoglobin	R.B.C. (millions)	Protein	Albumin	Chloride	Bicarbonate	Urea
12/3/40 17.30	110	90/60	57	116	6.2	4.2	2.8	726	44	26
Plasma infusion (1,600 c.cm.) from 17.30, 12/3/40, to 5.00, 13/3/40										
23.30	140	116/94								
13/3/30 22.00	110	—	52	114	6.2	3.8	—	600	52	30
14/3/40	130			122						
15/3/40	144	110/90	41	98	5.3	—	—	548	56	35
16/3/40	142									
17/3/40	150	100/70								

Died, with respiration rate of 50 and signs of consolidation. Permission for necropsy could not be obtained.

Case 2 (Time of burn, 12/4/40, 6 a.m.).—A woman aged 57 was admitted on April 12, 1940, after sustaining burns of second degree involving the whole of the back and the right arm, when her nightdress caught fire. On first admission, four hours after the burn, she was little shocked, with B.P. 135/90 and pulse 106. After cleaning and spraying the burns with tannic acid 5% the systolic B.P. was 90. The stage of shock lasted for seventy-two hours.

Date and Time	Pulse	B.P.	Haematocrit	Haemoglobin	R.B.C. (millions)	Protein	Albumin	Chloride	Bicarbonate	Urea
12/4/40										
10.00	106	135/90								
11.30	100	90/—								
14.00	80	—	54	106	5.5	8.0	4.8	580	56	36
18.00	90	80/40								
150 c.cm. of four times normal serum from 18.15 to 18.30										
18.30	90	120/80								
500 c.cm. of citrated plasma from 18.30 to 20.30										
22.00	80	110/70								
13/4/40										
10.00	100	115/70	51	104	5.7	6.5	4.0	534	63	37
19.00	96	118/80								
14/4/40	100	110/70		99						
15/4/40	104	130/85	50	89	5.7	7.0	4.0	513	59	54
16/4/40	102	—		86	4.4					
23/4/40	80		43	84	5.2	5.0	3.0	586	62	52
7/5/40			39	84	4.8	5.8	3.8	550	50	20

Case 3.—This patient, a pilot officer aged 24, was admitted after a crash, with second- and third-degree burns involving the whole of the face and both hands, and about half the skin surface of the legs. He had had morphine grain 3/4 and 1.5 pints of normal saline at the R.A.F. station. He was severely shocked, with a pulse rate of 150 and B.P. 96/58. While the burns were being cleaned and tanned 300 c.cm. of four times normal serum was injected by syringe. This was followed by considerable improvement in the general signs of shock; on the next day a further infusion of citrated plasma had to be given. By the following day the signs of severe shock had disappeared. Healing of the burns was good, and the patient was discharged.

Date and Time	Pulse	B.P.	Haemoglobin	R.B.C. (millions)	Urea	Protein	Albumin	Chloride	Bicarbonate	Plasma Vol.	Haematocrit	Blood Vol.	Na	K
10/6/40														
5.00	156	96/58												
5.45	156	—	100	5.0	30	7.3	6.0	560	53	2.5	47.5	4.8	270	21
300 c.cm. of four times normal serum from 6.00 to 6.30														
7.00	100	90/50												
7.30	96	100/60												
11.30	90	—	112	5.7	33	7.5	6.0	—	—	2.6	46.5	4.9	305	18
15.00	78	150/90												
11/6/40														
11.00	100	114/70	122	6.0	33	6.0	4.4	595	54	2.0	57	4.6	—	—
1,500 c.cm. of citrated plasma from 14.00 to 15.00														
15.00	100	150/80												
23.00	86													
12/6/40														
10.00	80	140/90	114	5.6	38	6.0	4.4	—	—	—	53	—	282	—
13/6/40	92	140/94	118	5.3	41	6.0	4.8	560	52	—	51	—	—	26
14/6/40	—	—	100	5.0	35	5.5	4.4	570	56	—	44	—		
18/6/40	86	155/90	98	5.0	24	5.8	4.2	—	—	—	33	—	298	15
22/7/40	86	118/80	102	5.4	24	7.8	4.6	540	67	2.7	45	4.9	307	16

Case 4 (Time of burn, 25/6/40, 7 p.m.).—This patient, a woman aged 61, was admitted on June 25, 1940, from a mental institution, where she had set fire to her clothes, sustaining second-degree burns of more than half of the body surface. She was very shocked on admission, and the infusion of 300 c.cm. of four times normal serum produced only a transient improvement. The burns were cleaned and tanned, but she died on the morning after admission. Blood pressure could not be taken.

Date and Time	Haemoglobin	R.B.C. (millions)	Haematocrit	Urea	Chloride	Bicarbonate	Protein	Albumin	Pulse
25/6/40									
22.00 ..	106	—	52	32	—	34	5.8	4.0	130
26/6/40									
10.00 ..	132	6.8	59	60	641	43	6.3	4.2	135
13.00 Died									

At necropsy, carried out by Dr. A. H. T. Robb-Smith, extensive oedema of the subcutaneous tissue in relation to the burnt areas was seen. A small pleural effusion was present on both sides, and the lungs were congested and oedematous. A fresh preparation showed occasional fat embolism in the pulmonary vessels. A few endocardial haemorrhages were found. The liver was congested and fatty, the spleen pale and contracted. The suprarenal cortex contained abundant lipoid.

Case 5.—A man aged 25 was admitted, after an aeroplane crash, with third-degree burns of the face and hands. First dressing with tannafax was left on for five days, after which the hands were treated by coagulation with gentian violet and extension on a racket splint. On admission the patient was moderately shocked, with some collapse of superficial veins. Definite lowering of blood pressure persisted for forty-eight hours and oedema in relation to the burnt areas for seven days.

Date and Time	Pulse	B.P.	Haemoglobin	R.B.C. (millions)	Urea	Protein	Albumin	Chloride	Bicarbonate	Plasma Vol.	Haematocrit	Blood Vol.	K	Na
4/7/40														
16.30	90	106/92	136	6.5	65	5.0	4.0	569	50	1.5	66	4.5	27	—
Plasma infusion (2,000 c.cm.) from 22.00, 4/7/40, to 13.00, 5/7/40														
5/7/40														
16.30	90	104/74	116	5.3	40	5.5	4.2	571	49	3.0	51	5.9	23	337
6/7/40	100	130/80	116	5.1	39	5.5	3.8	532	50	—	47	—	23	—
7/7/40	95	134/60												
8/7/40	80	120/80	100	4.3	35	5.0	3.0	572	50	—	44	—	—	—
5/9/40	80	—	92	4.5	25	6.0	3.6	572	50	2.7	41	4.6	—	—
9/10/40													17	356

Case 6 (Time of burn, 23/9/40, 9 a.m.).—This patient, a woman aged 27, was admitted with a scald of the face and right arm, of the first degree. Although she was in considerable pain, shock was very slight, and she was discharged on September 24, the day after admission. She is of interest in showing slight but definite haemoconcentration in the absence of other blood changes, and of any increase in pulse rate or decline in blood pressure.

Date and Time	Haemoglobin	R.B.C. (millions)	Plasma Vol.	Haematocrit	Blood Vol.	Urea	Protein	Albumin	Chloride	Bicarbonate	K	Pulse	B.P.
23/9/40 10.00	108	5.6	3.0	47	5.6	32	6.5	4.5	589	61	21	80	124/82
24/9/40 10.00	98	—	—	43	—	44	6.5	4.4	597	56	—	80	—

Case 7 (Time of burn, 5/10/40, 5 a.m.).—This patient, a woman aged 58, was admitted on October 4, 1940, with second-degree burns of the lower part of the back, and the back and sides of both legs. These were cleaned and tanned with 10% tannic acid and silver nitrate. She was treated by the infusion of 1,500 c.cm. of citrated plasma and by oxygen administration by the B.L.B. mask. The condition of shock lasted for less than forty-eight hours. She left hospital against advice on October 9.

Date and Time	Pulse	B.P.	Haemoglobin	R.B.C. (millions)	Haematocrit	Urea	Protein	Albumin	K
5/10/40 11.00	96	—	140	7.5	65	30	5.5	—	24
13.00 to 23.00 : 1,500 c.cm. of citrated plasma (B.P. at 23.00, 140/80)									
7/10/40 10.00	110	140/80	116	5.5	55	80	6.5	3.8	22
8/10/40	90		114						

Case 8.—This patient, a man aged 26, was admitted three hours after an aeroplane crash in which he sustained severe burns of head, trunk, and limbs covering more than half of the body surface. Very shocked on admission, pale and sweating. 1,200 c.cm. of citrated plasma was run in rapidly, after venespasm had been overcome by heat and pressure with a Higginson syringe. The burns were cleaned and coagulated in theatre, and the plasma drip was continued till 3,500 c.cm. in all had been given. He had then lost all signs of shock, but his temperature was 104° F. and his pulse 134, and he appeared to be suffering from toxaemia. Calcium gluconate was given in a dose of 4 grammes intramuscularly. On the next day his condition was worse, and his respirations had increased to 40, although there were no abnormal signs in the chest. He vomited bilious fluid several times before death. Owing to the extent of the arm injuries blood pressure and volume could not be estimated, and blood was obtained from the femoral vein.

Date and Time	Pulse	Haemoglobin	Haematocrit	R.B.C. (millions)	Protein	Albumin	Chloride	Bicarbonate	Urea	Sodium	Potassium	Blood Sugar (mg. per 100 c.cm.)	Bilirubin
21/10/40 23.30	186	132											
Plasma infusion from 23.45, 21/10/40, to 12.00, 22/10/40 (3,500 c.cm.)													
22/10/40 9.00	132	92		5.2	(W.B.C. 64,000)								
11.00	130	98	42	5.0	4.5	4.2	656	52	120	280	10.2	—	—
23/10/40 10.00	136	102	45	5.0	4.8	2.8	592	49	132	—	—	141	0.2

(The plasma in both venous samples contained haemoglobin and methaemoglobin, but no methaemalbumin. The urine contained haemoglobin and red cells ; the patient's group was O (IV), while the plasma used was A (II), so that the patient's cells would not be agglutinated from this cause. Urobilin was also present in the urine, but no excess of urobilinogen.)

Necropsy was done by Dr. Robb-Smith at 16.30, 23/10/40, five hours after death. Oedema was limited to the neighbourhood of the burnt areas, which showed no evidence of sepsis. A few petechial haemorrhages were present on the lining of serous cavities, but there were no endocardial haemorrhages such as are found in patients dying of shock. The heart muscle was good, although there was right-sided dilatation. The lungs showed haemorrhagic areas, such as might be produced by concussion injury ; some fat embolism was present. The spleen was large and congested, and the liver capillaries contained masses of packed red cells. The kidneys had "haemoglobin casts" in the tubules. The necropsy findings were not those to be expected had death been caused by shock or excessive fluid administration ; the organic changes were compatible with death being caused by a combination of renal damage and traumatic injury to the lung.

NOTE.—Throughout the protocols haemoglobin results are expressed as percentage Haldane, red cells as millions per c.mm., haematocrit as per cent., blood and plasma volume as litres, urea as mg. per 100 c.cm. of blood, protein as grammes per 100 c.cm. of plasma, and sodium, potassium, chloride, and bicarbonate as mg. per 100 c.cm. of plasma.

Summary

Clinical, haematological, and biochemical observations have been made on eight patients with burns or scalds, of whom seven were treated with plasma or serum. In patients before treatment the main findings were:

(*a*) Increasing shock, with the classical symptomatology.

(*b*) Progressive haemoconcentration.

(*c*) A fall in the plasma volume, and therefore in the total amount of plasma protein, although the protein concentration was often quite high. Evidence was obtained that amounts of plasma protein equivalent to a quarter of the total plasma protein might be lost in a few hours.

(*d*) Plasma chloride was high and bicarbonate low, while blood urea was normal.

(*e*) Serum sodium was low and there was a slight rise in serum potassium.

Great clinical improvement followed infusion of dilute plasma and was accompanied by a rise in plasma volume in those cases in which serial observations of blood volume were possible. The results with four times normal serum were much less favourable ; one of the three patients treated with concentrated serum died, and the other two required an infusion of dilute plasma later.

A scheme is put forward for calculating the approximate dosage of plasma required, using the observed haemoglobin figure as a basis.

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The Argentine Republic has 11,244 practitioners, or one doctor to every 1,485 inhabitants, of whom 5,077 live in Buenos Aires, with the result that some districts are badly provided with doctors.