THE

TREATMENT OF BURNS

AND SCALDS

A.W. WILKINSON, M.B., Ch.B.Ed.

From the Royal Hospital for Sick Children, Edinburgh, and

the Royal Infirmary, Edinburgh.

C O N T E N T S.

INTRODUCTION.

This paper records certain investigations on a series of 116 cases of burns and scalds treated by coagulation with one per cent. gentian violet and ten per cent. silver nitrate, this method having been instituted to obtain a control series of cases for comparison with others treated with varying strengths of tannic acid. Of the cases in the present series, 114 were treated during the past two years in the Royal Hospital for Sick Children, Edinburgh, and 2 further cases treated in the Royal Infirmary, Edinburgh, have also been included. Over 30 of the cases were treated personally by the writer and the majority of the others were observed by him for part or the whole of their stay in hospital.

The aspects considered in this report include the clinical course of the burned patient, the circumstances of the injury, first aid treatment, rate of healing, the duration of stay in hospital and the incidence of infection. In addition, previous observations on shock, acute toxaemia and blood chemistry have been supplemented. An attempt has also been made to assess the value of certain therapeutic agents used in the treatment of the cases, and selected case histories are included to illustrate features of the clinical course and treatment.

An important aim of the paper is to stress the necessity for educating the public in the matters of (1) preventing the occurrence of burns and scalds in young children; and (2) the appropriate measures to be taken when such injuries do occur.

CLINICAL COURSE.

The clinical course of burns and scalds has recently been described in detail by Wilson, Macgregor and Stewart (1938), and it is intended to present here only a brief summary, following the classification into five stages introduced by Wilson:

1. Initial shock
2. Secondary shock
3. Acute toxaemia
4. Septic toxaemia
5. Healing.

1. Initial Shock.

The state of shock which follows immediately upon the injury resembles closely that produced by trauma of other kinds, and almost certainly arises in a similar manner. At present it is believed that a severe vasomotor upset occurs as a result of nervous impulses from the injured area. Initial shock in burns is usually transient and often mild; occasionally it is severe and prolonged; rarely it is progressive and passes into the stage of secondary shock. An arbitrary limit of 2 hours from the time of injury was chosen by Wilson et al. (1938) as the dividing line between initial and secondary shock.

2. Secondary Shock.

This stage may begin at any time after 2 hours and may last for a varying period. It may be present on admission or may develop subsequently, most often during or after local treatment. Secondary shock is a state of circulatory failure which is initiated by the loss of plasma from the circulating blood. The plasma escapes primarily from the blood stream through the walls of damaged capillaries in the area affected by heat, on to the raw surfaces and into the tissue spaces of the burned area. As a result of this loss /

loss of plasma, the volume of circulating blood is reduced, but for a time this change is masked by compensatory mechanisms; when these are no longer effective the blood pressure falls. The initial reduction in blood volume is due to the loss of plasma at the site of injury. Corpuscular concentration results, which leads to increased viscosity of the blood and slowing of the rate of blood flow, particularly in the capillaries, with a secondary loss of plasma due to stasis and increased outward flow of plasma through the walls of capillaries rendered more permeable by anoxaemia. This is a progressive phenomenon, and unless interrupted by efficient treatment, the resulting anoxaemia causes irreparable damage to the central nervous system and other vital structures.

Developing shock is indicated by a rising diastolic pressure and a decreasing pulse pressure, rising pulse rate and haemoconcentration. When shock is established, circulatory failure is already well advanced. The systolic blood pressure and pulse pressure are low; the respirations may be rapid, shallow and irregular; corpuscular concentration may be marked and the rectal temperature sub-normal. The patient may be mentally alert, apathetic or comatose; he is usually pale and intensely thirsty; vomiting may be frequent, particularly in the more severe degrees of shock. Apart from the constant lowering of the systolic blood pressure and pulse pressure, many of the changes associated with severe secondary shock vary greatly.

3. Acute Toxaemia.

The onset of acute toxaemia may be at any time between 8 and 60 hours, but is most common between 12 and 24 hours after injury. Symptoms and signs are at first usually mild; restlessness is often the first feature and may pass into delirium or coma. The rectal temperature and pulse rate rise, there is cyanotic or greyish pallor, altered blood is vomited, jaundice may be /

be marked, and little urine, containing much albumin, is passed; the blood pressure falls shortly before death. Haemoconcentration and bacterial infection are not features of acute toxaemia, which is more common in association with superficial than with deep injuries and has no constant relationship to the extent of the burn. The etiology of acute toxaemia is still undecided; infection with the haemolytic streptococcus (Aldrich, 1933), haemoconcentration, hypochloraemia and acidosis (Underhill, 1923) have not been generally accepted as the underlying factors. The evidence available suggests that this condition is an intoxication caused by the absorption into the blood stream of the products of autolysis of damaged cells in the burned area (Wilson et al., 1937). The toxic effects are most marked in the cardio-vascular system and in the liver.

4. Septic Toxaemia.

Before the introduction of coagulation treatment, this stage, like that of acute toxaemia, was of greater frequency; early infection of the healing surfaces was severe and almost invariable. The incidence of infection is related to the depth of the injury, being infrequent in superficial injuries but usually occurring in deep injuries where there is sloughing. Such infection may be limited or controlled by chemotherapy in the majority of cases.

Septic toxaemia begins usually at the end of the second week when liquefaction and separation of the sloughs is taking place in deeply burned areas. Infection spreads in the exudate and involves the sloughing tissue. The toxaemia lasts until the sloughs have separated and the infection has been controlled, the duration varying in different cases. The severity of the toxaemia is related to the extent of the area of deep injury, and the frequency with which the dressings are changed, each change causing considerable pain and /

and resulting in loss of serum and blood from the raw surface. Rapid loss of weight, anaemia and mental and physical exhaustion result.

5. Healing.

In superficial injuries healing is rapid and is complete when the tan separates, usually after 10 to 18 days. In deep injuries the stage of healing may be very prolonged, lasting for several months. The systemic disturbances of septic toxaemia subside as infection is controlled, epithelialisation of the granulating areas gradually takes place, its rate varying with different factors, being increased by environmental changes and by skin grafting.

TREATMENT.

The primary objective in the treatment of a burn is to save life, the secondary objectives are the promotion of healing, prevention of deformities and the restoration of function. Treatment by coagulation of the raw surface is directed towards avoiding or minimising the three main dangers to life: secondary shock, acute toxaemia and infection. The secondary objectives also are attained by the coagulation method, but in deeper injuries additional measures may be required.

The method of treatment used in the present series will be described for each of the stages of the clinical course.

1. Initial Shock.

The decision to admit the patient to hospital is made after rough estimates of the extent of the injury and the patient's condition have been made, without full exposure of the injury. On admission to the ward, the patient is put to bed wrapped in blankets surrounded by hot bottles. A heating cage is then placed over the patient; a small cage may be used for young /

young children, but for older children and adults a large cage enclosing the whole bed is preferable. No attempt is made to undress or bath the patient who should not be interfered with in any way until improvement in the general condition is evident. The removal of clothing or dressings and the inspection of the burned area should be delayed until the patient has been anaesthetised. The pulse rate, rectal temperature and respiration rates are taken and are recorded thereafter every hour. Blood pressure estimations are repeated every hour for the first 12 hours; for this purpose the nursing staff should be instructed in the procedure. Fluid by mouth should be withheld if an anaesthetic is shortly to be administered. Heroin (for children) or morphine sulphate (for adults) is given by subcutaneous injection.

TABLE 1.

Dosage of Heroin for Children up to 12 Years of Age (R.H.S.C.E.).

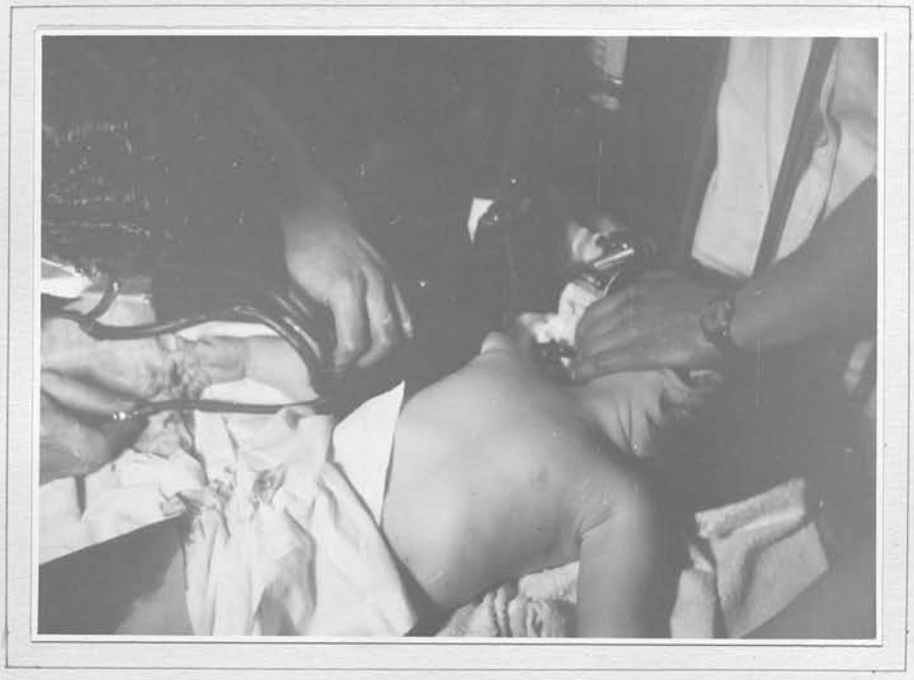
Age	Heroin in grains
Birth - 2 years	1/96
2 - 5 "	1/48
5 - 8 "	1/24
8 - 12 "	1/12

2. Secondary Shock.

Secondary shock must be prevented or minimised as far as possible. When it is present, immediate and energetic measures are essential. Treatment is directed towards restoration of the volume of circulating blood to a normal level and its maintenance at that level, and, as far as is possible, to prevention of further loss of fluid from the circulating blood.

General /

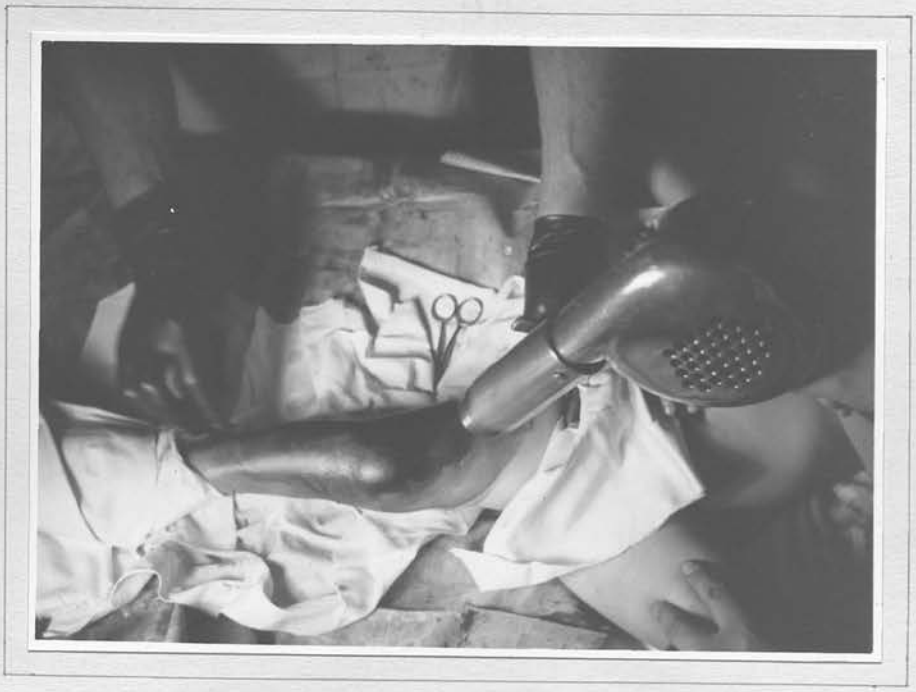
A series of photographs to show the steps in the local treatment of a scald of the thigh and leg, in a child of 11 months.



1. Measuring the blood pressure

2. Cleaning scald with saline

3. Application of 1 per cent. gentian violet

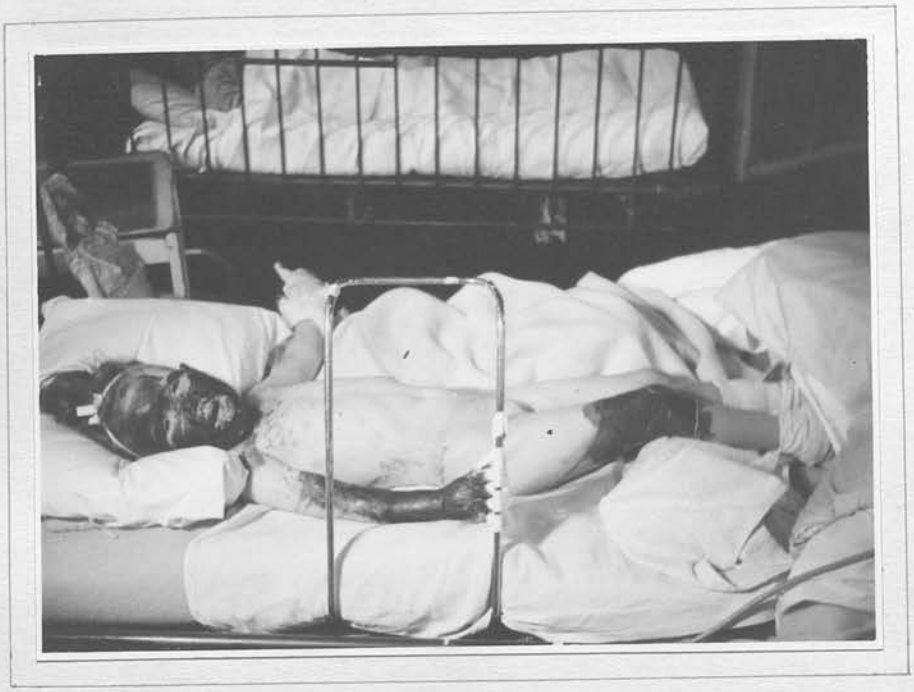


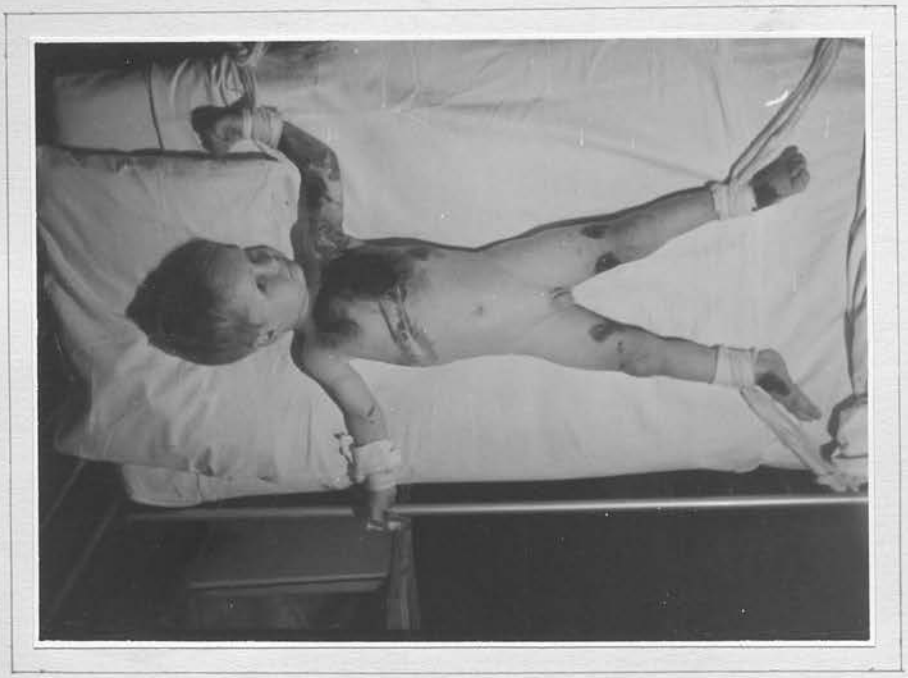
4. Drying with hair-drier after application of the coagulant

General Treatment. The patient is kept warm and quiet. If necessary (as indicated by blood pressure estimations), gum saline, plasma saline, plasma or reconstituted serum is given in sufficient quantities to restore the blood pressure to a safe level (see Discussion, p. 32). This level must be maintained by continuation of the infusion at a slower rate, if necessary during and after local treatment. Injections of heroin or morphine sulphate may be repeated if required, but not sooner than one hour after the previous dose.

Local Treatment. When the general condition of the patient is satisfactory, an injection of atropine sulphate is given. The local treatment may be carried out in the ward if a large cage is available, otherwise it is better to move the patient to an operating theatre or a well heated room, where full aseptic precautions may be observed. Anaesthesia is induced with nitrous oxide and oxygen, ether is only added subsequently if required; cyanosis must be avoided. The patient is then undressed and the burn is exposed. If the areas are large, they should be dealt with in sections. Hairy parts involved in the injury are shaved, including a margin of healthy skin for 2 inches round the burn. The burned area is cleansed with gauze swabs soaked in sterile isotonic saline warmed to body temperature. (The cleansing with saline is not an attempt to secure asepsis (though frequently this is achieved) but is directed towards removing gross contamination, charred clothing, oil, etc., and the dead skin. Particular attention must be paid to the edges of the areas where overhanging margins of skin may be left, forming sites in which infection readily occurs. All these tags of skin must be removed. This is more difficult in scalds than in burns as in the former there is frequently a marginal zone where the injury is not clearly defined but shades off gradually from second to first degree.) Hard rubbing or the use of scrubbing brushes is unnecessary and harmful. The cleansed surface is dried

by /





Methods of Fixation of Scalded Children.

by dabbing gently with gauze. One per cent. gentian violet is dabbed on with pledgets of cotton wool soaked in the solution, the whole raw area being covered together with a marginal belt of healthy skin 2 inches in width; the area is then dried by hot air from a hair dryer. Ten per cent. silver nitrate is similarly dabbed on and dried. The deep colour of the surface which has been painted with gentian violet takes on a greyish tinge immediately the silver nitrate is applied; this change indicates the formation of silver compounds. The application of gentian violet and silver nitrate may be repeated if oozing occurs after the coagulated surface has been dried.

After coagulation has been completed, the coagulated surface is exposed without dressings to hot air under a heat cage. The limbs of children should be controlled by bandages or splints even if not injured. The position of the patient must be so arranged that a minimum of the coagulum is in contact with skin surfaces or bedding.

Heroin or morphine sulphate should be given before the patient has completely recovered from anaesthesia, and should be repeated frequently, but not more often than every 2 hours. Pain is usually absent after coagulation. Thirst is always intense and drinks of sweetened tea or fruit juice should be given ad libitum. The position of the patient is often such that drinking is difficult; this can be overcome by the use of straws or rubber tubing.

3. Acute Toxaemia.

The treatment of acute toxaemia is primarily preventative by means of coagulation. A certain proportion of the damaged cells are fixed by the coagulant, and autolysis of these cells is thereby prevented. Coagulation, however, does not fix all the damaged cells, but the absorption of the products of autolysis of the unfixed cells is retarded or prevented by the immobilisation imposed by the coagulum. This preventative treatment is frequently, though

not /

not invariably, successful. Glucose is given in large quantities in drinks or by intravenous infusion in an attempt to protect the liver, the cells of which exhibit damage in fatal cases of toxaemia.

Established toxaemia is treated by the slow intravenous administration of glucose or glucose saline solution, and by the injection of suprarenal cortical hormone. This hormone, either in the form of an extract of the whole gland, or as desoxycorticosterone acetate, must be given repeatedly in large doses for a prolonged period if benefit is to be obtained. Chloral and bromide, phenobarbitone, heroin or morphine sulphate are given to control the restlessness.

4. Sepsis.

Preventative treatment begins with the cleansing of the burn during local treatment. The maintenance of a dry impermeable coagulum is necessary to prevent bacterial invasion, and is effected by exposure to hot air circulating in the heat cage. The use of any dressing or clothing over the coagulum or the contact of coagulum with bedding invariably results in softening of the tan; bacteria may gain entry through such soft areas or under the margins of the separating coagulum. The edges are painted twice daily with one per cent. gentian violet solution. Cracking of the coagulum is prevented by splints or fixation to side-bars; this is of great importance in young children who wriggle if not firmly fixed. Soiling of the tan with urine and faeces is particularly liable to occur in children and in aged subjects, and often leads to infection. Late blistering may develop during the first 24 hours and is a source of infection; the surrounding skin is therefore inspected at 12 and 24 hours for such blisters which are treated by cleansing and coagulation. Such preventative measures must be applied in superficial injuries until healing is complete, and in deep injuries until separation of the slough has been /

been effected by the formation of granulation tissue.

Burns which are infected at the time of admission may be treated by cleansing with saline followed by coagulation of the raw surface, provided that the injury occurred not more than 72 hours previously. Older infected burns of small extent are best treated by cleansing and the application of hypertonic saline dressings.

Infection occurring beneath the coagulum may necessitate the removal of the coagulum. This removal should be confined to the infected part and is best achieved by the application of hypertonic saline dressings. Isotonic or hypotonic dressings or fomentations should not be applied for fear of producing an acute toxaemia. The appearance of thick yellow pus at the margin of the tan is not an indication for removal of the tan, as such pus may contain no organisms and is usually a suspension of cells and leucocytes in the exudate of the granulating surface, and not of infective origin.

The severe and formerly fatal infection by the haemolytic streptococcus can now be controlled by the administration of full doses of sulphonamide.

Transfusion of fresh blood may be necessary and is of marked benefit in severe infections.

5. Healing.

In the great majority of superficial injuries healing is complete when the coagulum separates; any small untreated areas which may remain should be treated with a dressing such as gauze soaked in an aqueous solution of proflavine (1/1000) or with an ointment, for example zinc oxide and castor oil ointment. In deep injuries when the slough has finally separated, a granulating surface remains which may or may not be infected. Infection may be largely controlled by applications such as of gauze soaked in hypertonic saline. Healing will occur from the margins of healthy skin but may become slow or arrested; much improvement can be obtained from variation of the agents used in the dressings. When extensive granulating areas remain, skin grafting is necessary.

INVESTIGATIONS.

Total Incidence of Burns.

Below is a table to show the number of children suffering from burns and scalds seen for the first time in the Surgical Out-Patient Department of the Royal Hospital for Sick Children, Edinburgh, during four consecutive months for various years.

TABLE 2.

Month	1934-5	1935-6	1936-7	1937-8	1938-9	1939-40
November 30 days	12	16	10	12	9	22
December 31 "	11	15	12	12	20	24
January 31 "	12	9	16	12	11	29
February 28 "	16	5	16	15	11	22
Totals: 120	51	45	54	51	51	97

It will be noticed that the number up to 1939 was fairly constant at an average of 50.4, although the number in any month is subject to considerable variation; no explanation has yet been found for the latter feature. In the same four months of the first winter of the present war, the incidence has been nearly doubled.

Age Incidence.

In 101 cases occurring in children, there was the following distribution according to age:

TABLE /

TABLE 3.

Age in Years	Number of Cases	Per cent.		
0 - 1	10	9.1		
1 - 2	41	40.6	67%	76%
2 - 3	27	26.7		
3 - 4	5	4.9		
4 - 5	12	11.8		
6 - 12	6	5.9		
	101	99.0		

Forty per cent. of injuries occurred between the ages of 1 and 2 years, 67 per cent. between 1 and 3 years and 76 per cent. between birth and 3 years of age.

The Causal Agent.

Information was available in the majority of cases as to the agents producing the injury, and these are listed below:

BURNS:

Clothes set on fire	5 cases
Unguarded fire	4 "
Explosion of boiler	2 "
Hot plate	2 "
Gas ring	2 "
Celluloid comb on fire in hand	1 case
	16

SCALDS: /

SCALDS:

Cup of tea upset	27	cases
Kettle upset	18	"
Pot of tea upset	10	"
Saucepan boiling water	6	"
" boiling stew	5	"
" boiling cabbage water	2	"
" boiling potatoes	1	"
Hot fat from frying pan	3	"
Cup of shaving water upset	1	"
Plate of soup	1	"
Pan of Sister Laura's food	1	"
Sat in bucket of boiling water	1	"
Sat in basin of boiling water	1	"
Fall into tub of boiling water	2	"
Fall into copper full of boiling water	1	"
Cup of coffee and cocoa, 1 each	2	"
Burst rubber hot water bottle	2	"
Feet scalded while being bathed for inflammation	1	"
Baby born into bucket of boiling water	1	"
	86	

Of all these injuries, 2 only could be ascribed to an unavoidable accident (boiler explosion). The remainder were the result of lack of parental supervision or of carelessness.

The age of the patient and the causal agent of injuries involving 50 per cent. or more of the body surface have been extracted:

TABLE 4.

Age	Causal Agent	Result
2 4/12	Child pulled over kettle of boiling water	Recovered
2 11/12	" " " " " " "	
2 2/12	" " " saucepan " "	
3 1/12	Clothing on fire	Died
4 4/12	" " "	Died
7 1/12	" " "	Recovered
4	Scalds due to boiler bursting	Died

Types of Undesirable First Aid Treatment Commonly Employed.

In 85 cases enquiry was made as to the nature of treatment before the patient was brought to hospital. The evidence obtained is as follows:

Agent Used	Number of Cases	
Olive oil	30	35%
Picric acid	13	14%
Tannic acid jellies of various makes	12	13%
Flavine preparations	5	6%
Carron oil	4	4.5%
Flour	6	7.5%
Unspecified ointments	3	
Baking soda	2	
Castor oil - externally	1	
" " - internally	1	
Syrup of figs - internally	2	
Vaseline	1	
Soap	1	
White of an egg	1	
Sulphur powder	1	
Talcum powder	1	
Butter	1	
Total:	85	

Dressings of various kinds were employed over the above medicaments, such as gauze, lint, towelling and cloths of various kinds, applied dry or soaked with water. In many cases two or three of the above agents had been used together in the same areas. The result of such efforts was often to produce a caked mass on the affected part, consisting of flour, oil and plasma, contaminated with dirt and adherent to clothing or dressings.

Time Interval Between Injury and Admission to Hospital.

The time interval was investigated in 105 cases and the results are shown in the following table.

TABLE /

TABLE 5.

Time between injury and admission	Number of Cases		Percentage
0 - 1 hours	41		39
1 - 2 "	28		26.6
2 - 3 "	11		10
3 - 4 "	7	93	
4 - 5 "	5		
5 - 6 "	1		
			88.6
8 hours	1		
12 "	2		
18 "	1		
20 "	2	12	11.4
24 "	1		
1 day	2		
2 days	2		
3 "	1		

Extent of Injury.

Reliable evidence of the extent of injury was available in 109 cases, as follows:

TABLE 6.

Extent per cent. of body surface	Number of cases	Percentage of whole
0 - 10	55	49
10 - 20	32	29.3
20 - 30	9	8.2
30 - 40	3	2.7
40 - 50	2	1.9
Over 50	8	7.3
	109	98.3

Seventy-eight /

Seventy-eight per cent. of the injuries involved 20 per cent. or less of the body surface and 49 per cent. were of 10 per cent. or less. The number of very extensive injuries was small and represents the usual incidence of such injuries. The patients considered above do not comprise the total admissions during the 16 months' period covered by this survey, but only those treated by coagulation with gentian violet and silver nitrate. In addition, a large number of patients were treated with jellies as out-patients and by various methods as in-patients.

The Incidence of Shock at the Time of Admission to Hospital.

The diagnosis of initial shock on admission was limited to those patients whose systolic blood pressure was below 100 mm. mercury within 2 hours of injury.

TABLE 7.

Patients Admitted Within 2 Hours of Injury.

Type of Injury	Duration in hours	Age in years	Extent per cent.	Blood pressure mm. mercury	
Scald	1	9/12	2½	84/40	
Scald	½	2 6/12	20	90/76	
Scald	¾	2 4/12	20	95/70	
Scald	½	1 3/12	20	85/55	*
Burn	1	3 1/12	50	84/50	
Burn	1½	4 4/12	50	?/?	
Scald	½	2 4/12	50	92/54	
Burn	½	1 3/12	5	112/60	
Scald	1½	2	10	120/90	
Scald	1	7/12	10	118/70	
Scald	½	1 4/12	15	116/60	
Scald	¾	1 9/12	15	105/75	**
Burn	½	2 10/12	20	124/90	
Scald	1½	1 2/12	20	104/70	
Scald	1¾	4	20	100/70	
Scald	1	4 10/12	20	106/80	
Scald	2	2 1/12	35	115/65	
Burn	1	7 6/12	55	104/86	

* Systolic blood pressure below 100 mm. Hg.
** Systolic blood pressure above 100 mm. Hg.

The blood pressure of 26 patients was estimated on admission. Of 18 admitted within 2 hours of injury, 7 had a systolic blood pressure of less than 100 mm. mercury and were considered to be suffering from initial shock. Of these 7 cases, 3 were admitted within half an hour, 3 within one hour, and the other within one and a half hours of injury. In 3 cases the extent of the injury was over 50 per cent., in 3 it was 20 per cent. and in one case it was $2\frac{1}{2}$ per cent. of the body surface. In 5 cases the systolic pressure was below 90 mm. mercury; in one case no record could be made. In one case the pressure continued to fall and the patient did not respond to treatment and died of secondary shock. In 2 cases initial shock passed into secondary shock, but later these responded to treatment.

Rate of Healing and Duration of Stay in Hospital.

In a number of uncomplicated superficial scalds it was found that the coagulum began to separate in the marginal areas after 8 days, and that separation was complete after 10 to 14 days, leaving a healed surface. In a consecutive series of 39 unselected cases treated to completion in two wards, the average time taken for separation of the margins of the coagulum was 10.7 days, and for complete separation 19.2 days; the average duration of stay in hospital was 20 days. The time taken for separation of the coagulum and for healing to occur was related directly to the depth of the initial injury. Separation of the coagulum was rapid when the tan was thin, pliable and remained dry, and in areas of superficial injury, especially on the face and scalp. Separation was slow on the palmar surface of the fingers and hand and in areas of deep injury. In superficial injuries of the face the areas were frequently healed in less than 8 days.

Blood /

Technique of puncture of femoral vein

Blood Chemistry.

Biochemical investigations were carried out in 29 cases. The scope of the investigations was limited by the amount of blood which could be obtained in individual cases. Blood was withdrawn from the superior sagittal sinus in infants or from the arm veins of older children. Where neither of these sources was available, blind puncture of the femoral vein was carried out.

The technique of puncture of the femoral vein does not appear to be widely known, and as this is a valuable and often the only means of obtaining blood, the method employed will be described. It can be used equally well in adults and in children, but is less often necessary in the former. The needle used should be of wide bore $1\frac{1}{2}$ inches in length, with a short bevel. The landmarks are the femoral artery and the pubic tubercle which are felt for with the index and third fingers of one hand. The needle is then entered upwards and medially, midway between these two points, just below the level of the pubic crest, and passed in until it strikes the bone; it has then transfixed the vein against the pectineus fascia and muscle, and the bone. The needle is withdrawn slightly while the plunger of the attached syringe is pulled back and blood usually flows into the syringe; if it does not, further attempts should be made to enter the vein. Failure to obtain blood is infrequent, and with practice sufficient for the usual estimations can be drawn. Precautions should be taken to ensure that the syringe and needle are quite dry before use; this is best done by autoclaving the needle in a test tube plugged with cotton wool; the syringe need not be sterile. Even with this precaution haemolysis occurs frequently; without it is invariable.

The following estimations were carried out on blood: sodium, potassium, chloride, non-protein nitrogen, urea nitrogen, sugar, CO_2 combining power, albumin, globulin, fibrinogen, cholesterol, iron, haemoglobin. At first attention /

attention was directed chiefly towards the values for serum sodium and potassium, but it was soon found difficult to obtain sufficient unhaemolysed blood for both sodium and potassium, since haemolysis occurs rapidly in blood from burns and scalds even with the most rigorous precautions. Where sufficient blood was obtained, other estimations were made, but sodium was given prior consideration. Blister fluid from 8 patients was examined for its sodium content, and the cerebro-spinal fluid from 4 patients for sodium potassium, total protein and chloride. In addition, in 2 cases estimations of cell volume and haemoglobin were made.

Results:

Forty-three estimations of serum sodium were made, the results being grouped as below.

TABLE 8.

Mg. per cent.	No. of Cases	Grouping
Under 300	12	Very low
300 - 325	24	Low
325 - 330	1	Normal
Over 330	6	High

There was a fairly constant low value in blood taken soon after injury and before local treatment was carried out, ranging from 252 to 319 mg. per cent. In 2 cases the sodium was high, 335 mg. at $1\frac{1}{4}$ hours (Case 17) and 333 mg. per cent. at $2\frac{3}{4}$ hours (Case 12), both being extensive injuries which involved over 50 per cent. of the body surface, one a burn, the other a scald. In one there was severe shock on admission, in the other no shock was evident. In Case 2 a gradual rise of the sodium level was found during the 50 hours after injury. This was present in other cases though not so clearly demonstrated. /

demonstrated. In two cases (8 and 9) this rise was found but later a fall occurred, associated in each patient with the presence of infection of the healing areas and the loss of large quantities of fluid from the surface. The influence of sodium derived from intravenous saline in causing a rise of the serum sodium cannot be excluded in every case, but in Case 2, where no intravenous fluid was given, the rise was early and maintained. The body seems, in certain individuals, to be able to restore the level of the sodium completely, whilst in others such restoration does not occur spontaneously or to a lesser degree. The factors controlling this mechanism are not known.

Eight estimations of blister fluid sodium were made; the values obtained corresponded closely with, but were always a few milligrams higher than, those obtained from the serum of the same patient at the same time. The difference varied in individuals but was not connected with extent or duration or the existence of shock.

TABLE 9.

Comparison of the Levels of Sodium in Serum and Blister Fluid in 8 Cases.

Case reference No.	Blister fluid mg. per cent.	Blood serum mg.per cent.	Blood pressure	Time after injury, in hours	Extent per cent.
3.5935	318	314	-	1	5
3.5939	308	307	-	1	15
3.6043	300	297	115/65	4	20
3.6046	334	316	105/75	1½	15
3.6059	319	315	90/76	2½	20
3.6060	321	319	106/80	1½	20
3.6272	315	312	115/80	1	25
3.6269	334	333	120/95	2¾	40

In the 7 cases in which the sodium level was below normal, the fall in blood sodium averaged 14-19 mg. per cent. (taking 325-330 as the normal level), 311 being the average sodium level in these cases. The blister fluid sodium

was /

was on the average 5.3 mg. per cent. above that of the serum sodium. No connection was found between the extent of the blisters (and thus of the amount of blister fluid formed and blister fluid sodium lost) and the level of sodium in the blood serum.

The normal level for serum potassium was taken as being between 18 and 22 mg. per cent. Thirty-five estimations were made, the results being grouped as follows:

TABLE 10.

Mg. per cent.	No. of Cases	Grouping
Under 18	2	Low
18 - 22	13	Normal
Over 22	20	High

The rise in potassium was much less constant than the fall in sodium which was not always associated with a rise in potassium. Usually a rise of potassium above the limits of normal was found only when there was a severe depression of the sodium level.

In 2 cases serum calcium was estimated. Taking the normal level as being 10 mg. per cent., in both these cases depression of serum calcium was present. Subsequent observations in each case showed that in one a fall in calcium accompanied a fall in sodium, while in the other a rise in calcium accompanied a rise in sodium.

The serum chloride fell when the level was uncomplicated by the giving of saline.

There was usually a rise of urea nitrogen and non-protein nitrogen following injury, but this change was neither constant nor closely related to the other changes.

Insufficient /

Insufficient estimations of other constituents of the blood were made to justify any conclusions being drawn.

Mortality.

Of the 116 consecutive patients treated by coagulation with gentian violet and silver nitrate, 8 died, a mortality rate of 6.9 per cent. The several causes of death were as follows:

Secondary shock	5 patients
Doubtful toxaemia	1 patient
Broncho-pneumonia	1 "
B. coli septicaemia	1 "

Of 7 patients whose injury involved over 50 per cent. of the body surface, 3 died, death in 2 cases being due to secondary shock. The 3 other patients who died of secondary shock had injuries of 30-40 per cent. of the body surface. A baby aged 8 days died of B. coli septicaemia; the scalded areas were uninfected and little systemic disturbance had arisen directly from the scald. In the case classified as doubtful toxaemia, most of the clinical and some of the pathological evidence was in favour of a diagnosis of toxaemia, but certain typical features were not present and the cause of death is not certain. The child who died of broncho-pneumonia was suffering from the effects of severe systemic disturbances in addition to having an injury which involved over 50 per cent. of the body surface.

DISCUSSION. /

DISCUSSION.

Writing in Edinburgh about a hundred years ago, de Quincey stated that, "Three thousand children per annum - that is, three hundred thousand per century; that is (omitting Sundays), about ten every day - pass to heaven through flames in this very island of Great Britain." In a footnote he adds that these deaths are "chiefly through the carelessness of parents." No information has been obtained as to the incidence of serious burns at that time, but it seems reasonable to suppose that the mortality rate then was probably higher than at the present time. It is interesting that the chief factor in the production of the injuries is the same as that alleged in this paper.

Burns are generally most common during the winter months, although in examining a large series of cases it was found that the monthly total of admissions might reach a very high level in any month without much relation to season. Because the incidence in the last 2 months of 1939 and the first 2 months of 1940 was abnormally high, it was considered worth while to examine the incidence in these four months during the previous 5 years. It was found that the total admissions in the 1939-40 period were nearly double the average for the previous 5 similar periods. This is a significant finding and may be explained by the abnormal conditions which existed during those months; the closing of schools, cinemas and other places of entertainment, the lack of alternative recreational facilities, the black-out, the occupation of parents with various forms of war work and their mental preoccupation and unrest resulted in a reduction in the amount and quality of supervision of children. This perhaps supports the contention that the "problem of burns and scalds" is a social one in so far as it concerns children.

The /

The annual reports of the Registrar-General for Scotland show that for the period of 8 years, 1931-1938 inclusive, the total deaths from accidental burns (excluding conflagrations) of persons of both sexes of all ages was 1977, an average annual mortality rate of 247. The average incidence in each month of the year for this period is shown below, and the incidence obtained from the Royal Hospital for Sick Children, Edinburgh, is in accord with these figures.

Average Incidence of Death from Burns per Month.

Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
28	26	23.3	22	16.6	17.7	14.6	15	14.75	20.6	23.1	25	247

Further reference to the annual reports of the Registrar-General for Scotland confirms the importance of the age incidence as is shown in the table below:

TABLE 11.

Year	Sex	Total all ages	0 - 1	1 - 5	0 - 5	5 - 10	10 - 15
1934	m.	105) 253	11	56	120	3	1
	f.	148)	9	44		11	5
1935	m.	90) 241	9	46	101	2	3
	f.	151)	5	41		5	8
1936	m.	85) 140	6	33	90	5	-
	f.	155)	7	44		4	4
1937	m.	101) 257	8	42	101	7	-
	f.	156)	7	44		8	8
1938	m.	87) 212	3	39	81	4	2
	f.	125)	5	34		4	2
Totals:		1203	70	423	493	53	33

0 - 5 years of age 493 deaths = 40.9 per cent.of all deaths at all ages.

The age incidence in the present series is even more significant: 40 per cent. of the patients were between 1 and 2 years of age; 67 per cent. between 1 and 3; and 74 per cent. were under 6. That only 5.9 per cent. of the children were over 6 years of age seems to indicate that special factors are concerned, and this is supported by an examination of the agents producing the injury. This age incidence is similar to that found by Denniston (1939) in a series of 398 cases treated at the Royal Hospital for Sick Children, Glasgow, in which the average age was $2\frac{1}{2}$ years. Of all deaths from accidental burns in Scotland, 40.9 per cent. occur in children under the age of 5 years. There is no significant difference in the sex incidence in the first 5 years of life; this occurs only after this period when it is marked, since the total deaths are approximately in the proportion of 3 females to 2 males.

Causal Agent.

Age and causal agent are closely connected. The commonest cause in this series was the cup of tea seized by the child and pulled over himself or upset by a clumsy parent or relative who was nursing the child at the table. The common injury produced in this way is a scald of the face, neck, chest, arms and hands. A remarkable number of such injuries were brought to hospital between the hours of 10 p.m. and midnight.

A certain number of these injuries were due to a combination of the inquisitive and acquisitive instincts of the young child and his lack of previous painful experience, but in all there was evidence of lack of supervision of the child by parents or responsible elders. This lack of supervision even occurred in small families living in uncrowded circumstances, though such cases are infrequent. The majority of the children came from large families living in the poorer districts of Edinburgh or the surrounding towns /

towns and villages. Several children in some families have been injured at different times and some children on more than one occasion. The affected child in a proportion of cases had been left in the charge of another child, often little older. Unguarded fires, though illegal, are a commonplace, kettles and saucepans are boiled on gas rings on the floor or table, the handles within easy reach of the avid hands of the young child, who, just able to walk and climb, may attempt, with disastrous results, to imitate the actions of his elders.

No estimate has been made of the cost of treating these injuries; it must be high and might be avoided almost entirely by educative propaganda. In an effort to reduce the incidence of these injuries, the following suggestions are made:

1. Intensive propaganda by posters exhibited in public conveyances, hospital and clinic waiting rooms, and cinemas. Such propaganda should give information as to the number of burns and scalds per year in Edinburgh, with mortality rates and a description with diagrams of the way in which burning accidents occur, how they may be prevented, what first aid treatment should be applied and where further treatment may be obtained.

2. Closer co-operation between hospital staffs and the Social Services. All burning and scalding accidents involving children should be notified to a statutory or voluntary organisation dealing with the welfare of children, and the house concerned should be visited within 48 hours by a suitably trained agent who would investigate the circumstances of the accident, give advice regarding prevention of a recurrence, and make a report to the organisation.

First Aid Treatment.

The evidence obtained regarding first aid treatment indicates that there is widespread ignorance of the principles involved and lack of judgment and knowledge /

knowledge in the application of the treatment usually employed. The agents which were used in the present series of cases are in aqueous solution and must be brought into direct contact with the cells exposed on the raw surface before a coagulating action can be exerted. Any application which interferes with this direct contact must be removed during the initial cleansing of the wound, thus involving extra trauma and expenditure of time. This objection applies particularly to oily and greasy substances which have no coagulant action and are unlikely to reduce shock to an appreciable extent. Severe pain is usually caused by the application of such oils and greases, and occasionally was said to have been worse than the pain of the actual injury.

Flour, baking soda, etc., had been applied in many instances, baking soda because it was thought to be soothing, and flour to exclude the air (oils were also used for this reason). In this connection certain observations have been made which discredit to some extent the widespread belief that air must be excluded as completely as possible from a burned area. Exposure of a burn to very cold air (draughts) or very hot air (as in front of a fire) undoubtedly causes pain. It has been repeatedly observed that during the final stage of local treatment, while the drying of the tan is being completed (the patient being only lightly anaesthetised) a current of hot air induces protective struggling especially when it is directed to the hands, face or upper half of the abdomen, while the cold air current is less irritating or has no irritant effect. Patients will lie comfortably and painlessly under a shock cage with an untreated burned surface exposed to warm air, but rapid changes in air temperature may induce pain. Patients were occasionally seen who had been treated by the application of a dry cloth alone. Allowing for variation in extent, depth and duration, such patients were invariably in a better condition than those who had received more active but less desirable treatment by oil, flour, jellies, etc. The use of wet cloths lowers the

body /

body temperature and increases the severity of shock.

While recognising the difficult circumstances under which treatment was carried out, the state of the injuries to which various types of coagulant jellies had been applied was creditable neither to the person who applied treatment nor to the jelly applied, which, when used properly on a suitable case, is a valuable and efficient agent. In all cases so treated it was found that areas of variable extent were left untreated, frequently elevated skin was not removed and blisters were not snipped or had arisen since treatment. It was quite obvious on many occasions that the person applying the jelly had no idea of the large amount necessary to treat the area properly or of the correct way in which to apply the dressing, and that the supply of jelly was inadequate. The use of such jellies by those not conversant with the indications for use and the inherent dangers is to be discouraged.

The administration of a purgative in drastic doses is an "old wife's remedy" designed "to put away the shock" and is used more frequently in burns and in other conditions than has been found in this series.

Any burning or scalding accident is very upsetting to both the victim and the onlookers be they related to him or not. At once all is confusion, and it is not surprising that this results in such primitive methods of first aid treatment. To recommend the removal of the patient to hospital without first applying some form of local treatment will almost certainly appear to be neglectful, but it cannot be too deeply stressed that treatment of the kinds described above is meddlesome and unnecessary and always inefficiently done, whether applied in ignorance or as a placebo.

The following instructions in first aid treatment are suggested. The guiding principles are two: (1) to treat shock already present; and (2) to prevent, as far as possible, further shock. Both can be followed by interfering as little as possible with the affected surface and by refraining from applying to it anything which will be removed later only with difficulty.

Remove /

Remove the patient from the burning room, flames, steam or hot water.

Extinguish blazing or smouldering clothes by wrapping the patient in rug, blanket, coat or jacket and then rolling him on the ground; do not allow the patient to run about the room or out of doors. Always lay down the patient with the flames uppermost so that further areas of clothing are not ignited. Smothering the flames is much better than throwing a bucket of water over the person.

Lay flat on the floor in a warm place out of draughts, or put to bed if this can be done easily. Do not lay in front of a fire as this often causes pain.

Send for a doctor or a police ambulance.

Cover burned or scalded area with a clean dry cloth, sheet, or towel.

Wrap in blanket, rug, or coat, then lay protected hot water bottles outside, and cover bottles and first blanket with another blanket, etc.

Hot sweet tea may be given, but no solid food as an anaesthetic will usually be given later.

DO NOT attempt to remove clothing as this causes pain; a dressing has to be applied in the place of the clothing, and will itself have to be removed at least once for examination by the doctor. Every interference of this kind causes pain, and therefore increases shock and is dangerous and unnecessary.

DO NOT apply to the affected area antiseptics or medicaments such as baking powder, soda or flour, oil or any oily dressing, picric acid.

DO NOT give a dose of castor oil to reduce shock, it causes vomiting and probably produces further shock.

DO NOT apply burn jelly or cream to severe burns or scalds, it induces a false and dangerous feeling of security.

Remember /

Remember that nearly everything applied to the area in the first aid treatment will have to be removed later, and the more easily the first aid dressing can be removed in its entirety, the more satisfactory such treatment is likely to be.

Time Interval.

The interval between the time of injury and the time at which the patient arrived at hospital varied very greatly, being influenced by such factors as transport difficulties (long distances and adverse weather conditions), and delay due to indecision and ill-advised home treatment by parents, relatives, neighbours or the medical practitioner. That 65 per cent. of the cases admitted arrived within 2 hours and 88 per cent. within 6 hours of injury is encouraging. Burns are as liable to infection as other wounds, and it would seem to be as important in their case as in other types of wound that active treatment should be instituted as soon after injury as possible, so that contamination may be removed before bacterial invasion begins. Less than 11 per cent. were admitted after 12 hours and of these in a certain number delay was due to attempted treatment at home by the parents or patient's doctor. The majority of such attempts failed because of systemic disturbances, the severity of which had alarmed parents and doctor alike, or because of infection of the burned area. It is emphasised that particularly in the case of extensive injuries, the sooner the patient is admitted to hospital the sooner efficient treatment can be instituted.

Extent of Injury.

The extent of the injury was in nearly half of the cases less than 10 per cent. of the body surface, and therefore what is usually considered to be a minor injury. Extent is not the only criterion on which the decision

regarding admission was made, consideration being given also to the site of the injury, age of the child and to a certain extent the home conditions. In connection with the age of the child, it must be remembered that the surface area weight ratio in the child is greater than that of the adult and decreases approximately as the weight increases. Thus an apparently minor lesion in a child is relatively more serious than a similar percentage extent in an adult, since the early systemic disturbances are usually related fairly closely to the extent of the injury. It is difficult to compare the incidence of injuries of varying extent found in this series with previous cases, since in many of these the data are not complete. However, in the opinion of surgeons who have treated such cases for many years, more injuries of small extent are treated as in-patients now than previously. There is no direct evidence that the number of injuries is increasing, but many more are receiving hospital treatment, the increase being in injuries of small extent. It was formerly stated (Fraser, 1926) that burns of a third of the body surface in adults, or one-eighth of the body surface in children, were invariably fatal. With the present methods, it is possible to hold out a reasonable chance of survival in injuries of up to 50 per cent. of the body surface of children; cases have been quoted in which survival was obtained in burns and scalds involving up to 60 per cent. More extensive injuries than this are unlikely to survive since there is a limit to the amount of the damage which can be tolerated, which appears to be in the region of 70 per cent., but may depend to a certain extent on the site.

Shock.

The information regarding the incidence of initial shock is too slight to permit wide conclusions to be drawn. It seems to indicate, however, that the condition is more frequent than has been suspected. Wilson et al. (1938) stated /

stated that it was rare. Of 26 cases in the present series in which blood pressure records had been made on admission, initial shock was present in 7; in many others shock was stated to be present on admission, but in the absence of blood pressure records these have been disregarded. Wilson et al. (1938) found initial shock in 5 out of 35 cases, in 2 of which death ensued without improvement; such a sequel occurred once only in the 7 cases of the present series. Occasionally initial shock was conspicuously absent as in the case of a boy of $7\frac{1}{2}$ (Case 8) admitted one hour after burns of 55 per cent. of the body surface, with a blood pressure of 104/86. Here initial shock had passed off and secondary shock developed later after local treatment, and was severe and prolonged. It is evident, therefore, that lack of evidence of depression of the blood pressure on admission is no reason for assuming that such a fall will not occur. There is some evidence that initial shock may be more severe in injuries of large extent, but such a connection is not invariable. No evidence was obtained to support the belief that shock is more severe when the affected areas are situated over the abdomen, thorax or skull.

Certain aspects of the treatment of the various stages of the clinical course merit discussion in addition to the description already given. Initial shock has been discussed in some detail already. Secondary shock occurred either before, during or after local treatment; where it was energetically and efficiently treated the response was usually satisfactory, but in certain cases which have been quoted treatment was either tardy or insufficient and in some cases death ensued. An examination was made of the data obtained from the patients suffering from severe secondary shock and certain conclusions have been drawn. Injuries which involve less than 10 per cent. of the body surface are unlikely to result in severe secondary shock, provided that due attention is paid to the provision of warmth and relief from pain, and that anaesthesia and local treatment are not prolonged; even in very small burns

in /

in young children shock may be severe if local treatment is not expeditiously carried out.

Secondary shock is most severe in extensive superficial injuries, but it cannot be emphasised too often that it is a most treacherous state, particularly in young children, and that constant watchfulness must be exercised over all patients, but especially over those whose injuries are of more than 20 per cent. of the body surface. From the evidence presented, it is obvious that in severe shock the combination of coagulation and intravenous infusion of gum saline or plasma saline may be the only means of preventing death. It has been clearly shown that coagulation alone may be insufficient, and evidence obtained from subsequent cases has shown that infusion of gum saline or plasma saline alone is similarly ineffective. The feature of secondary shock which is most susceptible to accurate estimation as a criterion of the severity of the shock is the lowered blood pressure (for the reason that the personal and experimental errors are considerably less than in the case of haemoglobin estimation and red blood corpuscle counts); the blood pressure level is probably also a more direct indication of the severity of shock.

In a particular case it may be difficult to decide when an intravenous infusion is necessary; on the basis of the present series of cases certain guiding principles may be stated. Frequent estimations of the blood pressure are necessary for the diagnosis of the shocked state; the blood pressure levels may be classed as follows:

TABLE 12.

Systolic blood pressure, mm. Hg	Degree of Shock
Below 60	Condition critical
60 - 80	Very severe shock
80 - 100	Severe shock
100 - 120	Mild shock
Above 120	Shock absent

In general it may be stated that patients with injuries of less than 20 per cent. of the body surface and a systolic blood pressure of 100 mm. mercury or higher do not require intravenous infusion. Where the systolic pressure is between 80 and 100 mm. mercury, the decision is modified according to the response to warmth and sedatives and the extent of the injury. Patients whose systolic pressure is below 80 mm. mercury should receive intravenous infusion if a substantial rise has not occurred within an hour. All patients with injuries of more than 40 per cent. of the body surface should receive intravenous therapy before local treatment, irrespective of their blood pressure, since a fall in blood pressure is inevitable during or after treatment. Where the pressure is below 60 mm. mercury, an intravenous infusion must be given immediately. A rise in diastolic pressure, which is associated with a slight rise or no change in the systolic pressure, is to be regarded with suspicion since it usually heralds a fall in both systolic and diastolic levels.

The amount of gum saline or plasma saline to be given varies according to the initial systolic blood pressure, the extent of the injury and the age, weight and body surface of the patient. The best practical guide to the amount required is that volume of fluid which will restore the systolic pressure to at least 100 mm. mercury and will maintain the pressure above this level. It is of value to be able to estimate the amount of fluid which may be required for a particular patient. One way of estimating this amount is on the basis of the calculated blood volume of the patient.

According to Best and Taylor (1937) the whole blood is about 1/11 and the plasma 1/20 of the total body weight, that is about 9 and 5 per cent. respectively. Expressed as volumes, the whole blood is about 90 c.c. per kilogram of body weight and the plasma about 50 c.c. per kilogram, therefore a man of 70 kilos. body weight has a blood volume of 6300 c.c. Rowntree (1929) has /

has shown that the blood volume is related to the surface area of the body, being about 3300 c.c. per square metre. The total blood volume per square metre is higher in males, but the plasma volumes in the two sexes are nearly equal. The body surface is greater in proportion to the body weight in children than in adults. Children have a greater blood volume in proportion to their body weight, but a smaller volume in relation to surface area than adults; they have also a greater surface area in relation to weight than adults. From the above facts it is evident that injuries which affect the body surface such as burns will produce a much more profound systemic effect on children than on adults, and this effect will be more marked in smaller subjects.

A table is appended in which is shown for each year of age up to 12 the average body weight, the calculated total blood volume, the plasma volume, the estimated infusion (that amount of fluid which will be required to restore the blood pressure to a level above 100 mm. mercury and maintain it at that level), and the amount of fluid usually given (this latter is the amount of fluid which can be tolerated when infused at a fast rate and is based on the amount of fluid usually given to children of these ages, for example as a single blood transfusion). These volumes of fluid are intended for use in the cases of gravely shocked children only. The figures are somewhat arbitrary but have the advantage of being based on well accepted principles. The volumes have been checked by estimations of blood and plasma volume made by finding the body surface area from the nomogram of du Bois (1924), and have been found to be accurate.

TABLE /

TABLE 13.

Age in years	Weight lb.	Weight Kg.	Whole blood in c.c.	Plasma in c.c.	Estimated infusion in c.c.	Amount usually given in c.c.	Amount usually given in oz.
½	16	7	630	350	180	135-150	4½-5
1	20	8.75	790	440	220	150	5
1½	22	10	900	500	250	180	6
2	26	11.75	1060	560	280	210	7
3	30	13.5	1200	670	330	210	7
4	34	15.3	1380	780	390	225-240	7½-8
5	40	18.2	1640	910	450	240	8
6	45	20.4	1840	1020	500	255	8½
7	49	22.5	2030	1110	550	255	8½
8	53	24	2160	1200	600	255	8½
9	59	28	2520	1400	700	270	9
10	65	30	2700	1500	750	285	9½
11	71	32	2880	1600	800	300	10
12	80	36	3240	1800	900	300	10

Plasma saline composed of equal volumes of plasma from Group I citrated blood and 0.9 per cent. saline was used in 3 cases of this series as a substitute for gum saline in the treatment of secondary shock. This plasma saline was obtained from the Edinburgh Blood Transfusion Service, to whom thanks are due. It was prepared by pipetting the supernatant plasma from citrated blood which had been in storage for a period of ten days or more; the plasma was then mixed with an equal volume of 0.9 per cent. saline. This agent has been found to produce an effect comparable to that obtained from the use of gum saline; it has the advantage that apart from the citrate no foreign substance is introduced into the blood stream.

A synthetic preparation having the properties of the active principle of the suprarenal cortex - desoxycorticosterone acetate (D.O.C.A.) - has been used in 9 cases of the series in doses varying from a single injection of 5 mg. to 20 injections of 5 mg. This preparation, which can be obtained in oily (Organon) or watery (Ciba) solution, may be given by intramuscular or intravenous /

intravenous injection. It has largely replaced extracts of the fresh gland in the treatment of severe circulatory failure in secondary shock and toxaemia. Insufficient data are available for a detailed statement regarding the action of this drug, but some conclusions may be mentioned. In a few cases striking benefit followed the administration of the drug, and it is believed that such benefit can be ascribed to its action. Repeated injections are necessary in order that the maximum effect may be obtained; it is recommended that at least 5 mg. should be given at not more than two-hourly intervals (10 mg. at one hour intervals has not been followed by any undesirable effects).

The use of D.O.C.A. was confined to those cases in which severe secondary shock or acute toxaemia was found. The drug was withheld in certain cases until the condition of the patient was such that death appeared inevitable. It was considered that only in this way could its value be demonstrated since by this means spontaneous improvement, independent of the drug, could be excluded. In one such desperately ill child (Case 19), D.O.C.A. appeared to prolong life; in other cases reported D.O.C.A. produced improvement which depended on its continued administration. It now appears legitimate to advise that the drug be given before such extreme degrees of circulatory upset are reached, provided that other measures such as coagulation and gum saline or plasma are not neglected. The blood pressure level and the observation of signs of peripheral circulatory failure in the patient remain the standards by which the need for D.O.C.A. should be assessed.

With regard to anaesthesia, nitrous oxide and oxygen with the occasional addition of a small quantity of ether were the agents used in all cases. Nitrous oxide when combined with sufficient oxygen to prevent cyanosis is the anaesthetic of choice; it is followed by less post-operative shock than any other form of anaesthetic (Clark, 1937). It may be necessary to add a little ether and this is desirable if the required depth of anaesthesia can otherwise

be /

be obtained only with coincident cyanosis. The use of ether alone is followed by a greatly increased loss of serum from the raw surface and by severe shock. Mitchiner (1935) and others recommend that local treatment be carried out under the influence of full doses of morphine, heroin, or tincture of opium; this method has not been employed on any case of this series, and in severely shocked patients it is considered that nitrous oxide anaesthesia is probably superior.

Gentian violet was applied before the silver nitrate because it was found that the dye delineated clearly the exact boundaries of the cleansed raw area and any tags of skin which remained at the margin. A mixture of gentian violet and silver nitrate is unsatisfactory.

Tannic acid was introduced as a coagulant in the treatment of burns by Davidson (1925) because it had the property of precipitating proteins, and he hoped that it would fix any toxic agent which might have been formed in the tissues. A more recent view (Wilson et al., 1938) suggests that the action of tannic acid is to precipitate the proteins of the damaged cells and so reduce greatly the amount of autolytic change which can occur; subsequent toxin formation and absorption is thus minimised. Other workers have suggested that tannic acid exerts its undoubted beneficial action by relieving pain, or preventing or reducing bacterial infection or the loss of fluid from the burned surface. There is no doubt that coagulation reduces the incidence of toxaemia, but tannic acid treatment does not always prevent toxaemia.

It was therefore considered desirable that a series of patients should be treated by an alternative method as a control of the tannic acid method. In view of the undoubted life-saving properties of that method in the treatment of secondary shock in severe injuries, it was thought to be unjustifiable to use the ideal control method of using no coagulant. It was decided that silver nitrate should be employed instead of tannic acid, combined with the same /

same antiseptic as before, namely one per cent. gentian violet, 10 per cent. silver nitrate being known to be an effective coagulant. In practice silver nitrate was found to have certain advantages. Coagulation was as rapid and effective as with 20 per cent. tannic acid, the tan formed was thinner, more pliable, and its edges did not damage the healing surfaces as frequently as was noticed with tannic acid. It has the disadvantage of being rather more expensive. The following table has been prepared to show the incidence of acute toxaemia in three series of patients treated by coagulation with different agents.

TABLE 14.

Series	Coagulant	Total Cases	Cases of acute toxaemia	Died
Wilson, 1929	2.5% tannic acid	98	4	1
Wilson et al.,1938	20% tannic acid	100	4	2
Present series	10% silver nitrate	114	4	1

The incidence of acute toxaemia does not differ appreciably in the three groups of cases. In the present series the fatal case was clinically like an acute toxaemia though the onset was very late, but the post mortem appearances were not those usually found in fatal toxaemia treated by tannic acid. Of the other 3 cases 2 were mild but one was very severe though not fatal. On the present evidence silver nitrate does not appear to have affected the incidence of acute toxaemia though there is a strong impression that the clinical course has been modified.

The prevention of infection has to be considered as an integral part of the whole problem of treatment, but in the initial stages must be subordinated to the treatment of the immediate severe systemic disturbances. The fact

that /

that fatal toxaemia may occur even in relatively small injuries must be remembered. On the basis of the present conceptions of the pathology of secondary shock and acute toxaemia, the use of dressings and baths seems undesirable since by such means the control of shock and toxaemia is impossible. The maintenance of a dry impermeable coagulum is the best method of preventing the access of infection. Infection most commonly occurs in injuries which involve or approach the mouth, nose, anus or the flexures, and when it is not possible to expose the whole coagulum to the air (as in extensive injuries to the whole of the trunk). Some degree of infection usually occurs in extensive deep injuries and here conditions are particularly favourable to the multiplication of organisms. When infection is present on admission within 72 hours of injury, there is no contraindication to local treatment by coagulation after cleansing with saline, and this is usually all that is necessary to control the infection. It is permissible to treat such areas by the application of dressings of hypertonic saline provided that the area is not large and the general condition of the patient is good. When infection occurs beneath the coagulum, it is best treated by the application of hypertonic (6 per cent.) saline soaks, isotonic or hypotonic solution may lead to fatal acute toxaemia by liberating the previously fixed proteins or their autolytic products. Before such applications are made, the presence of infection should be proved by direct films and cultures made from the exudate beneath the coagulum obtained by incising the suspected area of coagulum. The coagulum is removed all too often because thick yellow pus is oozing from beneath the margins; this pus is composed of a suspension of necrotic cells, debris and leucocytes suspended in serum, and rarely contains living organisms.

In a number of cases of extensive injuries sulphanilamide was given prophylactically from the second morning, and the incidence of infection appeared to be slightly reduced. It was decided, however, that since proved infection could /

could usually be brought rapidly under control by adequate dosage of sulphanilamide, prophylaxis which reduced the available margin of dosage should be discontinued.

The extensive granulating surfaces which remain after the separation of the tan from deep injuries almost invariably become infected; control of such infection is difficult but has been attained by chemotherapy combined with frequent changes in the type of dressing used and transfusion of small quantities of fresh blood.

Healing.

The process of healing of a burn or scald depends primarily on the depth of injury, being rapid in injuries involving destruction only of the superficial layers of the skin, and slow in deep injuries where the skin is completely destroyed. In a superficial injury healing occurs by proliferation of the undamaged deeper layers of the skin. In deep injuries the skin loss can be made good only by epithelialisation from the margins or from any remnants of sweat glands and hair follicles which may happen to have been undamaged. This is a slower process, particularly as further delay is necessary while sloughs separate and granulation tissue forms.

The average stay in hospital of 39 patients treated until healing was complete was 20 days. This observation is of importance in relation to the rising number of burned patients admitted for treatment and the reduced accommodation available. The ultimate result is unsatisfactory and healing is delayed in a proportion of patients who are discharged before treatment is completed to receive further attention as out-patients. Other possible means of reducing the duration of stay in hospital depend on changes in policy with regard to the method of treatment employed which should not be lightly undertaken. The primary aim of treatment is to save life, and the coagulation

method /

method is designed to minimise or prevent secondary shock and acute toxaemia, the chief lethal factors. An alternative method which does not satisfy the primary objective is not justified because the stay in hospital is reduced. A wide margin of safety is necessary in any method since secondary shock or acute toxaemia may occur even in minor injuries.

Blood Chemistry.

The investigation of the changes in the blood chemistry was seriously hampered by the difficulty of obtaining sufficient blood for analysis because of the small patients whose veins frequently were impalpable, collapsed and deeply buried in abundant subcutaneous fat. While puncture of the femoral vein has solved this problem to a certain extent, blood from extensively burned patients is often so concentrated that it can hardly be drawn into a syringe and rapid clotting is common; this latter factor interferes particularly with haemoglobin estimations which involve the use of a fine pipette; in addition blood from these patients exhibits a marked tendency to haemolysis which renders invalid estimations of such constituents as sodium and potassium.

In blood withdrawn soon after injury the serum sodium level was usually lowered in burns of moderate or severe extent, but this was by no means invariable. The variations are difficult to explain since very low levels have been found in minor burns and values well above normal with burns of 50 per cent. of the body surface. In two of this latter type of case there was no disturbance of the levels of potassium chloride or non-protein nitrogen. In one of these cases severe shock was present and death followed at 6 hours; in the other shock developed only later and was controlled and the patient recovered.

The sodium level usually falls during the first 24 hours, the time at which the level is lowest appears to be between 12 and 36 hours, though the volume /

volume of evidence obtained is not large. A rise in sodium follows and lasts until a normal level is restored, this restoration occupying a varying period of time. A secondary fall in serum sodium may occur during the healing stage, coinciding with the loss of large quantities of serum from the granulating surfaces.

The sodium of blister fluid was found to be constantly a few milligrams per cent. higher than the serum sodium of blood drawn simultaneously from the same patient. No connection was found between the extent of blistering, the approximate comparative amount of blister fluid and the level of sodium in serum and blister fluid.

It may be said therefore that while a low level of serum sodium is often associated with shock, severe injuries and extensive injuries, this association is not invariable, though when depression of the sodium level occurs this usually coincides with the period during which secondary shock is most marked.

Lowdon et al. (1939) have stated on the basis of experiments on cats under Nembutal anaesthesia, that after scalding, the level of sodium in the serum of arterial blood and in cerebro-spinal fluid steadily declined, and they suggested that the sodium was being lost into the scalded tissues because of the following facts: (1) no significant fall in serum sodium occurred if the circulation to the scalded area was effectively occluded before scalding; (2) the serum of venous blood from scalded skin contained less sodium than did the serum of arterial blood; (3) when the isolated hindquarters of the cat were perfused with heparinised blood, a sharp decline of plasma sodium began about one hour after scalding, and the plasma of outflow blood had a significantly lower sodium content than the plasma of inflow blood. These observations suggest that the sodium is lost into the tissues at or near the site of injury. In order that a fall may occur in any of the constituents of the serum of the circulating blood, it is necessary that one of two mechanisms should /

should act, either loss of a constituent without loss of serum, or loss of whole serum (that is including all constituents) with subsequent replacement of this volume in the blood by a fluid less rich in these constituents than is the lost serum.

A fall in serum sodium is not the only change however; there is usually also a lower chloride level and a rise in serum potassium, non-protein nitrogen and urea. The rise in serum potassium may be accounted for in part at least by the haemolysis which accompanies severe burning injuries. The raised nitrogen level may be a compensatory reaction or may result from other mechanisms. Chloride may be lost into the tissues and at the raw surface. It seems evident that there is not simply a loss of whole plasma after burning, but rather that there is some change in the selective permeability of the cell membrane in the cells of the capillary walls and the other tissues in and around the burned area.

Mortality.

Prior to the introduction of coagulation methods of treatment, the mortality rate in the Sick Children's Hospital for burns and scalds was in the region of 40 per cent.; in a series of 300 cases recorded in 1926 by Fraser, the rate was 38.7 per cent. and death was due to the following causes:

Shock	2½ per cent.
Acute toxaemia	80 " "
Sepsis	15 " "
Causes unconnected with the injury	2½ " "

In a series of 117 cases treated with tannic acid and reported in 1929 by Wilson, the mortality rate was 11.11 per cent., and was due to the following causes:

Shock	30.7 per cent.
Acute toxaemia	33.7 " "
Sepsis	33.7 " "

In /

In the present series of 116 cases treated with gentian violet and silver nitrate, 8 died, a mortality rate of 6.9 per cent., the causes of death being:

Shock	5 cases,	62.5 per cent.
Doubtful acute toxaemia	1 case,	12.5 " "
Broncho-pneumonia	1 "	12.5 " "
B.coli septicaemia	1 "	12.5 " "

The comparative significance of these figures can be better appreciated if they are expressed as the number of deaths from each cause per 100 cases treated by each method:

TABLE 15.

Causes of death	Non-coagulation, per cent.	Tannic acid per cent.	Gentian violet silver nitrate per cent.
Shock	1.0	3.3	4.3
Acute toxaemia	30.95	3.6	0.77
Sepsis	5.8	3.6	-
Other causes	1.0	-	1.5

The increased incidence of death from shock in the two later series is probably explained by the stricter criteria now used in the diagnosis of shock. It is at least likely that some of the cases labelled toxaemia in the first series would now be classified as suffering from shock. Even so, there has been a marked reduction in the mortality due to acute toxaemia since the use of the coagulation method was introduced, and there were fewer cases in the present series than in any previous one; whether this is because silver nitrate is a more effective coagulant than tannic acid or has some specific action, or because treatment is better organised and applied, is a matter for speculation. It is certain that silver nitrate is at least as good as tannic acid. No case in the present series died of sepsis; this is due in part at least to the use of sulphanilamide and sulphapyridine in the prophylaxis and treatment of infection. It is regrettable that patients

died /

died of secondary shock; it has been sufficiently stressed already that injuries of up to 65 per cent. of the body surface are not necessarily fatal, though the risk to life is very grave.

As mentioned above, the low incidence of acute toxaemia raises two important points: firstly, is silver nitrate a more efficient coagulating agent than tannic acid; secondly, has silver nitrate in some way modified the symptoms, signs and course of acute toxaemia as these were observed in cases treated with tannic acid. In answer to the first it may be said that silver nitrate produces a coagulum in a few seconds, and the resulting "tan" is thinner and more pliable than that obtained by the use of tannic acid. While several patients of this series exhibited the symptoms and signs of acute toxaemia, no evidence was obtained at post mortem examination of liver degeneration and necrosis which were invariably associated with toxaemia when the coagulant used was tannic acid. Post mortem examination of the liver of all fatal cases treated with silver nitrate who exhibit the symptoms and signs of acute toxaemia, and of all fatal cases occurring under other methods of treatment, is the only way in which further information is likely to be obtained. A feature which has not previously been recorded was the finding of yellow specks on the surface of the liver, which microscopically were found to be fatty degeneration which was confined to these areas.

Eosinophil leucocytes were found in the liver of 2 patients, both of whom died early. In the cases quoted by Wilson, Macgregor and Stewart (1938), eosinophils were found at various stages, and they suggested that these were apparently not always associated with early death, being found in one case which died on the seventh day. These cells were situated chiefly in the portal tracts though in one case they were found in the spleen at the margins of the malpighian corpuscles. The significance of these cells is still unknown. /

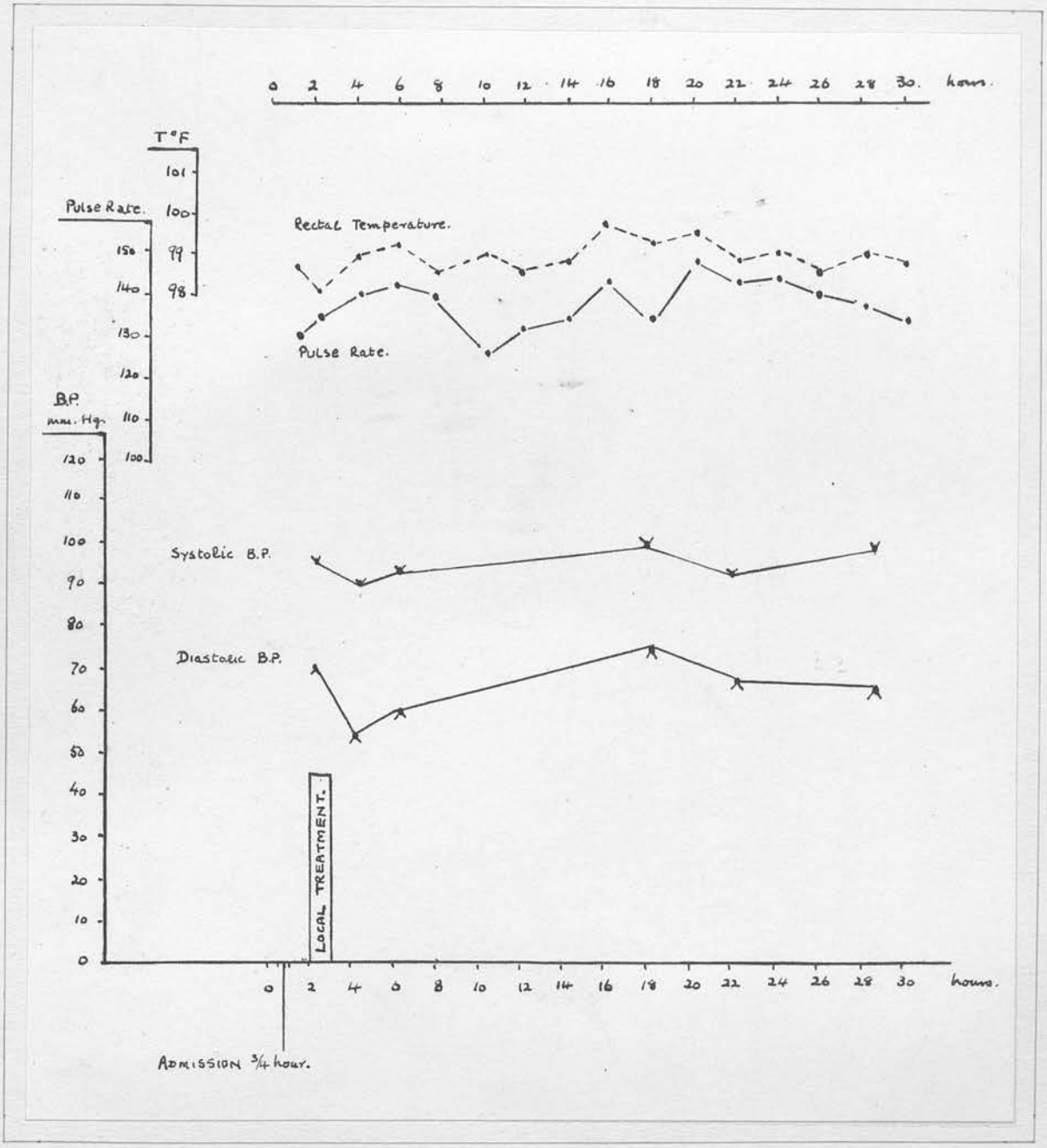
unknown. Eosinophils were found also in the liver of the baby (Case 22) who died at the age of 8 days as a result of septicaemia unconnected with the injury; here the eosinophils may be regarded as a normal feature in view of the age of the child; the other changes present in the organs were few and of a mild degree, and included pallor and slight evidence of damage to cells, chiefly in the liver. No evidence of bacterial infection, coexisting with toxaemia, was recorded.

ILLUSTRATIVE CASES.

These cases have been selected and grouped to illustrate the features of the clinical course and treatment of the various types of injury encountered in this series. All the fatal cases have been included. The grouping is as follows:

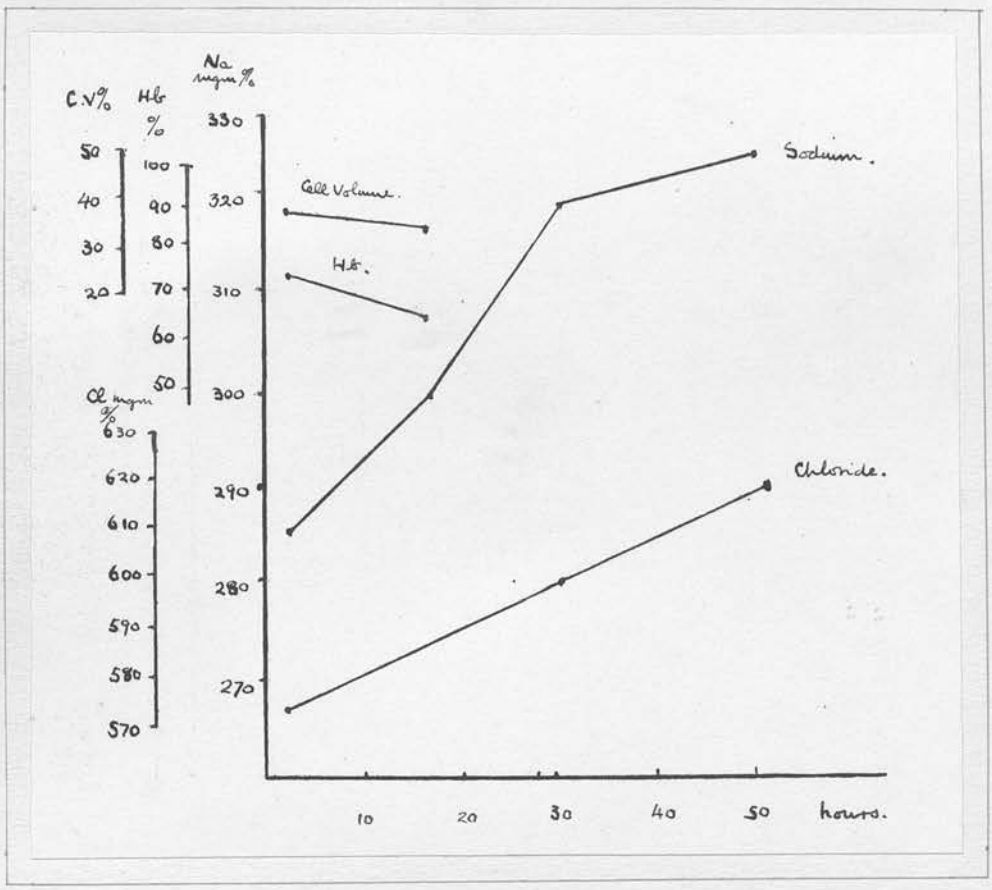
TABLE 16.

Feature	Case Numbers
Uneventful course	1, 2
Initial shock	3, 4, 10, 14, 16, 18
Secondary shock	3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18,21
Acute toxaemia	4, 13, 19, 20
Fatal Cases	
Secondary shock	14, 15, 16, 17, 18
Doubtful toxaemia	19
Broncho-pneumonia	21
B.coli septicaemia	22
Treatment	
Gum saline	4, 8, 10, 11, 13, 14, 15, 18, 21
Plasma saline	9, 18, 21
Cortical hormone	9, 10, 12, 13, 14, 15, 18, 19, 21



CASE 2.

Chart of Clinical Course.



CASE 2.

Blood Chemistry.

CASE 1 (Hospital Register number 4.5872). E.J. Female. Aged 2 2/12 years.

Admitted 1 hour after scalding by hot tea from tea-pot upset by child. Olive oil applied by mother.

Extent:

Lower abdomen, perineum, vulva, buttocks, small areas both thighs (15 per cent. of body surface).

Points:

1. Little shock on admission.

2. Involvment of perineum and vulva presented a difficult nursing problem. Met by placing sandbag beneath buttocks and tying arms and legs to supports at side of bed. Nursed in large cage.

3. Tan began to separate on 7th day, completely off on 10th day. No toxaemia, no sepsis. Discharged on 11th day.

Comments:

This case emphasises the importance of good nursing. The rapid separation of the tan may be partly explained by the fact that the scalds were not deep; the tea was hot but not boiling.

CASE 2 (3.6074). R.K. Male. Aged 2 4/12 years.

Admitted ¾ hour after scalding by sitting in a pot of hot water. No first aid.

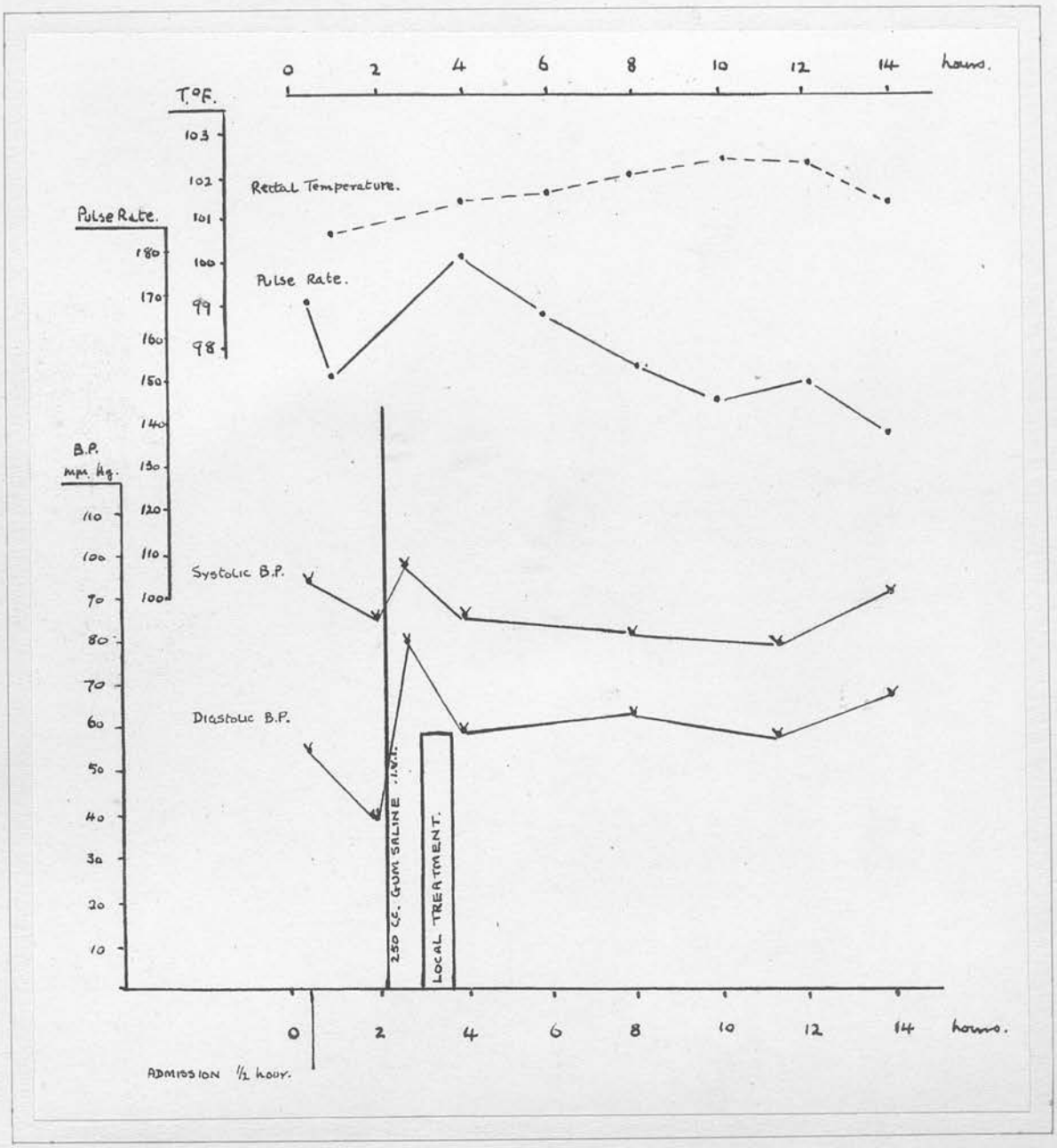
Extent:

Lower part of back, both buttocks and perianal region.

Points:

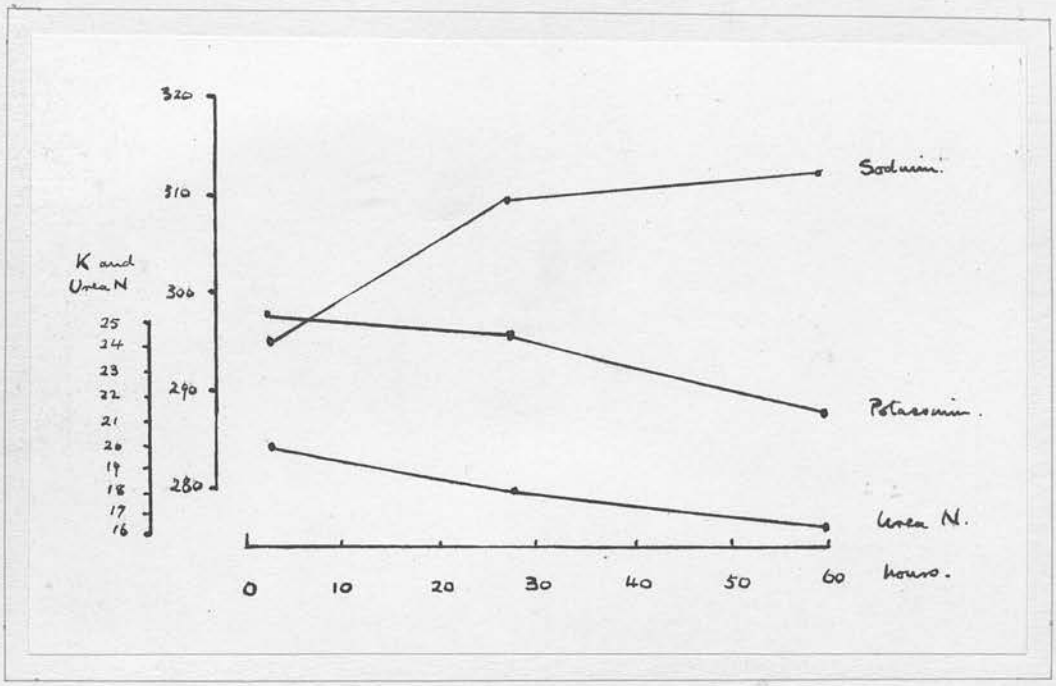
1. Shock present on admission, persisting during and after local treatment. Gradual recovery. (No blood pressure records).

2. /



CASE 3.

Chart of Clinical Course.



CASE 3.

Blood Chemistry.

2. Tan began to separate on 8th day, all off on 14th day. Healed, discharged on 15th day.

3. Serum sodium and serum chloride levels were low on admission, both rose during treatment.

Comments:

Quoted as an example of good healing and a normal course.

CASE 3 (3.5977). J.W. Female. Aged 2 4/12 years.

Admitted ½ hour after scalding by boiling water from kettle. Nightdress removed. No other treatment.

Extent:

Both thighs, buttocks, abdomen, back (50 per cent. of body surface).

Points:

1. Initial shock present on admission, B.P. 95/55, became worse in spite of warmth and sedatives; gum saline, 250 c.c., given intravenously, with subsequent improvement.

2. Condition poor at finish of local treatment but gradually improved.

3. Legs slung to gallows splint and sandbag placed under back to raise buttocks clear of bed.

4. Tan began to separate on 9th day. All off on 17th day. Some infection present as pustules and an indurated area on buttocks. Gentian violet applied to pustules and magnesium sulphate soak to indurated part. Indurated area formed abscess and discharged. M. & B. 693 given - resolved. Discharged on 23rd day.

Comments: /

Comments:

Initial shock progressed into secondary shock, which was treated with intravenous gum saline immediately it became evident that warmth and sedatives had proved ineffective. This prevented a further big fall in pressure during local treatment, after which the general condition gradually improved. Infection occurred in the healed area on the buttocks after the separation of the tan and delayed the discharge of the patient.

CASE 4 (3.6197). M.K. Female. Aged 1 3/12 years.

Admitted $\frac{1}{2}$ hour after scalding by hot tea. No first aid treatment.

Extent:

Both ankles, lateral half of left thigh, left buttock, left half of chest and left arm (20 per cent. of body surface).

Points:

1. Severe shock on admission which did not improve much with warming and sedative. At $2\frac{1}{2}$ hours was very restless, pale and cold. (No blood pressure records).

2. Condition deteriorated during local treatment which lasted 50 minutes. Pressure fell. Gum saline, 200 c.c., given rapidly intravenously. Some improvement at $4\frac{1}{2}$ hours. Condition still poor at 13 hours.

3. Temperature elevated from 1 to 11 hours as result of cage temperature being raised to combat chilling of child. Rise of temperature present from 36 to 120 hours, with associated rise in pulse and respiration rates.

4. Discharged healed on 15th day.

Comments:

In the absence of sufficient data it is difficult to explain the elevated temperature, but the appearance of the child was suggestive of a mild degree of toxaemia.

CASE 5 (3.5738). T.M. Male. Aged 1 6/12 years.

Admitted about 16 hours after scalding by boiling water from a saucepan upset by child.

Extent:

Face, anterior half of scalp, neck, front of both thighs and legs, and one foot (15 per cent. of body surface).

Points:

1. No shock on admission. (No record of blood pressure).

2. Treated inadequately by own doctor with Amertan smeared on lint and applied only to face as a mask. No treatment of other areas. Tan had taken only slightly in a few small areas.

3. Infection present on admission, most marked on scalp where some blisters amongst hair were still unbroken. Blister fluid: haemolytic streptococci on direct film and culture.

4. On admission marked oedema of face and eyelids, eyes shut. Pale, ashen, unhealthy toxic appearance.

5. During local treatment collapse occurred and blood pressure could not be recorded. Treatment suspended, preparations made to give gum saline. Half an hour later blood pressure 130/100. Anaesthesia restarted and treatment completed. Very restless after treatment for some hours but was drinking well and not vomiting.

6. Prontosil soluble 2.5 c.c. I.M.I. on 4 occasions and 1 g. by mouth t.i.d. Infection subsided.

7. Reinfection of area on chin where tan was rubbed off. Further injections of Prontosil and application of gentian violet. Discharged on 23rd day.

Comments: /

Comments:

This case illustrates the dangers inherent in even a small scald and some of the difficulties encountered in treatment. The treatment attempted at home was unsatisfactory, the scald was not completely treated and insufficient jelly was applied. Facial injuries should always receive the best possible attention. The fall of blood pressure during local treatment emphasises the danger of secondary shock arising during local treatment. Oedema of the face was marked on admission though the tan was incomplete and very thin on the scalded area. The toxic appearance of the child is usual in this type of case and is probably due to the oedema and associated pallor.

CASE 6 (3.5715). M.R. Female. Aged $4\frac{1}{2}$ years.

Admitted $\frac{1}{2}$ hour after scalds by boiling tea from teapot upset by child. Butter applied.

Extent:

Front of left thigh, leg, ankle, foot and toes and splashes on right ankle (5 per cent. of body surface).

Points:

1. No shock on admission. (No record of blood pressure).

2. Local treatment at 2 hours. At $2\frac{1}{2}$ hours collapsed, pulseless, blood pressure could not be recorded. Hot cage applied. Repeated attempts to record pressure, first successful at $3\frac{1}{2}$ hours, 110/?, at $4\frac{1}{2}$ hours 120/70. Colour remained good throughout though cheeks were pale when pulse failed. Further progress uneventful. No toxaemia, no sepsis.

Comments:

A severe fall of blood pressure occurred during the local treatment of a very small injury; the pressure returned to normal when heat was applied.

CASE 7 (3.5862). J.G. Male. Aged 1½ years.

Admitted 3 hours after scalding by boiling water and potatoes from saucepan upset by child. Olive oil applied by family, Amertan by own doctor.

Extent:

Front of both thighs, patches on abdomen, front of chest, and on left arm (20 per cent. of body surface).

Points:

1. No shock on admission. (No record of blood pressure)

2. Local treatment at 4 hours, severe shock developed during this, and at 4¾ hours blood pressure could not be recorded; child was very restless. Hot cage and heroin gr. 1/96. At 5 hours pressure was 118/100, at 5¼ 136/110 and at 6¾ 140/110. Further course uneventful.

3. Serum sodium was low but potassium was not as high as expected at 4 hours.

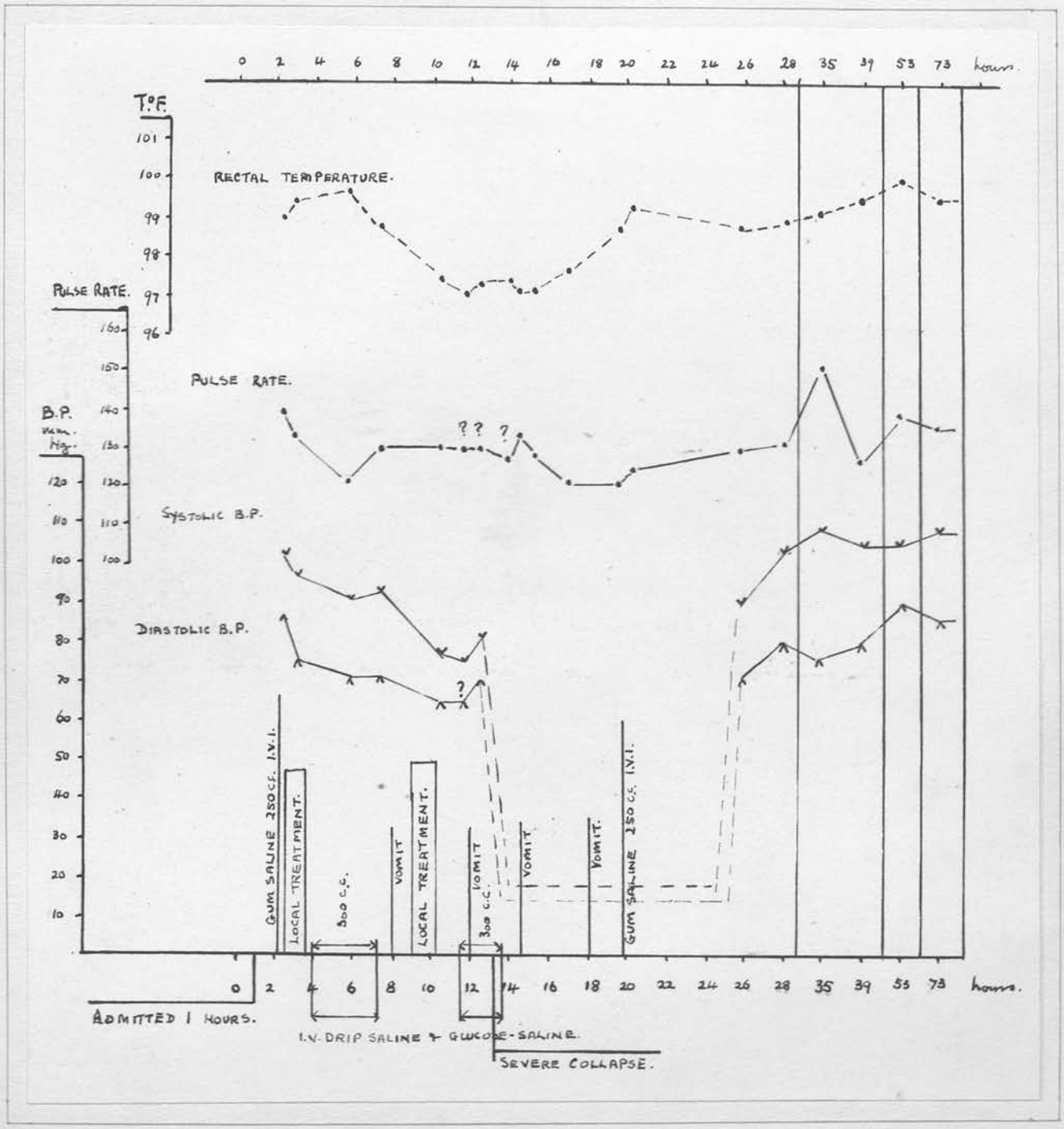
Comments:

Another example of severe shock occurring during treatment but passing off with warmth and sedative. The first aid treatment is a good example of the type of interference commonly practised. To apply Amertan after olive oil is useless, since the jelly is in a watery base and cannot reach any areas covered by oil; in this case the amount of jelly used was inadequate and the areas tanned negligible. Three hours elapsed between the time of injury and admission to hospital.

CASE 8 (3.5874). D.P. Male. Aged 7½ years.

Admitted 1 hour after burns as a result of his shirt-tail catching fire. Flames extinguished, shirt removed, covered with clean cloth.

Extent: /



CASE 8.

Chart of Clinical Course.

Extent:

Whole of back from shoulders down, buttocks and round anus, back of both thighs to below knees, right flank and a strip across the abdomen, both axillae, arms and patches on forearms and wrists; some central areas were third degree, considerable areas of first degree round margins (55 per cent. of body surface).

Points:

1. Looked severely shocked on admission but pressure was 104/86. Pallor, cyanosis, restlessness, little exudation of serum.

2. Gum saline 250 c.c. at $2\frac{1}{2}$ hours to combat shock. Heroin gr. 1/24 at 1 hour and gr. 1/48 at $2\frac{1}{4}$ hours.

3. Severe collapse during treatment; 6 per cent. glucose in 0.9 per cent. saline by intravenous drip infusion at rate of 50 drops per minute.

4. Tan did not take well and exudation continued during the next few hours. Gentian violet and silver nitrate were reapplied at 9 hours. Fluid still exuded after retanning so drip rate was reduced in spite of intense thirst and vomiting.

5. Sudden collapse at $13\frac{1}{2}$ hours. Exudation very marked, drip stopped. Complaining of cold, cage temperature raised; pallid and cyanosed. At $19\frac{1}{2}$ hours given gum saline 250 c.c. Condition gradually improved. Mental disturbance present from 10 to 36 hours - bad tempered.

6. Tan began to harden after 30 hours, became very thick subsequently and cracked and wrinkled, the result of the unusually heavy applications.

7. Tan separated early and raw areas were left. Some infection occurred but was controlled fairly well by M. & B. 693.

8. Raw areas dressed with different agents, acriflavine and paraffin, and yeast preparations. No difference in rate of healing was observed. Later in course exuberant granulations were burned down with copper sulphate (blue-stone). /

(blue-stone). Discharged in 23rd week.

Comments:

The importance of treating existing shock before attempting local treatment and the necessity for anticipation of shock occurring as a result of cleaning and tanning are evident. Secondary shock was treated by glucose saline infusion without effect; the exudation of fluid on the tanned surface was not controlled even by repeated tanning. After gum saline was infused at 19½ hours the severity of the shock lessened but did not pass off until after 36 hours. The absence of acute toxaemia was noteworthy and was probably related to the greater depth of the burn. Septic toxaemia did not occur though sepsis was present.

At 2 hours the serum sodium was low and at 64 hours the potassium was raised. Both showed a tendency to return to their normal values. The sodium was low on the 13th and 27th days, due to the loss of body fluids in the exudations of the large infected healing areas.

CASE 9 (3.6372). M.N. Female. Aged 7 years.

Admitted 3 hours after extensive burns, the result of her clothes catching fire.

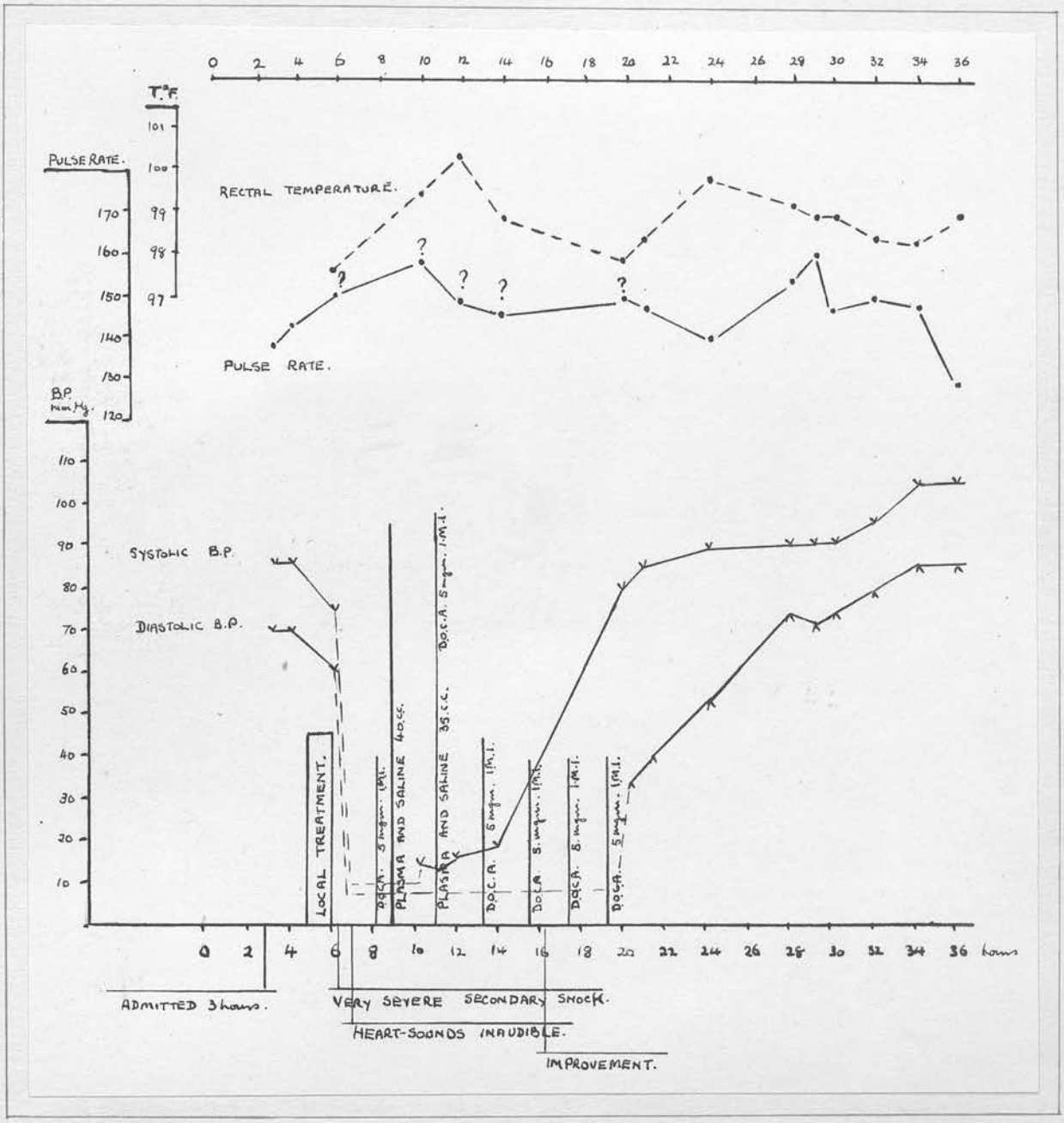
Extent:

Lower right quadrant of abdomen and front of right thigh, front of lower half of left thigh, half of front left arm, whole of right arm, upper part of back and left side of neck, chin and left cheek (25 per cent. of body surface.

Points:

1. Severe shock on admission.

2. Right arm was tense with oedema and olecranon bursa was distended with /



CASE 9.

Chart of Clinical Course.

with fluid.

3. Fall of blood pressure after treatment at 5 hours passing into complete collapse at 6 hours with absence of pulse and heart sounds; signs of impending death. D.O.C.A. 5 mg. was given intramuscularly and shortly afterwards pulse beats could be felt but pressure could not be recorded.

4. Plasma and saline infusion was attempted at $7\frac{1}{2}$ hours but veins were all collapsed and only 40 c.c. could be run in. Another attempt was made at 10 hours but only 35 c.c. ran in before the flow ceased.

5. The improvement of the circulation after D.O.C.A., warmth and sedative was indicated by improvement in pressure, colour and skin temperature.

6. Infection with haemolytic streptococcus occurred at tan edges in neck and abdomen, controlled with M. & B. 693 and hypertonic saline dressings.

Comments:

On the basis of previous experience, when the first injection of D.O.C.A. was given there seemed to be no hope of the child surviving. The recovery was dramatic and sustained. It was impossible to obtain blood for biochemical analysis even from the femoral vein. This was the first occasion on which plasma saline was used in the treatment of secondary shock. It was unfortunate that more could not be run in; no reaction was observed.

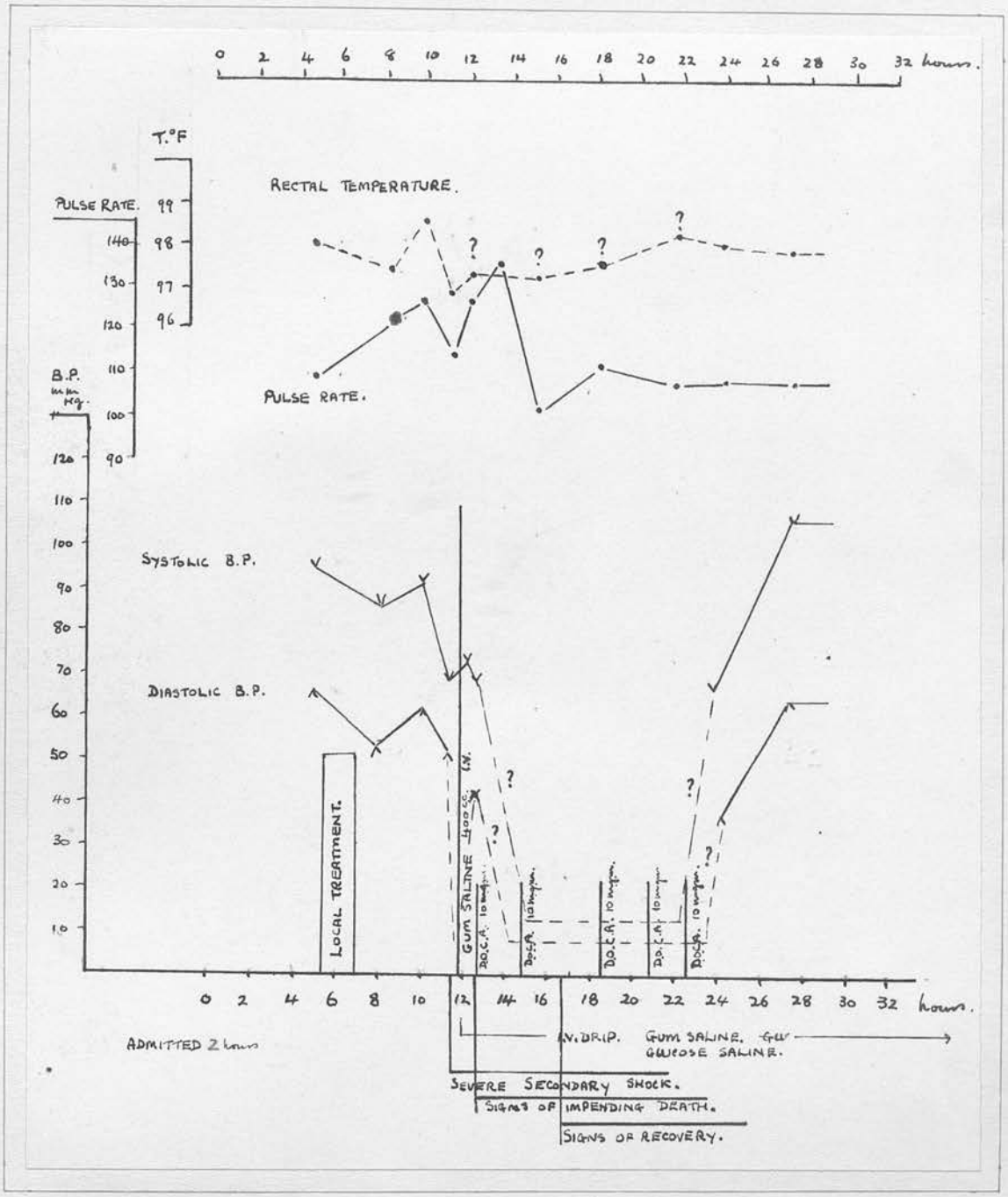
CASE 10 (R.I.E. 7/8, Q711). M.F. Female. Aged 16 years.

Admitted 2 hours after scalding as a result of falling into a boiler containing almost boiling water.

Extent:

Whole of front of trunk below level of nipples to groins, both flanks, back from lower angle of scapulae to mid thighs, upper half of front of both thighs, small area on anus (65 per cent. of body surface).

Points: /



CASE 10.

Chart of Clinical Course.

Points:

1. The girl stated that she was so frightened at the time of the accident that she felt no pain at first, and having been pulled out of the boiler (in which she remained for nearly 1 minute), she was able to walk 100 yards across a field to the farmhouse. When her mistress pulled off her clothing and applied oil to the burns, the pain was so intense that "she thought she would go crazy."

2. On admission no apparent shock, suffering little pain and talking rationally and cheerfully; blood pressure 95/60.

3. Local treatment at 5 hours. Tan did not take well at first - further applications made. Condition was poor, some cyanosis and pallor.

4. Collapse occurred at 12 hours and condition appeared hopeless, but improved after D.O.C.A. had been given intravenously and with further normal and gum saline. Difficulty was experienced in maintaining the cage temperature at a sufficiently high temperature and she complained frequently of cold. Pallor and some cyanosis persisted until 29 hours when both had improved.

5. The fact that both front and back of the trunk were involved made nursing very difficult. It was found impossible to keep the tan dry even with frequent turning which was intolerable to the patient. It was therefore decided that as the front was less widely involved than the back, she would have to be nursed on her face. That part of the tan soon became soft and peeled off and retanning was attempted on several occasions but was in vain as the new tan which always took well, soon peeled off. She developed incontinence of urine and this contributed to the water-logging of the tan, as all control over the bladder disappeared for some weeks. It was found that the application of silver nitrate induced intense pain for a few minutes but than gentian violet was painless.

6. /

6. From the 12th day there was a profuse purulent discharge from beneath the tan especially in the moist areas, but at first no organisms were found on culture. The discharge was at this time due to liquefaction of sloughing tissue beneath the tan. Later staphylococcal infection occurred, and a few days after this B. proteus was found on culture in this case and in another in the male ward.

7. Completely healed in 11 weeks.

8. A point in the nursing procedure is of importance. The vulva and anus were both involved and in an attempt to prevent soiling, regular catheterisation was carried out. The rectum was regularly emptied by giving an olive oil enema and following this with a soap and water enema, both then being siphoned off.

Comments:

Gum saline or plasma saline should have been given before local treatment in view of the extent of the injury. An inadequate volume of gum saline was given. The action of D.O.C.A. appeared to be vital at 12 hours when the condition of the patient was apparently hopeless. It is difficult to assess how much of the dramatic improvement can be attributed to D.O.C.A. and how much to intravenous infusion.

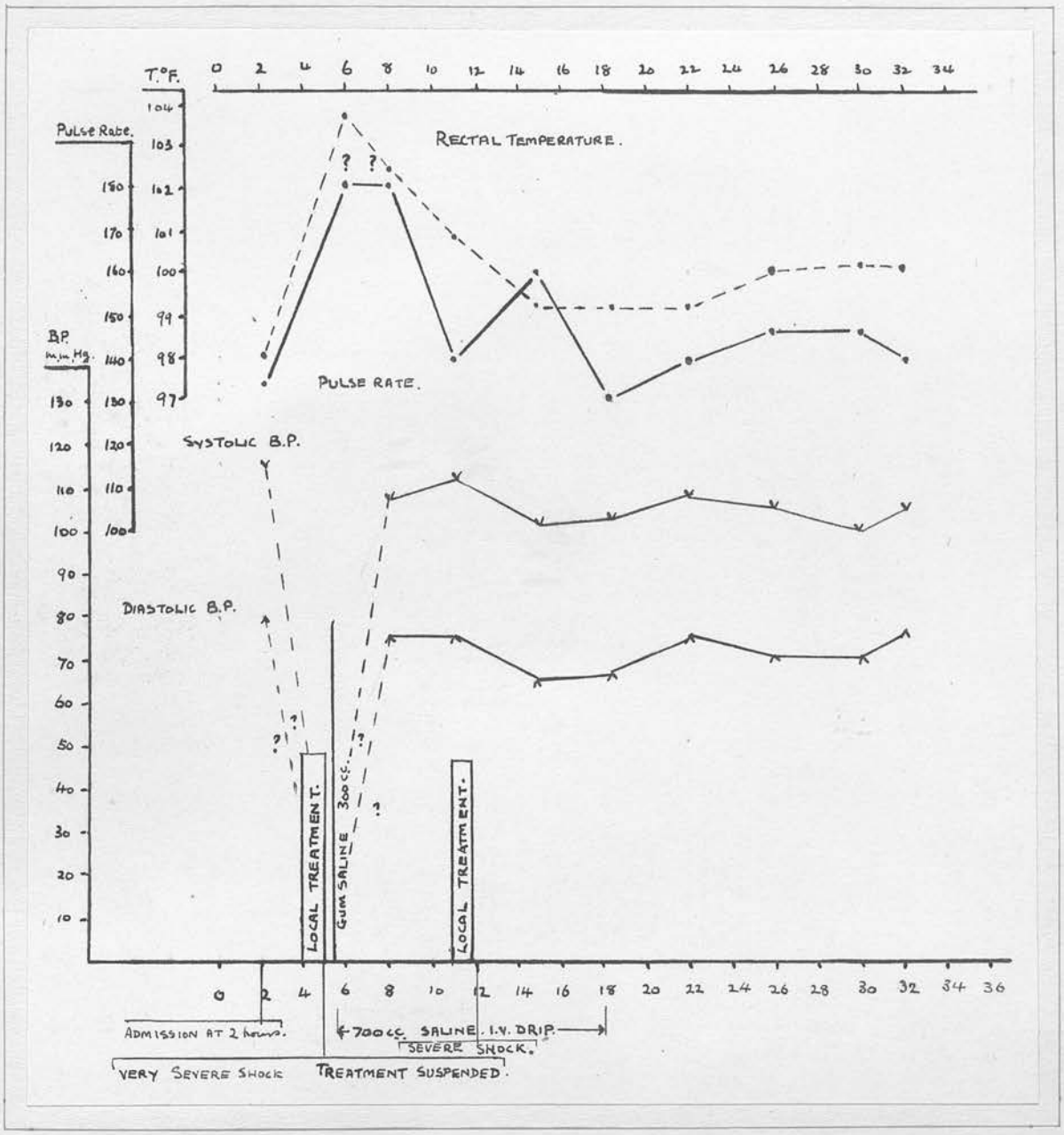
CASE 11 (3.6043). J. McB. Male. Aged 2 1/12 years.

Admitted 2 hours after scalding by contents of a pot of boiling stew. Tannic acid jelly was applied by doctor $\frac{1}{2}$ hour after injury.

Extent:

Lower half abdomen, penis, scrotum, perineum, both buttocks, both thighs, left ankle (20 per cent. of body surface).

Points: /



CASE 11.

Chart of Clinical Course.

Points:

1. No shock on admission. (No blood pressure record).

2. Local treatment begun at 4 hours. About halfway through the child's condition became so bad that cleaning was stopped and 300 c.c. gum saline were run rapidly into the saphenous vein. A further 700 c.c. of saline were run in slowly during the next 6 hours. Local treatment was completed at 11 hours and a further slight fall in pressure occurred.

3. Condition was satisfactory until 80 hours apart from occasional twitchings and persistent pallor and appearance of shock. At 80 hours peripheral circulation became very poor and pulse was "weak and thready." A rash appeared at 50 hours on the abdomen and spread to chest and arms during the next 24 hours, but had faded at 96 hours. Streptocide, 0.5 gm. t.i.d., and later M. & B. 693, 0.5 gm. t.i.d., were given.

4. Temperature raised from 2nd to 9th days. No cause found except some moistness of tan in groins.

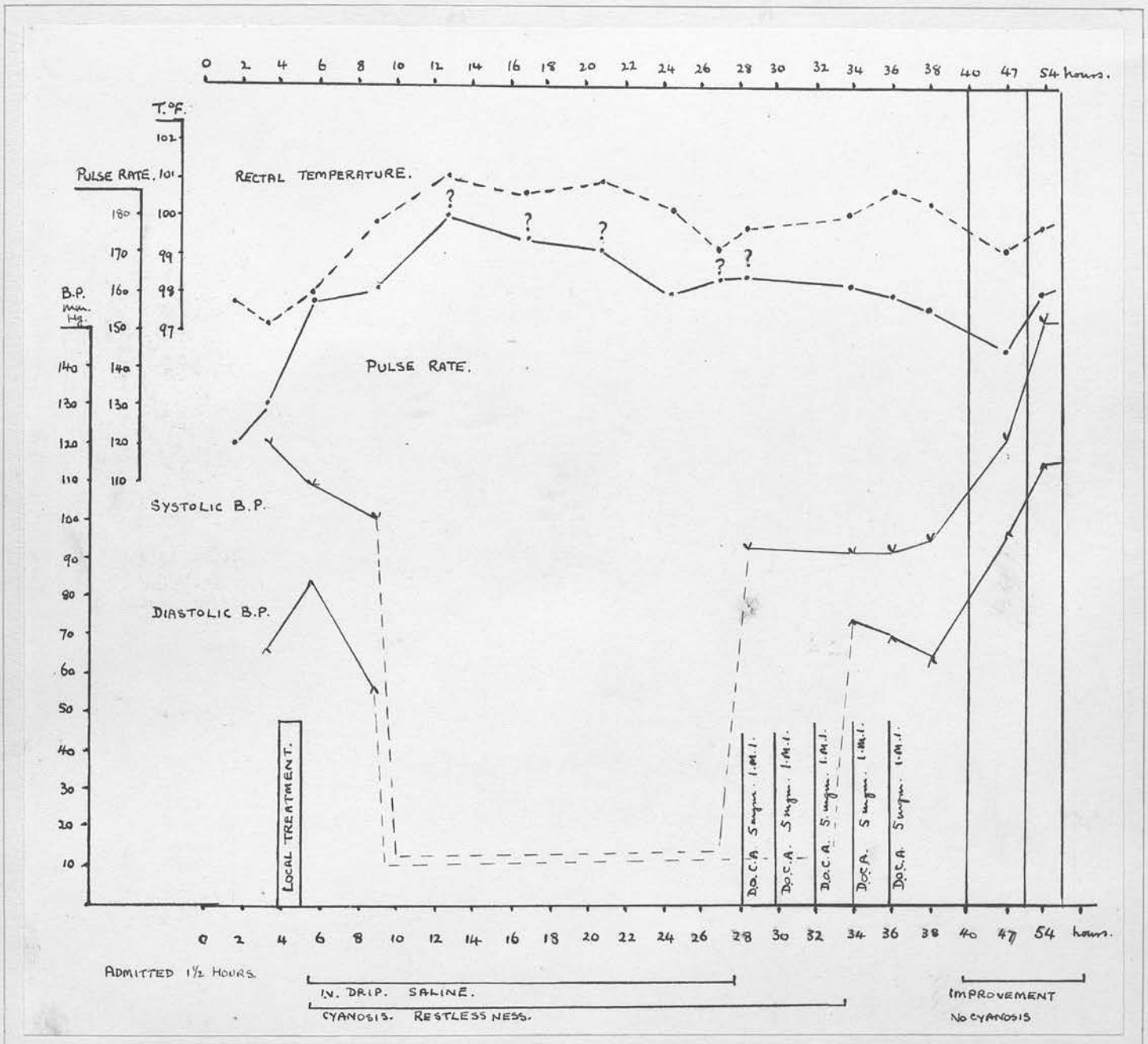
5. Tan began to separate on 11th day. All off on 18th day.

6. Abscess developed in groin on 18th day - haemolytic streptococcus. Another abscess formed in the cubital fossa and was drained on the 23rd day - haemolytic streptococcus. Discharged with all areas healed on 34th day. M. & B. 693, 0.5 gm. t.i.d., was given from the 20th to 24th days.

Comments:

The fall in blood pressure during local treatment could hardly have been predicted; it recovered with gum saline but recurred, though less severely, when the cleaning and tanning were completed some hours later.

The persistent elevation of temperature may have been due to a low-grade toxaemia, but it seems more likely that the rash was an outward sign of a generalised streptococcal infection which later became localised in the groin and cubital fossa. The dosage of streptocide and M. & B. 693 was inadequate and the /



CASE 12.

Chart of Clinical Course.

the drugs could not have reached effective concentration. Children tolerate these drugs very well in doses which are relatively very large; unless adequate amounts of the drugs are given they are of little value.

CASE 12 (4.6269). A.C. Male. Aged 2 11/12 years.

Admitted 1½ hours after scalds by boiling water from a kettle on an unguarded fire.

Extent:

Whole of right half of body, front and back, right arm and part of right hand, whole of right thigh and leg, left leg below knee but not foot (50 per cent. of body surface).

Points:

1. No shock on admission.

2. Blood chemistry normal at 2¾ hours.

3. Blood pressure fell during local treatment at 4 hours. Coramine, 0.5 c.c., and intravenous infusion of saline.

4. Very restless after anaesthesia passed off, semi-delirious, heaving about, jerky movements, marked cyanosis. All these features persisted and cyanosis became more marked. Oxygen was given at intervals.

5. Marked alkaptonuria at 24 hours.

6. No vomiting.

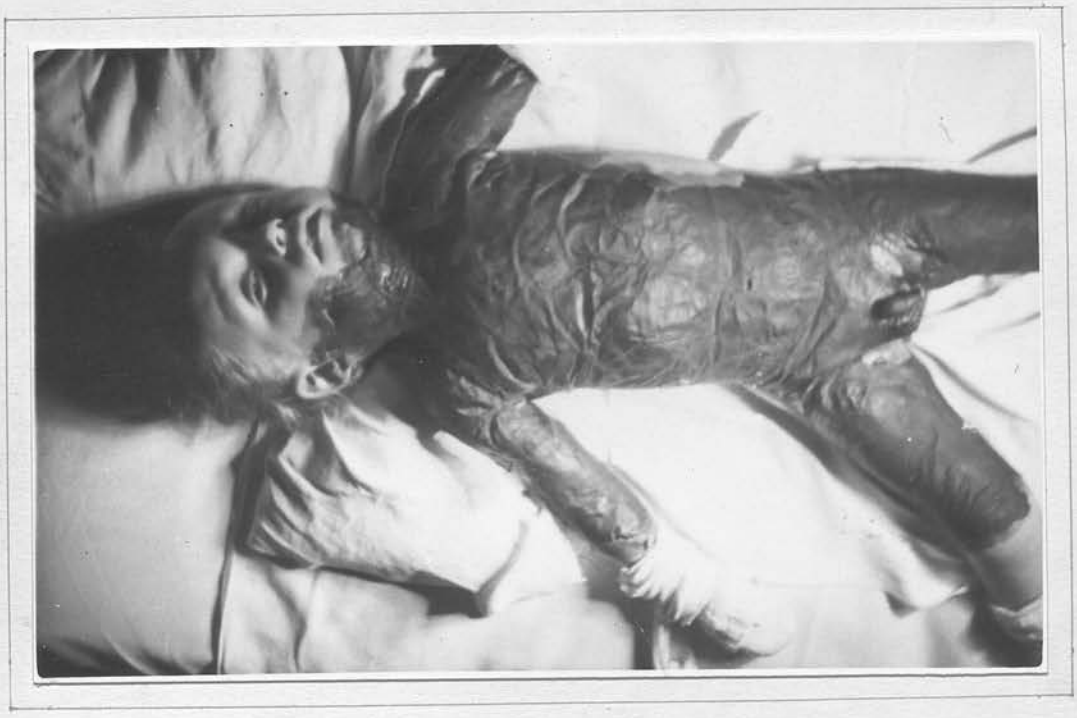
7. D.O.C.A., 5 mg., 5 two-hourly intramuscular injections.

8. Paraldehyde 1½ dr. at 34 hours.

9. At 40 hours marked improvement, temperature normal, taking fluids well by mouth.

10. Discharged on 14th day, some small areas still healing.

Comments: /



CASE 13.

Extent of Scald.

Comments:

The normal blood chemistry at $2\frac{3}{4}$ hours is striking. Secondary shock began to develop after local treatment. Improvement was not evident until 28 hours and was marked only after 40 hours.

CASE 13 (3.5879). D.L. Male. Aged 2 2/12 years.

Admitted 2 hours after scalding by boiling water from a pan upset by the child.

Extent:

Lower part of face, neck, whole of anterior surface of trunk, both thighs to the knees and arms to the elbows, part of left forearm (50 per cent. of body surface).

Points:

1. First aid treatment: cold water poured over patient by guardian. Own doctor covered areas with clean linen.

2. Severe shock on admission. Barely conscious, pulse imperceptible.

3. Gum saline, 350 c.c., rapid intravenous infusion.

4. Local treatment at 3 hours caused shock to become worse, condition was desperate at the end of treatment in spite of rapid completion and continuous intravenous infusion of gum saline. At times child appeared to be dead.

5. Improvement in condition after local treatment.

6. Acute toxaemia from 70 to 110 hours with recovery. At 94 hours condition desperate, pulse rising and temperature falling. Death appeared to be imminent. Marked improvement subsequent to injections of D.O.C.A., 5 mg. repeated 4 times at intervals of 2 hours.

7. Sulphanilamide given prophylactically from 5th day.

8. /

8. Infection with haemolytic streptococcus, treated with sulphanilamide.

9. Some areas of tan remained adherent for 5 weeks indicating deep injury.

10. Healing delayed by infection, great extent and unusual depth of injury. Severe haemorrhage occurred from large veins in granulating surface.

11. Pinch-grafted during 9th month.

12. Discharged after 272 days in ward.

Comments:

But for the immediate administration of gum saline on admission, this child would have died before local treatment could have been begun. As it was, shock was so severe that death appeared to have occurred more than once during local treatment. Recovery from shock was gradual. Acute toxaemia was nearly fatal but appeared to be relieved in a dramatic way by the giving of D.O.C.A. The very long course is unusual in scalds and was due to the severe degree of injury present.

CASE 14 (3.6220). C.O. Female. Aged 4 4/12 years.

Admitted 1½ hours after severe burns due to clothing catching fire. Treated with olive oil by parents.

Extent:

Face, neck, chest, abdomen, both arms, part of both hands and the whole of the back of the body, buttocks and the perineum (65 per cent. of body surface).

Points:

1. Severe initial shock present on admission. Blood pressure could not be recorded, pulse only occasionally present; pale, cold and quiet.

2./

2. Local treatment started 1 hour after admission, lasted 2 hours. Slight improvement in early stages of anaesthesia. At end of treatment condition had deteriorated markedly, grey colour, cold, peripheral circulation very poor.

3. Gum saline intravenously at 5 hours.

4. D.O.C.A., 5 mg. at $5\frac{1}{2}$ hours.

5. At 6 hours slight improvement evident but still cold. Died $9\frac{1}{4}$ hours after injury. Blood pressure could not be recorded at any time.

6. No post mortem examination.

Comments:

Initial shock passed into a state of severe progressive secondary shock which did not respond to gum saline or D.O.C.A. Gum saline or plasma should have been given before local treatment. Local treatment lasted far too long; a child of this age will not withstand easily anaesthesia lasting 2 hours even when condition at the outset is excellent. A burn of this extent is compatible with survival provided that adequate treatment is applied energetically and early.

CASE 15 (4.6480). A.L. Male. Aged 2 5/12 years.

Admitted $\frac{1}{2}$ hour after having fallen into a tub of boiling soapy water. Clothes removed, no other treatment.

Extent:

Whole of back, left leg, left flank, buttocks, perineum and half of abdomen (45 per cent. of body surface).

Points:

1. Incomplete data.

2. /

2. Severe shock after local treatment at 2½ hours, treated by intravenous gum saline, 200 c.c. D.O.C.A., 1 c.c., given hypodermically at 6 hours.

3. Sudden collapse at 8 hours. Coramine and oxygen given; died.

4. No post mortem examination.

Comments:

Severe secondary shock was not adequately treated and the terminal collapse was due to sudden failure of compensatory mechanisms which had maintained the circulation until this time. Coramine and oxygen are of no value in the treatment of this grade of shock. A single dose of 1 c.c. of D.O.C.A. probably has little effect.

CASE 16 (3.6025). I.S. Female. Aged 3 1/12 years.

Admitted 1 hour after burns due to clothing catching fire.

Extent:

Lower part of face, left side of neck, both arms, right hand, front of chest, both thighs, whole of back (60 per cent. of body surface).

Points:

1. Severe initial shock on admission, blood pressure 84/50.

2. After local treatment (duration 35 minutes) at 1½ hours the blood pressure could not be recorded, the limbs were cold.

3. Condition poor until 6½ hours when further collapse was noted; the respirations had risen to 40 per minute. Death occurred 10 minutes later.

4. The serum sodium was normal at 1½ hours but fell at 5 hours; there was a coincident rise in urea nitrogen.

5. Progressive rise in temperature to 104°F. shortly before death.

Post /

Post Mortem Appearances:

Liver: Slightly paler than normal. On upper surface of right lobe, just under diaphragm, there was a group of yellow patches, bright golden yellow in colour, irregular in shape, and slightly firmer than rest of liver substance, penetrating into liver substance for fully 1 cm. in some instances. No similar areas in other parts. Microscopically these areas were seen to be patches of severe fatty change. They were circumscribed; no other areas of fatty change present. In yellow areas there was disintegration of the liver cell-columns and nuclear pyknosis. Many portal tracts were densely infiltrated with small mononuclear cells amongst which were some eosinophils. This change was not confined to the areas showing fatty change, but was most marked in those regions.

Spleen: No excess of eosinophils.

Suprarenals: Normal.

Kidneys: Slightly pale and swollen.

Lungs: Healthy.

Comments:

Death was due to secondary shock, no attempt at active treatment of this shock having been made. Local treatment of such an extensive injury with a blood pressure of 84/50 is contraindicated.

The yellow patches on the surface of the liver are of great interest, but their significance is not yet clear.

The progressive rise in temperature was probably due to overheating.

CASE 17 (4.6014). R.G. Female. Aged $3\frac{1}{2}$ years.

Admitted $1\frac{1}{4}$ hours after burns due to nightdress catching fire. First aid treatment: picric acid applied by own doctor.

Extent:

Lower part of face, palms of both hands, both knees and thighs, perineum, right buttock (30 per cent. of body surface).

Points:

1. Incomplete data.

2. "Not very shocked" on admission. Pulse rate 150, restless.

3. Local treatment at 5 hours, condition satisfactory at finish and during night until 13 hours when collapse occurred suddenly and death ensued in 40 minutes.

4. Blood chemistry: sugar, non-protein nitrogen and potassium were raised, and sodium reduced, at 5 hours.

5. Slight terminal rise in temperature to 101°F.

Post Mortem Appearances:

Lungs: No evidence of change. Pharynx and epiglottis showed a certain amount of inflammation of the mucous membrane but there was no evidence of injury by heat in trachea or bronchi.

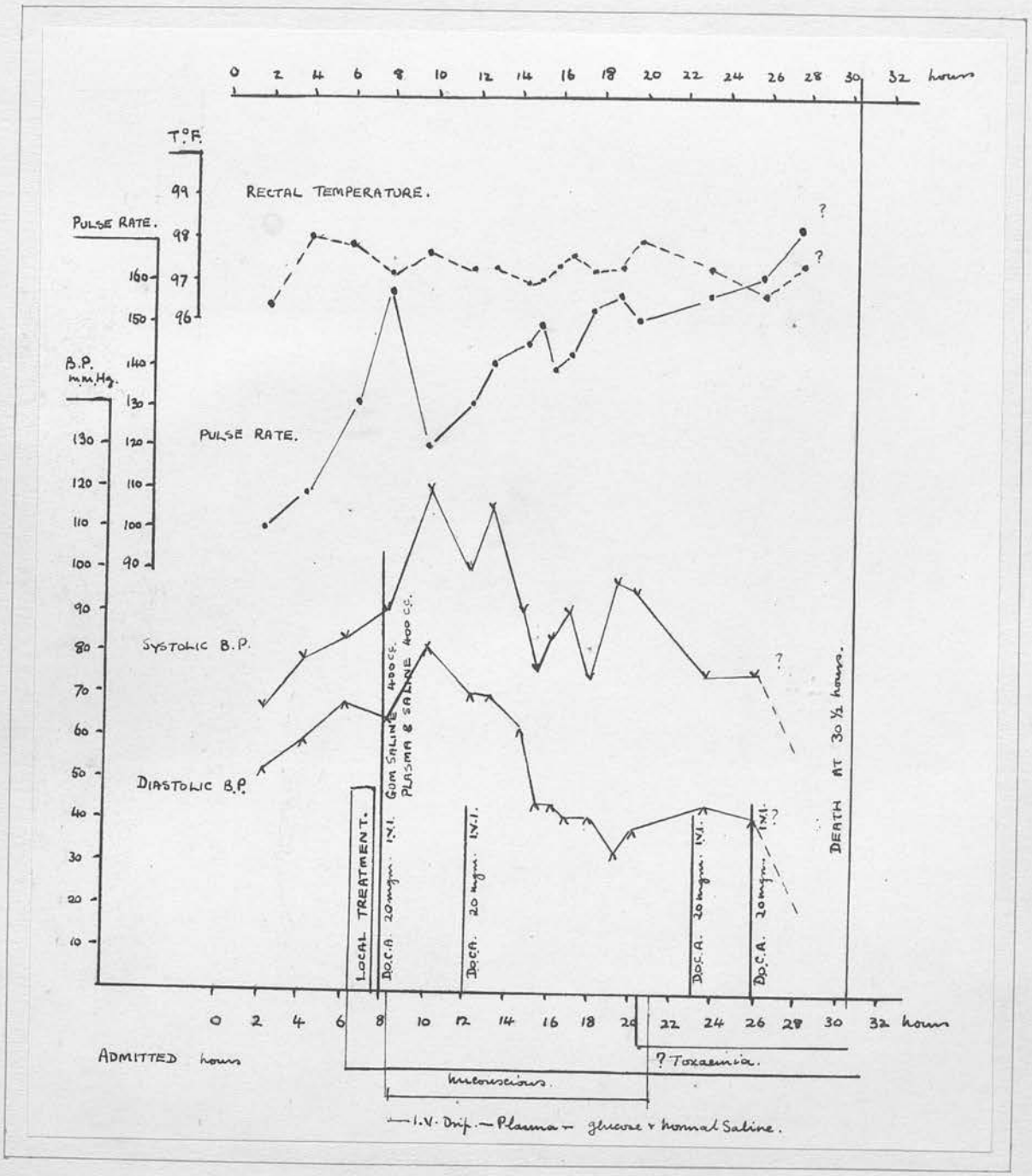
Liver: Slightly paler than usual. No appearance characteristic of toxaemia of burns. Cells showed slight karyolysis. No necrosis or fatty degeneration in central zones or elsewhere. Increase in cells in portal tracts, lymphocytes and plasma cells with a few eosinophils.

Spleen: Increased number of eosinophils not numerous but more than usual, occurring throughout the pulp and especially at the edge of the malpighian bodies.

Comments: /

CASE 18.

Extent of Injury.



CASE 18.

Chart of Clinical Course.

Comments:

This child died from unrecognised progressive secondary shock. This case is similar in some respects to one quoted by Wilson et al. (1938, Case 9) in which picric acid was used, and they suggested that death might have been due to picric acid poisoning in a susceptible individual. Eosinophils were found in 2 cases recorded by Wilson et al. (1938) in which death occurred at $5\frac{1}{2}$ and 7 hours after injury, and have been found also in Case 16 of the present series.

CASE 18 (R.I.E. 7/8, Q.726). Mrs J. Aged 73 years.

Admitted $1\frac{3}{4}$ hours after burns as a result of her nightdress catching fire.

Extent:

See photograph.

Points:

1. Severe shock and subnormal temperature on admission. Conscious and rational.

2. History of previous hypertension and at least one cerebral embolism, resultant difficulty in estimating degree of shock present in spite of blood pressure readings. Poor general condition.

3. There were several large areas of skin which had not been raised as blisters, but were coagulated by heat in situ, giving rise to a tough, shiny yellowish-brown patch which could not be removed, but which apparently tanned well. (Similar areas have been noticed before in other cases, and it has always been found that in these parts healing is delayed and large sloughs separate after 3 or 4 weeks, while in other areas the tan lifts more or less normally. Scarring is always marked in such regions, which represent sites of deeper destruction, for example third and fourth degrees).

4. /

4. Local treatment at 7 hours. Blood pressure 120/75 at start. Intravenous gum saline 400 c.c. started during treatment. Anaesthetic: nitrous oxide and oxygen. Condition poor at end of treatment. Blood pressure 110/70.

5. D.O.C.A., 20 mg., given intravenously at $7\frac{3}{4}$ hours. A marked rise in pressure had occurred by 10 hours. This cannot be attributed entirely to D.O.C.A. since gum saline and later plasma saline were also given during this period.

6. No unfavourable reaction of any kind observed during or after giving of plasma and saline.

7. Patient did not recover consciousness after the anaesthetic and occasionally her appearance was suggestive of intracerebral haemorrhage. During most of the time her whole musculature was flaccid, pupils dilated and respirations deep, sighing or grunting. Signs of pulmonary congestion were present in last few hours. Died at $30\frac{1}{2}$ hours.

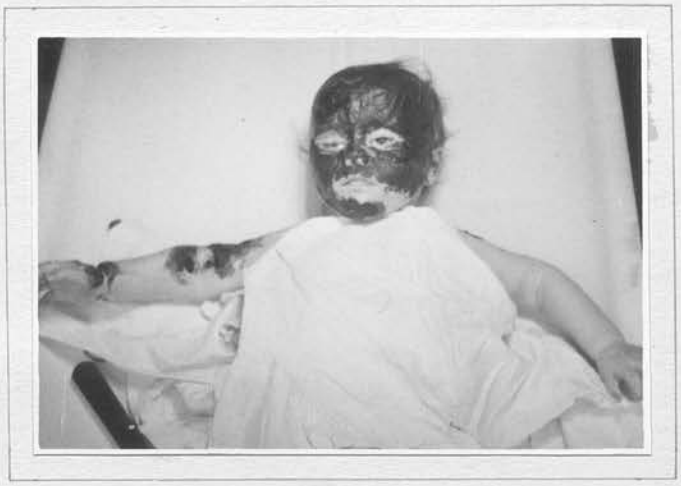
Comments:

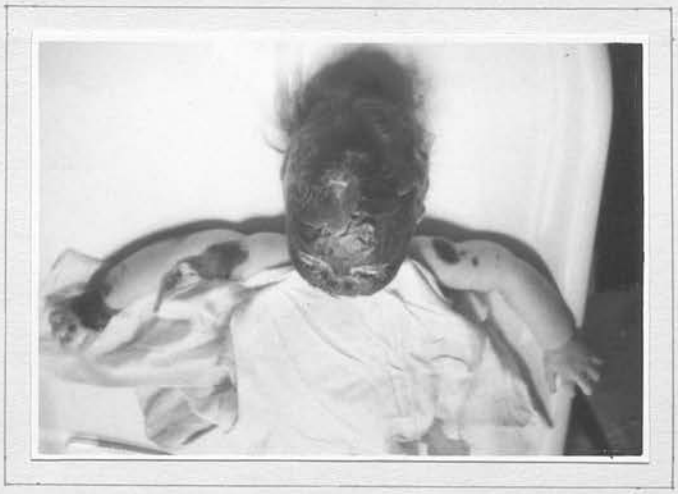
The prognosis was bad from the outset. A further fall in serum sodium occurred between 4 and 14 hours in spite of intravenous saline and D.O.C.A. A rise in non-protein nitrogen and chloride occurred in the same period. The fact that no post mortem examination could be obtained was a great disadvantage. Death was due to progressive secondary shock in an aged subject. Two pints of plasma and saline were given to this patient without any untoward reaction being observed, and the effect on the blood pressure was beneficial.

CASE 19 (3.5694). T.S. Male. Aged 11/12 year.

Admitted $1\frac{1}{2}$ hours after upsetting cup of boiling tea. Olive oil applied.

Extent: /





CASE 19.

Extent of Scald.

Extent:

See photograph.

Points:

1. No initial shock, blood pressure 110/80 on admission.

2. Tolerated local treatment well.

3. Restless on 5th day but no other disturbance.

4. Temperature 99.6°F. on 9th day. No sign of sepsis - thought to be due to variations in cage temperature.

5. Tenth day - fluctuation of pulse rate began in morning. Some restlessness. General condition poor during night. Toxaemia was considered but was tentatively excluded owing to lateness of onset.

6. At 6 a.m. on 11th day condition was alarming - cyanosis, pallor, pulse poor. Coramine induced some improvement but $3\frac{1}{2}$ hours later death appeared to be imminent. Eucortone, 0.5 c.c., was given I.M.I. at 9.30 a.m. and repeated at 10 a.m., 11 a.m. and 11.30 a.m. Slight improvement in appearance was evident until 11.40 when became cold and clammy and cyanosis increased. Some transitory improvement was noticed after these injections and after glucose saline into the sagittal sinus. Death at 12.15 p.m.

7. Terminal fall in blood pressure but no rise in temperature.

8. There were several features of interest in the biochemical findings. There was a rise in chlorides and later a rise in non-protein nitrogen. There was a progressive and marked fall of both sugar and CO_2 combining power. The haemoglobin was remarkably constant. There was a rise in sodium and potassium followed by a fall in the terminal stages.

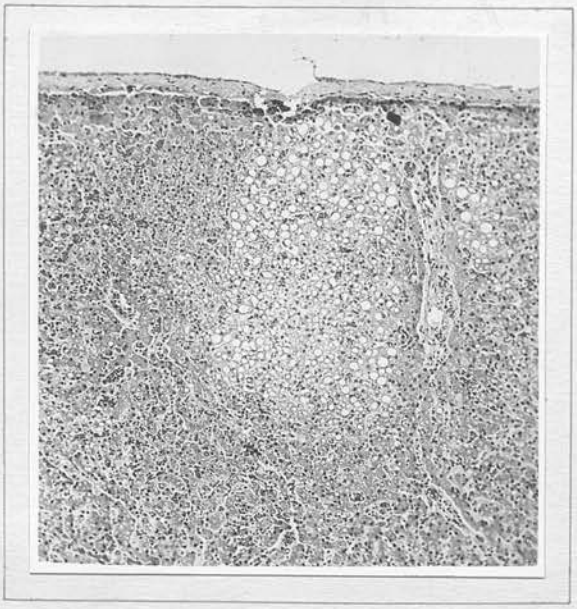
9. No signs of sepsis in tanned surfaces.

Post Mortem Appearances:

Lungs: Some congestion and oedema of the bases.

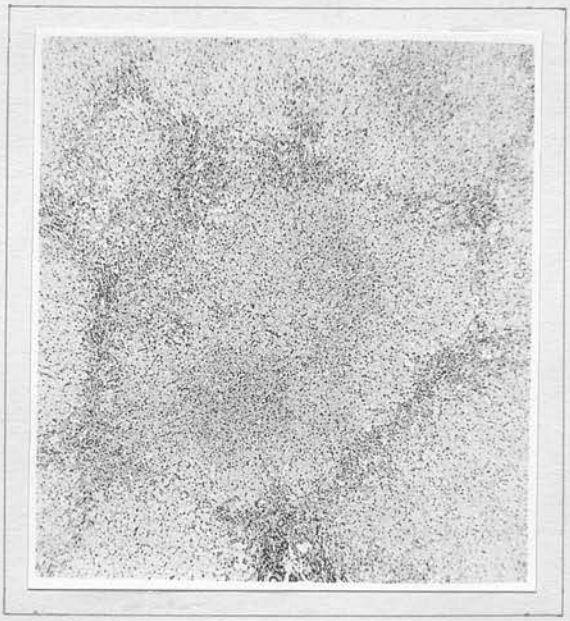
Liver: /

Sections from Fatal Cases treated with Gentian Violet and Silver Nitrate.



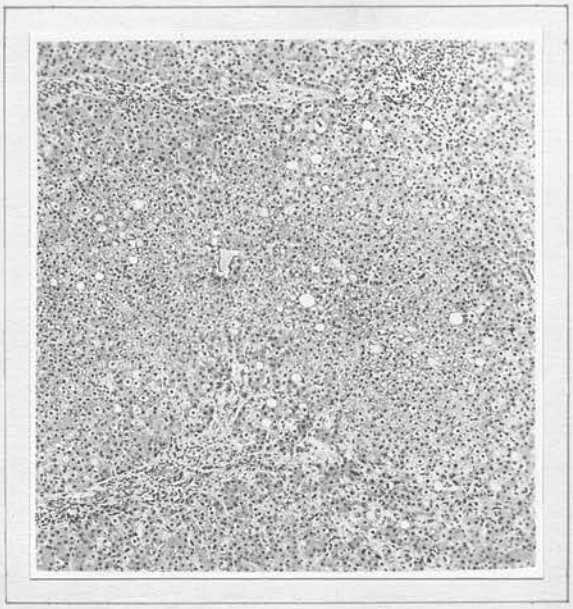
Liver x 100

Area of fatty degeneration just beneath the capsule, giving rise to yellow area seen in the gross specimen.



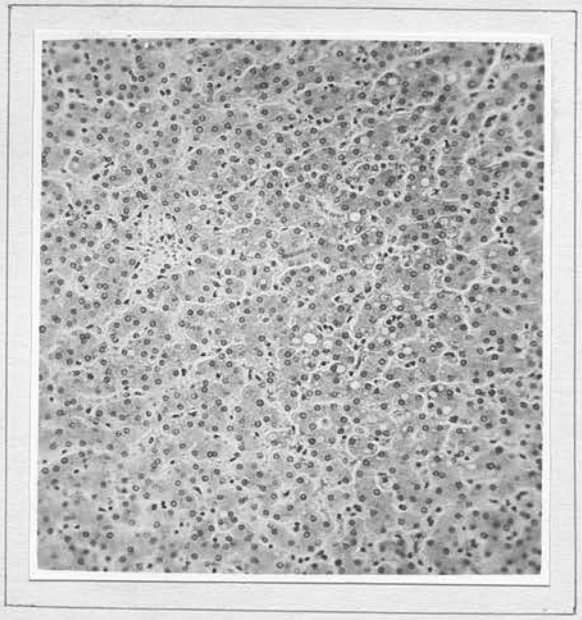
Liver x 70.

Typical appearance found in acute toxaemia of burns treated with tannic acid (20 per cent.). Central necrosis of the liver lobules, the only surviving cells being at the periphery of the lobules. Fatty degeneration. (For comparison)



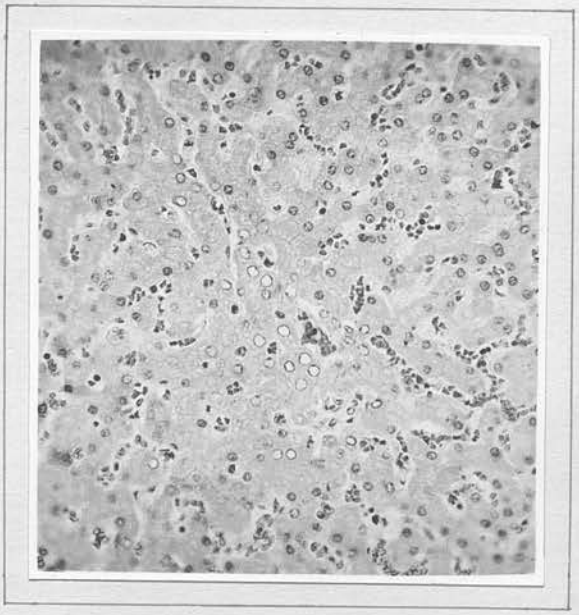
Liver x 100.

Diffuse fatty degeneration and mild necrosis, neither of which are central in distribution. Marked infiltration of the portal tracts.



Liver x 150.

Diffuse fatty degeneration and necrosis



Liver x 200

Nuclear degeneration

Liver: Pale yellow, some small deeper yellow specks on right lobe, gross appearance suggested considerable toxic degeneration. Microscopically was seen a diffuse fatty change lacking the concentration in the central zones characteristic of burns and was not as severe as expected. No central necrosis. The appearance was not that of a burns toxaemia.

Kidney: Parenchymatous degeneration and possibly some fatty degeneration present.

Suprarenal: Cortical cells slightly deficient in lipoid.

Pituitary and Hypothalamus: Apparently healthy.

Blood Culture: No growth. Culture from beneath tan - no growth. The conclusion reached was that the appearances were not those of a typical toxaemia of burns.

Comments:

This was in many ways an interesting case. Both clinically and pathologically it exhibited features which did not seem to have any other explanation than that they were due to an atypical acute toxaemia. The late onset is unique. The condition was insidious in onset and progressive in nature. A possible explanation might be that a mild toxaemia began on the 5th day but was only intermittently evident until the 10th day when it became more severe.

The biochemical findings are suggestive of toxaemia. The absence of sepsis was established and is notable.

This case illustrates the difficulty of giving a prognosis and the dangers inherent in even a minor scald under satisfactory treatment.

This was the first case of suspected toxaemia which had occurred since the introduction of the gentian violet silver nitrate method, and raised the question as to whether the course of acute toxaemia might be modified after this method as compared with the appearances found in cases occurring after

tannic /

tannic acid treatment.

CASE 20 (3.5870). H.R. Male. Aged 7 4/12 years.

Admitted 1 hour after scalds due to hot water bottle bursting in child's bed. Olive oil applied.

Extent:

Both feet, back of left leg and thigh (5 per cent. of body surface).

Points:

1. No shock on admission.

2. Tolerated local treatment well.

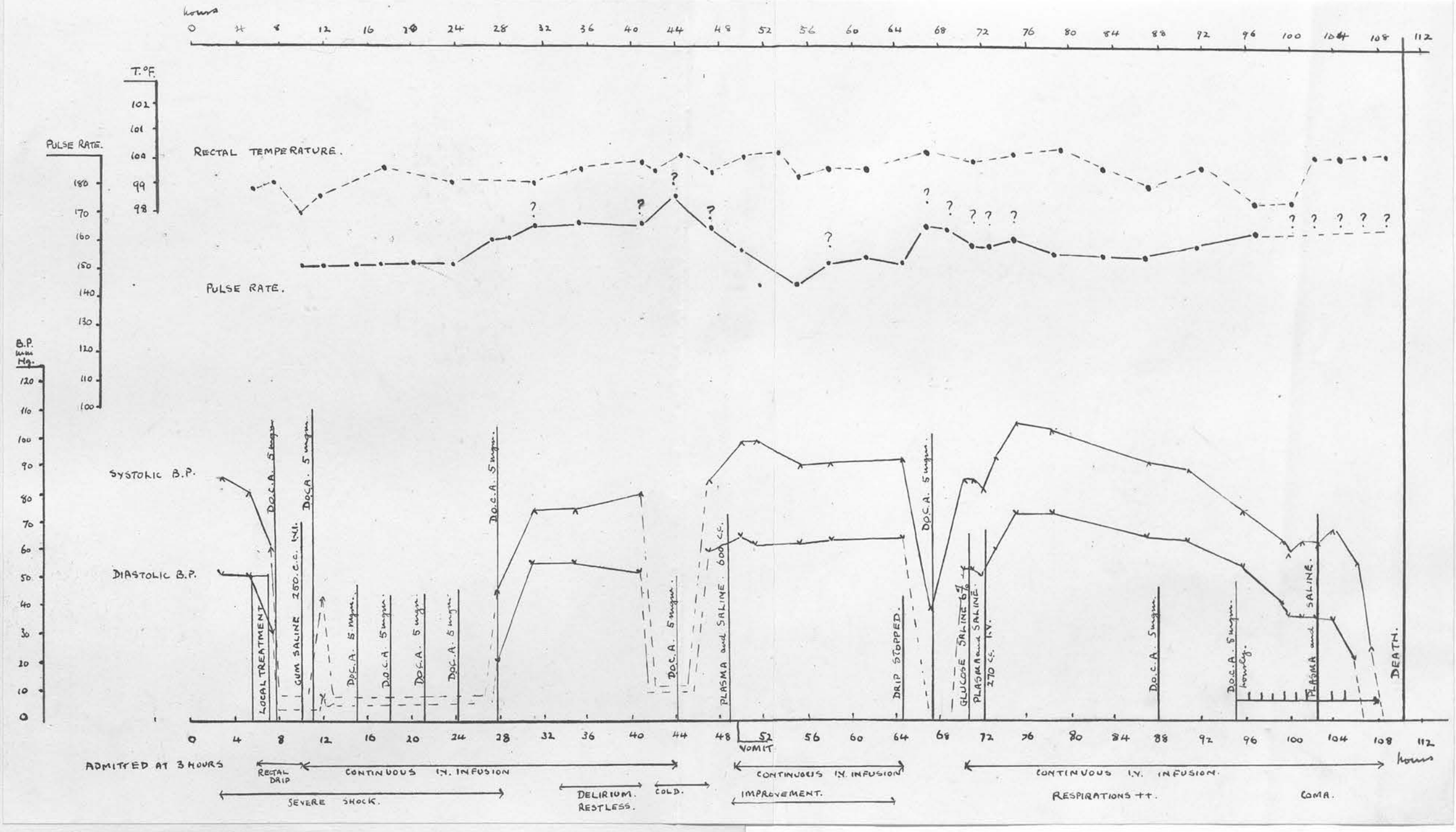
3. Toxaemia of slight degree from 25 to 90 hours indicated by clinical appearance - cyanosis, vomiting, restlessness, pallor; elevation of temperature and pulse rate, subsided without active treatment. Streptocide given prophylactically. Passed green stools on 5th day only.

4. Toes difficult to tan, oozing was persistent especially in clefts, tan separated late and raw areas were left on toes and in clefts which were dressed with flavine and paraffin.

5. Discharged on 32nd day still not completely healed; flavine dressings on and between toes.

Comments:

Mild toxaemia in small scald. An example of the difficulty often experienced in treatment of fingers and toes where skin is thick. If the fingers and toes are not separated, the tan on the apposed surfaces becomes moist and lifts. The fingers may be kept separated by wads of loosely rolled gauze.



CASE 21.

Chart of Clinical Course.

CASE 21 (3.4865). A.W. Male. Aged 4 years.

Admitted 3 hours after scalding as a result of a boiler bursting during severe frost.

Extent:

Whole of one arm and both legs, half of back, face, neck, small areas on chest (50 per cent. of body surface).

Points:

1. Very severe shock on admission after long journey by ambulance; pale, cold, cyanosed; blood pressure not taken.

2. Local treatment at 5 hours lasted $\frac{3}{4}$ hour.

3. At 8 hours saline given by rectal drip. At 11 hours condition very poor, had become progressively worse. Gum saline, 250 c.c., intravenously slowly; blood pressure could not be recorded until $12\frac{1}{2}$ hours when it was 38/?. Pulse rate could not be estimated as beats were irregular in time and force and frequently impalpable. Heart beat could not be heard.

4. Plasma saline was given at 49 hours, 600 c.c. being run in rapidly. No reaction apart from a large fluid vomit about half an hour later. Blood pressure rose in the next few hours. Plasma saline, 250 c.c., were given on each of two later occasions without reaction, being run in with a 2 feet head of pressure.

5. Progressive fall in blood pressure from about 90 hours in spite of D.O.C.A. and intravenous fluid, accompanied by a rise in temperature which was not due to an increase in the cage temperature.

6. The child never really rallied. He was comatose from the time of admission and could only be roused during a period 2 hours at 56 hours. Much of the time he lay restlessly with glassy staring eyes occasionally becoming wildly delirious, then sinking into uneasy coma again.

7. Death at 110 hours.

Post /

Post Mortem Appearances:

Head: No abnormality in skull, meninges or brain.

Lungs: All parts oedematous, emphysematous in front, congested at back. Right lower lobe, some pneumonic patches on section showing bronchitis and broncho-pneumonia.

Stomach: Contained a quantity of altered blood, no bleeding point could be found in gastric or oesophageal mucosa.

Duodenum: In the first part just beyond the pylorus there was a small submucous haemorrhage and over this the mucous membrane was eroded and a small strand of blood clot was adherent. This appeared to be the bleeding point. There was a considerable quantity of altered blood mixed with the intestinal contents as far down as the ascending colon.

Liver: No pathological change. The central degeneration and necrosis characteristic of acute toxaemia was not present.

Spleen, kidneys and suprarenals: Healthy.

Comments:

The shock present on admission was not treated except by warmth and heroin. Gum saline or plasma saline should have been given before local treatment. Rectal saline had no effect on the shock. Gum saline and D.O.C.A. produced a temporary improvement which was later consolidated by plasma saline. When the infusion was stopped at 64 hours, the pressure fell again but was restored by plasma saline. The later terminal failure was not controlled by repeated injections of D.O.C.A. or by further plasma saline. Death was due to the early broncho-pneumonia, superimposed on the effects of the scald. The presence of an early ulcer in the duodenum is interesting and rare. The duodenal ulcer, which was formerly a common feature of the stages of septic toxaemia (Curling's ulcer), was believed to be due to septic absorption; there /

there was no evidence of infection here apart from the pneumonia which was an unlikely cause. The absence of any post mortem signs of acute toxaemia was surprising in view of the clinical appearances which were very suggestive of toxaemia.

CASE 22 (3.6410). B.M. Female. 8 hours old.

Admitted 8 hours after having been scalded during an unsupervised labour by falling head first into a bucket of boiling water placed between the mother's legs.

Extent:

Scalp from forehead to occiput and to the ears on each side (5 per cent. of body surface).

Points:

1. No evidence of shock on admission.
2. On 4th day feeds were poorly taken, sometimes refused. No meconium was passed. Jaundice was marked. Tan healthy.
3. Meconium passed on 5th day. Feeds well taken, still jaundiced.
4. On 7th day refused feeds in evening, became pale and seemed cold; pulse and temperature unchanged and normal but respirations rose from 35 to 40 per minute. Died at 2.30 a.m. on 8th day.

Post Mortem Appearances:

Mildly jaundiced in skin and conjunctivae. No infection of umbilicus.

Scalp: Slight oedema under tan affecting deep tissues and periosteum under whole of scalded area, did not suggest infection.

Pleural cavities: Contained a small amount of turbid fluid and some purulent exudate was sticking to the serous surfaces.

Pericardial /

Pericardial sac: Contained turbid sticky fluid and some flakes of purulent exudate.

Lungs: Intensely congested, pneumonic consolidation of left lower lobe, less marked in right lung. No abscesses were found in either lung. On section, widespread consolidation and in one area from left lower lobe, evidence of inhalation of septic material (gastric contents or milk).

Liver: Slightly enlarged, much congested, little evidence of toxic changes. On section, little change in parenchyma. Some excess of eosinophils were present in portal tracts, a not unusual feature in a child of this age. No degeneration in cells of central parts of lobules.

Spleen: Congested.

Kidneys: Congested and bile-stained.

Bacteriological findings (post mortem):

Lung: Direct film, many coliform bacilli phagocytosed by cells. Culture B. coli.

Pleural exudate: Cells mostly mononuclear, a few coliform bacilli. Culture B. coli.

Blood culture: B. coli.

Culture from beneath tan: No organisms in direct film. Culture sterile.

Conclusions:

A genuine B. coli infection, not contamination. It may be a septicaemia or a primary B. coli pneumonia, common in the new-born. Pathological findings suggest septicaemia but source is doubtful.

Comments:

It is fairly evident that the scald was well controlled and was not the cause of death, neither did it give rise to toxaemia. There was no clinical evidence of the septicaemia and the fact that there was no rise in temperature or /

or pulse rate would seem to indicate that there was no reaction to the infection on the part of the child. The scalded area was in good condition and no growth was obtained on culture of material from beneath the tan, although the blood culture was positive.

SUMMARY and CONCLUSIONS.

1. A review has been made of 116 cases of burns and scalds treated by coagulation with 1 per cent. gentian violet and 10 per cent. silver nitrate.

2. The average number of burned and scalded children admitted to hospital per month was fairly constant until the winter months of 1939-40 when an increase occurred, the number being nearly doubled; reasons for this rise have been suggested.

3. Of 101 patients under the age of 12 years, 40 per cent. were aged 1-2 years, 67 per cent. 1-3 years and 74 per cent. under 6 years.

4. The circumstances under which the injury was sustained show that carelessness and lack of supervision on the part of parents or guardians were in the majority of cases the chief causal factors. Any attempt to reduce the number of injuries will meet with difficulties, and the only approach considered likely to yield results is by widespread propaganda and education of the public in the causes and dangers of such injuries.

5. The first aid treatment applied to the cases of this series was unsatisfactory; a scheme of instruction has been drawn up for inclusion in the propaganda suggested above.

6. /

6. Of 105 cases 39 per cent. were admitted within one hour of injury, 65 per cent. within 2 hours and 75 per cent. within 3 hours. There is still a proportion of cases which arrive at hospital only after attempts at treatment at home have been made and abandoned.

7. In 78 per cent. of the patients the extent of injury was less than 20 per cent. of the body surface; in 7 per cent. it was 50 per cent. or more. Extent of injury per se is not now as lethal a factor as formerly.

8. Initial shock was infrequent and showed no constant relation to the extent of the injury. Secondary shock was common and its severity was related to the extent of injury and the duration of local treatment. Gum saline and plasma saline had beneficial effects on the shock when given in adequate amounts. The administration of cortical hormone proved beneficial in a number of cases. In 5 cases death was due to secondary shock; in certain of these treatment was inadequate. The importance of the combination of general resuscitatory treatment with local treatment of the burn has been emphasised.

9. In 39 consecutive cases healing was complete in an average time of 19.2 days and the stay in hospital was 20 days. In some cases healing was complete in from 8 to 14 days; in others it took many weeks, the time depending on such factors as depth and site.

10. Investigation of the blood chemistry has confirmed previous observations; it has been suggested that there may be a change in the selective permeability of capillary walls in and near the burned area as a result of the injury, and that this change is at least in part responsible for the alteration in the relative amounts of certain constituents of the circulating blood.

11. /

11. The mortality rate in this series of 116 patients was 6.9 per cent. (8 deaths), which compares favourably with that in other series in which tannic acid was used as the coagulant.

12. The nursing of burns and scalds is of the greatest importance. Unremitting care and a deep interest in the problems peculiar to the injury are essential. Much of the credit for the successful treatment of these injuries by the coagulation method must be given to the nursing staff whose part is greater than in almost any other surgical condition.

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REFERENCES /

REFERENCES.

Aldrich, R.M. (1933): New Eng. Journ. Med., 208, 299.

Best, C.M. and Taylor, N.B. (1937): "The Physiological Basis of Medical Practice," London.

Clark, A.J. (1937): "Applied Pharmacology," London.

Davidson, E.C. (1925): Surg., Gynecol. and Obst., 4, 202, 221.

Davidson, E.C. (1926): Amer. Journ. Surg., 40, 114.

Davidson, E.C. (1926): Arch. of Surg., 13, 262.

Denniston, J. (1939): Lancet, ii, 1107.

de Quincey, T., "Suspira de Profundis: The Dark Interpreter," The Nonesuch Press, London, 905.

du Bois, E.F. (1924): "Basal Metabolism in Health and Disease," Philadelphia, quoted by Best and Taylor.

Fraser, Sir John (1926): "The Surgery of Childhood," I, London.

Lowdon, A.C.R., McKail, R.A., Rae, S.L., Stewart, C.P. and Wilson, W.C. (1939): Journ. Physiol., Proc. of Physiol. Soc., 116.

Mitchiner, P.H. (1935): "The Modern Treatment of Burns and Scalds," London.

Report on the Medical Treatment of Men Burned in Colliery Explosions (1933), Mines Dept., H.M. Stationery Office.

Rowntree, L.G. and Brown, G.E. (1933): "The Volume of the Blood and Plasma in Health and Disease," Philadelphia; quoted by Best & Taylor.

The Annual Reports of the Registrar-General for Scotland (1931-38): H.M. Stationery Office.

Underhill, F.P. (1930): J.A.M.A., 95, 852.

Underhill, F.P., Kapsinow, R. and Fisk, M.E. (1930): Amer. Journ. Physiol., 115, 302, 315, 325, 330, 334, 339.

Underhill, F.P., Carrington, G.L., Kapsinow, R. and Fisk, M.E. (1923): Arch. Int. Med., 32, 31.

Wilson, W.C. (1929): Spec. Rep. Series Med. Research Council, No. 141.

Wilson, W.C. (1935): Edin. Med. Journ., 42, 177.

Wilson /

Wilson, W.C. (1936): Practitioner, 136, 394.

Wilson, W.C., Rowley, G.D. and Gray, N.A. (1936): Lancet, i, 1400.

Wilson, W.C., Jeffrey, J.S., Roxborough, A.N. and Stewart, C.P. (1937): Brit. Journ. Surg., 24, 601.

Wilson, W.C., Macgregor, A.R. and Stewart, C.P. (1938): Brit. Journ. Surg., 25, 826.