The treatment of these injuries still presents many problems and the prominence given to the subject in the medical press and in lecture programmes bears witness to its importance. This is enhanced at the present time by international tension and by the likelihood that, in the event of war, the effects of ionising radiation will be added to those of thermal and blast injuries. The multiplicity of therapeutic procedures which have been advocated in recent years is evidence of the mediocrity of the results which have so far been attained and of the anxiety of those concerned with the treatment of burns to improve this state of affairs.

The objects of treatment are to save life, to prevent, minimise or rectify deformity and disfigurement, to reduce suffering and to shorten invalidism. The principal causes of failure are the inadequate early treatment of shock, the occurrence of secondary infection, failure to replace dead skin soon enough, and loss of morale from pain, protracted treatment and fear of deformity and disfigurement. Both the physical and mental aspects of the problem must receive attention if the patient is to be given the maximum chance of living a useful happy life.

Assessment of seriousness of a burn

This is a matter of clinical judgment depending on many factors, all of which have to be weighed in every case. The most important of these are:

1) Extent. The systemic reaction which is to be expected is proportional to the size of the burn. This, when expressed as a percentage of the total surface area, is of assistance in calculating the amount of fluid required to combat oligaeic shock. Berkow's table has recently been simplified by Wallace² for ease of memory by expressing the areas of the body as 9 per cent. and multiples of 9 (Fig. 1). He states that children below the age of 9 rarely need intravenous therapy with burns of less than 9 per cent. and adults with burns of less than 18 per cent.

2) Depth. The functional and cosmetic result, the duration of invalidism and the necessity for skin grafts depend on the depth of the burn. Unfortunately it may be extremely difficult to assess this accurately in the first few days after injury. The detailed classification of Dupuytren³ who recognised six levels of burning, has been abandoned, and even the war-time classification of first, second and third degrees is now often shortened into two groups called partial and total epithelial loss.
Fig. 1. Wallace's chart for assessing the surface area burned. This is expressed as 9 per cent. or in multiples of 9 per cent. for ease of memory. The fluid requirements and normal urinary output at different ages are also built up round the figure 9. The system is called by Wallace the "Rules of Nine." (See text.) Figure reproduced with Mr. Wallace's permission.

This has resulted from the recognition that the only important consideration is whether or not epithelial cells have survived the burn. Superficial burns are easily diagnosed since they either cause an erythema (1st degree) or leave unruptured blisters (superficial 2nd degree). Deep burns (3rd degree) are also easily recognised from the obvious coagulation of the full thickness of the skin. The difficult decision is between deep second degree burns in which some epithelial islands have escaped, and those at a slightly deeper level in which all have been destroyed including those round the hair follicles and sweat glands. In such cases the blisters are ruptured and the colour of the exposed dermis is the best guide. Punctate vascularisation of the papillae which show as red dots suggests the probability that some epithelium has survived whilst an opaque white surface is of more sinister import. The extent of a burn is also some guide to its depth since it is very rare for an extensive burn to be superficial throughout.

(3) Site. McIndoe has pointed out that burns in some part of the body can be followed by such crippling disabilities and deformities that
they should be regarded as serious even if they are small and apparently superficial. These areas of functional importance are the face, hands, feet, joint flexures and external genitalia. It is wise to seek expert advice in the treatment of all but the most trivial burns in these situations.

(4) **Time Since Burning.** Delay in treating a burned patient may have serious consequences both systemically and locally; systemically because the biochemical upset which commences immediately the burn is inflicted may already have progressed sufficiently to produce oligaeemic shock, and locally on account of the secondary infection which will have supervened in unfavourable circumstances.

(5) **Other injuries.** Fractures and soft tissue injuries which are frequently received with a burn may add greatly to the risks to life and to the difficulties of treatment. This is particularly noticeable when these injuries necessitate prolonged immobilisation, and due account must be taken of them when assessing the prognosis in such a case.

(6) **Age and Physical Condition.** Burns carry a greater risk in young children and old people than in young adults. They also carry a greater risk in obese subjects and in those with a history of renal, cardiovascular or pulmonary diseases. The middle-aged subject with mild hypertension and a reduced vital capacity from chronic bronchitis is likely to give much anxiety; this may be difficult for his relatives to appreciate since, under normal circumstances, he is well enough to be regarded by them as a fit man. It is extremely important, therefore, both for the patient and for the doctor to take a detailed history of previous illnesses in every case.

**CLINICAL PATTERN IN A SERIOUS BURN**

(1) **Shock**

(a) **Causation.**—This is due to loss of fluid, electrolytes and protein from the active circulation. It commences at once and rarely continues after 48 hours in an adequately treated case. Wallace has stated that the loss is maximal in the first 8 hours when half the total loss may be sustained. Despite this the reserves of the body in average health are such that the clinical manifestations of shock are often delayed for between six and twelve hours. It is of vital importance not to be misled by the patient's apparent well-being during this period and so miss the opportunity of immediate transfer to hospital for the instigation of therapy which may prevent subsequent collapse. This mistake is frequently made.

The loss is partly from the burned surface but largely into the tissue spaces in the neighbourhood of the burn, following increase of the capillary permeability. If the burn is deep enough to destroy the vascular plexus of the dermis there is a formidable destruction of red cells. This is often masked by a rise in the haemoglobin and haematocrit levels consequent upon the massive loss of fluid. If the dermal plexus is not burned there is little if any loss of red blood cells.
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As the result of loss of fluid from the circulation the blood pressure falls and the urinary output is reduced. As the circulatory crisis is resolved and the blood pressure rises urine is secreted in large quantities. This rids the body of part of the fluid administered intravenously to tide the patient over the crisis. The constitution of the urine may then reveal damage inflicted on the kidney during the phase of shock. The agent responsible for this damage is not known for certain. It may be tissue ischaemia or endogenous toxic materials elaborated at the burned site or as the result of the burn. The presence of damage is indicated by the passage of large quantities of protein and the massive loss of electrolytes. As a result the urine becomes very acid. Examination of the blood will reveal an acidosis with reduction in the amounts of sodium, potassium and protein in it. The kidney may take many weeks to recover completely but its powers of recovery are great provided treatment assists it.

In some cases the oliguria persists despite therapy and progresses to anuria. This may be due to initial delay or inadequacy in treatment, to the severity of the damage inflicted on the kidney or to pre-existing renal disease. There are probably other unknown causes as well. Bull and his co-workers have done much research into the pathology of anuria following shock. They have also emphasised the importance of recognising the condition early and treating it promptly. In their view the damage is sustained both by the glomeruli and the renal tubules. Once anuria has occurred, or oliguria shown itself to be persistent after the resolution of the cardiovascular crisis, the patient's symptoms are no longer due to shock but to uraemia. Estimation of the blood levels will confirm this by revealing a steady and dramatic rise in the blood urea and only a slight further reduction in the electrolyte levels. This slight reduction is due to continued seepage into the dressings. It affects the blood levels of the electrolytes so little, however, that injudicious feeding of these substances, orally or intravenously, may produce rapid rises when no urine is being secreted. Even in the face of such serious damage the kidney is capable of recovery in many instances with correct treatment.

Tissue anoxia is severe in burns deep enough to destroy the red blood cells and is then an important cause of generalised cellular damage. In more superficial injuries it probably does not play an important part.

Thus it is seen that the loss of fluid, electrolytes and protein occurs:

(a) by direct destruction at the time of the burn (including R.B.C. in deep burns),
(b) by subsequent surface loss at the burned site,
(c) by extra-cellular loss into the tissue spaces,
(d) by loss through the damaged kidney as the blood flow through it is restored.
The oligaemia which results from these disturbances produces the clinical manifestations of shock. Severe renal damage may lead to uraemia as the cardiovascular crisis of shock is overcome. Previous renal disease increases the likelihood of this complication.

(b) Clinical Picture.—Thirst, pallor, restlessness and anxiety are important forerunners of burn shock. The temperature falls and perspiration appears together with tachycardia and a fall in the blood pressure. At first the pulse pressure is reduced by a relative elevation of the diastolic pressure and only a slight fall in the systolic reading. Later both the systolic and diastolic pressures fall with the onset of peripheral failure. Oliguria follows. The urinary output is one of the most reliable guides to the state of burn shock and to progress in its treatment. In health the hourly output of a child of 9 should be $3 \times 9$ ml. ($=27$ ml.) and of an adult $6 \times 9$ ml. ($=54$ ml.)$^2$ As fluid is lost from the circulation haemoconcentration occurs and the haemoglobin level may rise as high as 140 per cent. in an adult and 160 per cent. in a child with corresponding rises in the haematocrit readings.

(2) Primary Toxaemia

This syndrome is rarely seen nowadays and it has been suggested that its disappearance is due to the abandonment of the local application of tannic acid. It is believed that it was due to the absorption of tannates which exerted a toxic action on the liver.$^6$ The clinical picture is unmistakable. It occurs about 100 hours after burning and starts with a fever which rises rapidly to $103^\circ$ or higher. The pulse rate is correspondingly increased and nausea and vomiting soon appear. Oliguria returns and in unfavourable cases the patient becomes jaundiced. Convulsions follow and the patient lapses into coma for some while before death ensues. Many cases have been reported in which the symptoms were less severe and recovery occurred. Lack of precise knowledge of its causation has necessitated empirical treatment, consisting for the most part of the administration of intravenous saline and cardiac stimulants.

(3) Secondary Toxaemia

This occurs about two weeks after burning. It is due to sepsis and the secondary toxic anaemia which this produces. The patient ceases to make progress, becomes apathetic and is intensely irritable. He sleeps badly, loses his appetite and tries the patience of all around him. On examination he is seen to be pale and to have lost much weight. His wounds are obviously infected and local healing seems to have stopped. Sloughs are slow to separate, granulations are pale, and growing epithelial edges become attenuated. There is a regular evening rise of temperature to about 100$^\circ$ and the mean pulse rate over twenty-four hours is increased by about 15 beats per minute. Laboratory investigations reveal a moderate leucocytosis, a severe secondary anaemia, marked hypoproteinaemia, and sometimes an electrolyte deficiency. This toxic
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state persists, unless treated, until the wounds have healed; this takes a long while under such adverse conditions. Foresight in the management of the case can usually prevent its onset and prompt energetic treatment can dispel it quickly.

TREATMENT

First Aid

Treatment at the site of the accident is simple and it is important to resist the attempts of onlookers to anoint the victim with their favourite salve. Burned tissues and charred clothing are initially sterile but are readily contaminated by these well-meant efforts of friends. Ideally the burns should be wrapped in a sterile cover. The Bunyan oiled-silk envelope is the best first-aid cover, being easy to apply, and light. Failing this, a clean towel or pillowcase is satisfactory. The patient’s most urgent needs are reassurance and sedation. Morphia is life-saving and is well tolerated in large doses by children and adults after burning. It should be given in full therapeutic doses which can be repeated as often as necessary to keep the victim out of pain. There is no risk of dangerous over-dosage provided the respiration rate is not allowed to drop below 14 per minute. The prompt recognition of a serious burn and insistence on immediate transfer to hospital is the greatest first-aid service a doctor can render his patient. If the journey is long, an intravenous infusion of half-strength saline should be set up before it is undertaken; should it be so long that a second litre of fluid is needed blood or plasma should be used. The immobilisation of burned limbs by binding them loosely to splints over bulky cotton wool packing helps to diminish pain during transit. A doctor should travel with the patient in all serious cases.

Oligaemic Shock

(A) Sedatives. Restlessness and apprehension need to be overcome and pain relieved continuously and effectively in order to control incipient and established shock. Sedative drugs are also needed to reduce the oxygen requirements as much as possible, by putting the patient at complete rest and inducing sleep. Morphia is the most useful drug for these purposes at first, but later it is often found that soluble phenobarbitone, gr. 3 intramuscularly or in small doses by mouth, night and morning, is better. It is wise not to continue the administration of morphia longer than is essential. Sleep can be induced with a basal narcotic of which soneryl gr. 3 is very effective. Given with codeine to reduce pain this ensures a good night’s rest after the acute pain of the first few days has abated.

(B) Warmth. The extreme cold of the initial stages of burn shock add to the patient’s collapsed state and should be combated. But it is easy to overheat him, so increasing the oligaemia. The patient needs warming but not heating. No warmth should be applied to the burned
areas. Ideally, chilling should be overcome by increasing the temperature of the room and by adequate but light bed coverings. If artificial warmth is needed, protected hot water bottles are best. An electric light cradle delivers too much concentrated heat unless it is very carefully controlled.

(C) Fluid Replacement. This is an urgent requirement in all serious burns, and the importance of initiating it at once has already been stressed. The important questions to decide are how much fluid is needed, its nature, the rate at which it should be given and when it should be discontinued. Under ideal conditions all these questions can be accurately answered by laboratory tests frequently repeated. Often, however, these are not available and reliance must be placed upon clinical judgment aided only by routine urine tests and an occasional estimation of the haemoconcentration. The most important clinical pointers in assessing progress are the volume of urine passed per hour, the blood pressure, the disappearance or increase of restlessness and thirst, and the degree of haemoconcentration.

The belief that the maximum rate of loss of fluid occurs in the first few hours after burning implies the necessity for the replacement of a large quantity within the first 24 hours. The knowledge that the onset of diuresis indicates returning normality of capillary permeability and resorption of much fluid from the tissue spaces, demands curtailment of the fluid intake when the urinary output starts to rise. This happens after about 48 hours with adequate initial treatment. Intravenous therapy should then be discontinued and the oral intake limited to two pints in 24 hours.

(1) Amount of Fluid Needed.—Many complicated tables have been worked out to determine this figure, but a satisfactory simple formula for the total fluid requirement by all routes is:

3 ml. per kilogram body-weight, per 1 per cent. surface area burned in the first 24 hours.

2 ml. per kilogram body-weight, per 1 per cent. surface area burned in the second 24 hours.

(kg. = 2.2 lb. 1 stone = 6.3 kg.)

In all except the most extensive burns and the heaviest subjects it will be found that this total does not exceed nine litres in the first 24 hours and 6 litres in the second. Of these totals approximately 2 litres can be taken by mouth.

Wallace\(^2\) has worked out a table for burns involving 18 per cent. or more of the body surface which he has called the Rule of Nine.

Intravenously.—

Adults (aged 18 or over) :

1 litre plasma \{ per 9 per cent. body area burned in 48 hours.  
1 litre saline \}  

Maximum—12 litres.
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Child (9 years):—

\[
\frac{1}{2} \text{litre plasma /} \frac{1}{4} \text{litre saline per} \text{9 per cent. body area burned in 48 hours.}
\]

Maximum—6 litres.

**Orally.**—

Adult:—9 × 6 ml./kg. body-weight.

Child:—9 × 9 ml./kg. body-weight.

(2) **Nature of Fluid Given.**—Intravenously. Wallace has suggested that this should be varied with the depth of the burn depending upon whether red blood cells have been lost by the destruction of the vascular plexus of the dermis. In deep burns he advocates a ratio of:—

Plasma 1.

Saline 1.

Blood 2.

and in superficial burns of:—

Plasma 2.

Saline 2.

Blood 0.

This is a useful working rule but a more accurate assessment of the precise requirements can be made by biochemical methods. If an acidosis is developing and the sodium level is correspondingly low, the substitution of 1/6 Molecular Sodium Lactate for the plasma is beneficial. This can be continued until the alkali reserve and blood sodium levels return to normal.

**Orally.**—Glucose and water is the standard preparation for oral feeds and meets the metabolic requirements satisfactorily. There is no objection, however, to substituting milk or any other fluid the patient fancies provided it carries the necessary calorific value. The danger of acidosis in serious burns is great enough to warrant the routine addition of a teaspoonful of bicarbonate of soda to the feeds at three-hourly intervals. If given more often than this it may induce nausea or precipitate vomiting. The need for bicarbonate is obviously urgent if the reaction of the urine is markedly acid or if the respiration has become stertorous or there are other signs of severe acidosis.

(3) **Rate of Administration of Fluid.**—Modern teaching lays stress on the importance of giving a large amount of fluid quickly. Wallace advocates that half the calculated 48-hour requirement should be given in the first 8 hours and a further quarter in the next 16 hours. The remaining quarter is then given in the second 24 hours. Obviously the rapidity of infusion will be subordinate to the individual's ability to accept the fluid without detriment to the heart, and these figures will have to be modified in many patients whose hearts have been previously damaged or are in danger of embarrassment.

(4) **When to stop Intravenous Therapy.**—This should be discontinued when diuresis commences. If it is continued during this phase there is
a danger of over-loading the circulatory system and water-logging the tissues. Pulmonary oedema will then occur and pneumonia will follow. The massive loss of electrolytes which may occur with the diuresis demands the administration of an adequate quantity by mouth. Bull advocates fruit to ensure sufficient potassium intake. Ideally frequent estimations of the blood sodium and potassium levels should be made and mineral intake adjusted accordingly. In practice these adjust themselves readily provided it is remembered that the levels are likely to be low in this phase and the requirement is therefore large.

(5) Changes necessary in treatment upon the occurrence of uraemia.

Bull and his co-workers have pointed out that once it is clear that anuria is persisting despite the resolution of the cardiovascular crisis, the amount and composition of the fluid administered will determine whether the patient survives. The fluid requirement is restricted to the amount lost by extra renal routes and rarely exceeds one litre in 24 hours. Protein is harmful at this stage despite the losses resulting from the burn since the end products of its metabolism are retained. Loss of electrolytes is negligible in the absence of vomiting and Bull therefore recommends their exclusion from the diet at this stage. A calorific intake of 2,500 C. per 24 hours is provided by carbohydrate and fat, using glucose 400 g. and fat 100 g. This is made up in one litre of water with acacia to emulsify. Vitamins may be added. Since this mixture is nauseating, Bull recommends that it should be administered as a continuous intragastric drip feed through an indwelling plastic catheter passed through the nose. If the patient vomits, the vomit is filtered and fed back through the tube so avoiding the loss of electrolytes and essential calorific food.

This régime is continued until diuresis occurs when changes in the composition of the diet are made in accordance with the renal loss. During the anuric phase transperitoneal dialysis may be necessary if the level of urea in the blood becomes dangerously high. Its employment, however, upsets the electrolyte levels which have to be checked and adjusted afterwards. Moreover, its use is not devoid of risk from infection. It is, therefore, best avoided whenever possible.

(D) A.C.T.H. (Adreno-corticotrophic hormone). It has been known for a long while that adrenal extracts are on occasion helpful in tiding a patient over the initial crisis of a very severe burn. With the preparation of A.C.T.H. it was hoped that by stimulating the adrenal cortex this potent hormone would minimise shock by limiting excessive capillary permeability and reducing histamine synthesis. There is some evidence that it does, but its use in burns is still experimental and Clarkson reports personal communications from Moore and Converse indicating adverse side effects when it is discontinued. Its suspected tendency to slow the rate of healing when given in large doses also demands cautious administration. There are, however, many ways in which it may help the patient. The protection it affords the liver is one of the most
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important and it seems likely that it will ultimately prove to be of definite value in selected cases. The dosage at present recommended is 25 milligrammes six-hourly.

(E) Oxygen. This is only of assistance in oligoemic shock when the patient is cyanosed and when its exhibition abolishes or lessens this cyanosis. In many cases the cyanosis is due to failure of the peripheral circulation; oxygen is then of little value. Indeed in the majority of cases of cyanosis oxygen only helps when this cyanosis is due to a pulmonary or cardiac complication. In infants it is best administered in an oxygen tent, but this usually frightens an adult whose equanimity is already disturbed, and reliance has to be placed on intranasal catheters or a B.L.B. mask when the face is not burned.

Chemotherapy and Antibiotics

Reliance is placed nowadays almost entirely on the systemic administration of these drugs in the early stages of the treatment of burns. When sloughs are separating from deep burns local applications are sometimes helpful, but the rate of absorption appears to be erratic and, with the sulphonamides, can be dangerous when it is very rapid.

Most burnt surfaces are initially sterile unless the circumstances of the accident are such as to cause contamination. It is important, therefore, to make every endeavour to maintain this sterility. The early administration of an intramuscular injection of penicillin is one of the best ways of doing so. It should be given as soon as possible. The initial dose should be 600,000 units of procaine penicillin, and this should be repeated night and morning. If infection, or a systemic reaction suggesting it, occurs, immediate bacteriological investigation should be undertaken and antibiotic sensitivity tests made. As soon as the results are known the appropriate drug is given and, in the meantime, chloramphenicol 0.5 grammes is administered six-hourly by mouth in addition to the penicillin.

Secondary Toxaemia

The tendency for this condition to develop can be greatly diminished by careful attention to the prevention and control of infection with chemotherapy and correct local treatment, and by giving a diet with a sufficiently high calorific value and a full vitamin complement.

The calorific requirement after a serious burn is enormous and the need should be supplied as soon as diuresis indicates recovery from oligoemic shock. Blocker recommends an intragastric drip feed through a plastic catheter. This is passed through the nose and connected to an automatic pump regulated to deliver 2 ml. of a highly concentrated mixture per minute. This is made up of milk, eggs, dextrimaltose and protolysate. The formula provides protein 150 g., carbohydrate 340 g., and fat 60 g. per litre with a total calorific value of 2,500 C. He advocates the administration of 2 litres per 24 hours in addition to the ordinary
diet taken by mouth. This supplementary feeding is continued for
ten days or more and contributes greatly to the patient's well being.
It prevents wasting almost entirely.

Once secondary toxaemia has occurred small blood transfusions
(300 ml.) overcome it speedily. They should be repeated at intervals
of 3 days until the blood count is normal. At the same time approp-
riate local treatment must be instigated to overcome infection.
Sedatives, reassurance and patient nursing are also important.

Local Treatment

This has been the subject of controversy for many years and a review
of the literature demonstrates the number and variety of the remedies
which have been advocated. In the last fifty years or more, however,
one can discern the evolution of a reasoned plan of treatment in those
methods which have enjoyed widespread popularity in their day. In
1887, Copeland\textsuperscript{10} recommended the treatment of burns by exposure
in order to allow a dry crust to form on them. This method was
generally accepted in many parts of the world during the succeeding
twenty years; it gradually fell into disrepute because of the lethal
consequences of the occurrence of gross sepsis in deep and extensive
burns. It was followed by the use of a wide variety of medicaments,
none of which was outstandingly successful until Davidson\textsuperscript{11}
introduced tannic acid in 1925. This substituted an artificial eschar for the physio-
logical crust of exposure. The speed with which the eschar formed,
its durability and thickness were thought likely to prove an efficient
barrier to subsequent penetration by infecting organisms. They usually
were, but the thickness of the eschar prevented the escape of pus in those
cases in which tannic acid had been inadvertently applied to surfaces
already contaminated. This was a serious disadvantage. Another
was the length of time it took for the carapace to separate in deep burns,
since this resulted in permanent loss of function in joints and tendons.
Later, the suspicion that tannates were absorbed and damaged the liver,
added yet another disadvantage to the method.

By 1933 Aldrich\textsuperscript{12} had endeavoured to provide a compromise by the
introduction of ana line dyes which produced a more permeable crust
than tannic acid and at the same time exerted some bacteriostatic action
on a wide range of likely contaminants. This was partially successful
and the method was becoming popular when, in 1938, the sulphonamide
drugs became available.

The pleading of the plastic surgeon for early replacement of dead skin
in order to preserve function, gained reader listeners with the availability
of the sulphonamides and penicillin to hasten the preparation of the
recipient areas. To these ends, the policy most widely adopted in the
last war was to try to hasten the separation of the sloughs by repeated
saline washings, and at the same time to control infection by chem-
otherapy. The burns were covered with bulky absorptive dressings between
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treatments. The procedure became known as “open” therapy. It represented a considerable advance in several directions on anything which had been attempted before for deep burns, and is still the method of choice under some circumstances. It provides free movement of joints and tendons throughout, and helps to shorten the time needed to prepare septic burned surfaces for skin grafting. But secondary infection is extremely difficult if not impossible to avoid. Its occurrence can be disastrous when it leads to the death of partially damaged cells since it thereby increases the depth of destruction. Infected granulations then grow up despite the use of pressure dressings and greatly prolong treatment. It is particularly difficult to prevent cross infection in an open ward or a burn unit. Colebrook\textsuperscript{13} and Bourdillon\textsuperscript{14} have proved this conclusively and regard the danger serious enough to warrant the setting up of special dressing-rooms through which “clean”, filtered air is continuously passed.

Towards the end of the war Trueta\textsuperscript{15} and others advocated covering the burns with a plaster of Paris cast after preliminary cleansing in order to minimise the risks of cross infection. Not only did it do this, but it allowed the burned tissue to dry unless it was already infected. In this way it brought back, unwittingly perhaps, one of the principles of the exposure method of the nineteenth century.

It remained for Wallace, Truman Blocker and Pulaski\textsuperscript{16} to recommend more recently a full return to the exposure technique. To Wallace goes the credit of reminding us that light, dessication and cooling are three of the most potent agents which can be ranged against micro-organisms, and that the exposure method provides them all. Moreover, an additional advantage of drying is the reduction in fluid loss which it effects. The principles underlying the method advocated by Copeland in 1886 were sound, and reversion to his technique has been made practicable by the introduction of the antibiotic drugs, and by the operative and anaesthetic advances which have enabled the plastic surgeon to excise and replace dead tissue instead of waiting for healing by fibrosis.

Experience of the treatment of burns by exposure is still very limited within living memory, and the passage of time will no doubt permit a more critical assessment of its value. But it is already sufficiently well tried for some advantages over open therapy to have been demonstrated. Shock appears to be less severe and more quickly controlled. The patients experience less pain and lose less weight. They are fitter and happier throughout their invalidism. Anaemia is not so severe a problem since sepsis is largely avoided. Cellular destruction is limited to that due to the initial burn; it is surprising how much survives even in apparently severe cases if a dry crust is obtained from the start and maintained until it flakes away. Excision of full thickness losses and their replacement with grafts is attended by a smaller risk of failure
from sepsis than when the wound is weeping with an infected exudate. Recovery is therefore likely to be quicker.

Experience has, however, brought to light certain possible limitations of the method. Exposure in this country demands the maintenance of an adequate temperature in the ward despite ventilation. It also demands conditions stable enough to make it reasonably certain that the patient will remain in one institution throughout treatment. The difficulties, therefore, of applying the method to large numbers of casualties in wartime might be formidable. It has been suggested that these could be met by covering the burns initially with dressings impregnated with a chemotherapeutic paste and leaving them until the victim has reached a base hospital. This should not prevent adequate drying of the wounds later when the dressings are removed.

The treatment of encircling burns of the trunk by exposure also presents difficulties since it is very difficult to maintain a crust in the presence of pressure. Frequent change of position is a possible solution but is very tedious and not entirely successful. The writer has recently seen a completely satisfactory solution in Truman Blocker’s unit by sitting the patients up in a chair for long periods from the third or fourth day after burning. These were patients very severely burned by explosions in the oilfields (60 per cent. surface area or even more). They appeared to be comfortable and astonishingly well, but one has to remember that the climate in Texas is subtropical.

Burns several days old and already septic when received might also be thought to be unsuitable for treatment by exposure. They are certainly more difficult, but Wallace has used the method successfully on a number of such cases, including children. He recommends the repeated removal of heaped-up crusts under which pus is concealed, whereupon a flat clean crust ultimately forms. The topical application of a light dusting with chemotherapeutic powder accelerates this when the organisms are sensitive. Their sensitivity to a wide range of antibiotics should, therefore, be tested as soon as possible.

The only difficult problem encountered by the writer, in an admittedly small experience of the surgical management of patients by exposure, is to decide just when to excise the crust and graft the wound. It might be thought that the crust would always have separated within a fortnight if viable epithelium was present, and it is true that most of it does. But not infrequently postponement of operation for a further week sees complete separation of the remainder and healing with epithelium sufficiently free of scar to be cosmetically and functionally acceptable. This end is particularly desirable, since otherwise, one finds oneself exciting several small isolated areas of crust and making a patchwork graft. If, therefore, it is possible to lift small pieces from the edges of-remaining crusts each day during the third week, it is better, in my opinion, to postpone operation until after the twenty-first day. But if the main mass of crust is still firmly adherent at the end of the second week, it indicates
the presence of a large area of full thickness loss and there is no advantage in further delay.

Technique.—The only two procedures of which space allows description are absorptive dressings and exposure. It is difficult to imagine a burn which is not best dealt with by one or the other or a combination of these two.

Absorptive dressings should consist of bulky woollen packs applied over close mesh gauze and held in position by firm (not tight) crepe bandages. The wool must be replaced before it becomes sodden. These dressings should be used if the journey to the burn centre is likely to take long. They may also be used if gross painful oedema develops in an extremity during the first forty-eight hours of exposure and is unrelieved by elevation. On rare occasions they are also called for when serum loss from an exposed burn appears to be causing difficulty in controlling shock.

The addition of a chemotherapeutic paste is of value if it is desired to treat burns already septic with absorptive dressings instead of by exposure. The frequency with which the dressings have to be renewed is governed by the amount of discharge. They must not be allowed to become sodden. The chemotherapeutic constitution of the paste is determined by the sensitivity of the organisms. The base should be water soluble; lanette wax is satisfactory.

Those who are accustomed to the strenuous programme of routine dressings demanded by open treatment, will be astonished how little time it takes to attend to the burns of a patient treated by exposure. If the burns are clean and the patient received within a few hours of the accident, no cleansing is necessary. First-aid covers should be left until intravenous therapy has been started and shock controlled. They can then be removed under sedation with morphia or a general anaesthetic and the patient lifted on to a clean sheet. The unburned parts are covered and the burns left exposed. Where large areas are involved a covering sheet may be needed to keep the patient warm until the danger from shock has passed. It can usually be held away from contact with the body by suspending it as a tent over a cord passed between upright transfusion arms fixed to the sides of the bed. With adequate heating of the room even this may be unnecessary.

Dirty burns are cleansed by gentle swabbing with cetavlon 1 per cent. in the same way as for open treatment. Anaesthesia is often unnecessary but should be used for children to avoid fright and in apprehensive adults. In my opinion it is best not to cut away unruptured blisters at this stage. Their presence indicates a comparatively superficial burn from which rapid healing can be expected. The fluid in them is usually reabsorbed in about five days and the unbroken epithelium helps to prevent the loss of serum. Occasionally, however, they are so large that they will obviously rupture within a few hours. Those over movable parts are particularly likely to do so. These should be snipped and the cover cut off while the patient is sedated. The loose epithelium
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from ruptured blisters should always be removed. In the writer's opinion four-hourly spraying with penicillin powder hastens crust formation, but some authorities do not agree. The length of time which crusts take to form, varies considerably. In most cases they form within twenty-four hours, but on occasion they take much longer. If crust formation is slow and serum loss continues to be prolific, it is, in my opinion, advisable to apply temporary absorptive dressings to the most troublesome areas if any difficulty is experienced in controlling shock. In the absence of this difficulty, however, the loss itself need cause no alarm. It will be necessary to sedate the patient deeply or anaesthetise him to remove these dressings when shock is controlled.

The position of the patient in bed is determined by the site of the burn. It is designed to keep pressure off the area and to elevate extremities in the early stages to minimise oedema. Nursing care will involve frequent minor alterations in position to keep the patient comfortable and to protect the elderly from hypostatic pneumonia. No retention apparatus should be needed except the gallows splint in young children with perineal burns as advocated by Wallace; this is useful since it keeps the area from being soiled and relieves it from pressure. It is very well tolerated.

Reliance for prevention of infection is placed on parenterally administered chemotherapy and antibiotics. Penicillin should, therefore, be given regularly in large doses, and sulphonamides or chloramphenicol added, as sensitivity tests indicate, in septic burns or if infection threatens at any place in a case hitherto clean (see page 123).

Liberal sedation is needed until shock is controlled, but afterwards the patient's comparative comfort is reflected by the small quantity required. Morphia is the best drug to use during the initial phase and large doses are well tolerated. Later codeine and a barbiturate sleeping draught should suffice.

I wish to record my thanks to Mr. A. B. Wallace for permission to refer extensively to his recent paper, and to Professor M. L. Rosenheim for reading and criticising the relevant parts of this paper from the biochemical standpoint.

REFERENCES

3 Dupuytren, G. (1832) Leçons Orales de Clinique chirurgicale 1, 413.
8 Clarkson, P. (1951) Lancet 1, 460.
9 Blocker, T. J. (1951) Lancet 1, 498.