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The Emergency of burns: with special emphasis on burn shock

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THE EMERGENCY OF BURNS
WITH SPECIAL EMPHASIS ON BURN SHOCK

BY
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THE EMERGENCY OF BURNS
WITH SPECIAL EMPHASIS ON BURN SHOCK

Introduction

"It is said in Grecian mythology that man was the last race created, and in consequence thereof was the most poorly endowed with physical gifts. So Prometheus stole fire from the hearthstone of the gods on Mount Olympus and bestowed it as a gift which would set man apart from all other animals. And so it has. But the sword with which civilization was founded is two-edged, and since time immemorial the followers of Aesculapius have sought to bring relief to those luckless mortals who have felt its bite." (4)

Man has known of and suffered from thermal burns from the day he first knew fire, and the tale of burns stems from that day. Fire has been one of the most useful of all the tools of man, but it may be at the same time his most dangerous enemy. "Burns are, of all accidents, not only the most painful and agonizing, but they frequently condemn the patient to one of the most horrible and repulsive mutilations it is possible for man to have." (49)

Severe burns have come to be one of the more common and, therefore, one of the more

In the great industries, man is exposed to injury by fire in the course of his daily work to an extent that
was not possible previously. Thus, the management of burns has come to occupy an important place in industrial medicine. The great strides that have been made toward a better understanding of the burn phenomena have been made only in comparatively recent years, and the impetus could only have been that physicians are called upon to deal with these injuries more than previously. The high mortality rates of severe burns constituted a challenge to the medical profession. As with any morbid condition of the body, the treatment could have no rational background until the pathology was understood, and while the whole story of the pathology and pathological physiology of burns is not yet written, we have come far toward a better understanding of it and with our present day knowledge we are able to institute more adequate therapy.

The present war, with its concomitant injuries by burns, has added an additional stimulus to the study of burns. The war will undoubtedly bring about a better knowledge on this subject, and, without doubt, at the time that this paper is being written, new pages are being added to our aggregate knowledge. Out of the chaos will emerge a better understanding that will benefit future generations.

Burns are always an emergency, and the
is called upon to deal with them as such. It is the emergency aspect of burns which will be dealt with in this paper—the burn syndrome in the first few days following injury. The later manifestations and problems—the complications and plastic repair will not be considered as they are not a part of the emergency of burns.

So, this paper will concern itself with the sequence of events that take place during the first few days following a severe burn.

At this point, it is necessary to elucidate upon certain terminology used in the discussion of his sequence of events. In going over the literature, it is found that there is a discrepancy between the British and the American terminology. The British authors describe four stages: primary shock, secondary shock, acute toxemia and septic toxemia. Primary shock occurring at, or within minutes of the injury and terminating quickly; secondary shock occurring from two to ten hours following the injury and being identical in nature to surgical shock from other causes; acute toxemia occurring at about the twelfth to eighteenth hour following injury and reaching its maximum around the seventy-second hour. Septic toxemia is a late suppurative stage.

In the American terminology, the term primary shock
is used to denote not only the immediate syncope following injury (British primary shock) but also the stage of surgical shock (British secondary shock). In our terminology, burn-shock itself is known as secondary shock (acute toxemia in British parlance). The stage of septic toxemia is in agreement by both British and American authors.

It is of the stage in this sequence that is unique to burns alone--secondary shock, or as the British classify it, acute toxemia--that greatest emphasis will be placed in this paper. It is over the etiology of this stage that the controversy has raged. The other stages will be mentioned for continuity and completeness. The stage of surgical shock, of course, is the subject for controversy also but only a cursory discussion of the generally accepted theories will be given.

The local pathology of burns, together with necropsy finding and the blood and urine changes will be given to serve as a background for the physiological pathology and the theories that have been evolved to explain the mystery of burns--burn-shock.

In the section on local treatment, no attempt at a complete survey of all the treatments that have been used will be made. The generally accepted methods in use at the present time will be described. Since
adequate local treatment has only been developed in the last twenty years, only this will be discussed. The methods used prior to this are myriad and a complete survey would be beyond the scope of this paper.
Historical

The treatment of the ancients, as well as the accepted treatment up until comparatively recent times, was largely symptomatic, as is the case with any little understood condition. Up until that time the treatment was directed only toward the alleviation of pain, mostly accomplished by the use of oils and salves, and the treatment of physicians up to that time was not far away from the lard and aromatic oils of Hippocrates.

While the ancients accomplished relief from pain by the use of various oily substances, it is interesting to note some of their formulae for the compounding of mixtures to treat burns. Pack and Davis (41) describe the salve of Paracelsus as being made of "the fat of very old wild hogs and bears heated half an hour in red wine, then dropped into cold water, which was next skimmed and the fat rubbed up with roasted angle worms and moss from the skull of a person hung, scraped off during the increase of the moon, to which were added bloodstone, and the dried brain of the wild hog, red sandalwood and a portion of a genuine mummy." While at first glance this would seem barbaric and smacking of witchcraft, still when examined it will be seen that it is an oil and as far as the patient is concerned, would make him as comfortable as more modern treatment with oils and salves, although infections from such messes
must have given a high mortality rate from septic toxemia.

The first radical departure from the symptomatic treatment came in 1867, when Pirrie, of Aberdeen, advocated the use of phenol after hearing Lord Lister read a paper on its antiseptic qualities. Morris, in this country, in 1882 described the value of phenol when used as a primary dressing. Up until the present century, almost every burn was immediately smeared with some sort of grease before the patient was taken to the physician, thus making primary antisepsis an impossibility. And up until this time, with the physicians themselves, surgical cleanliness did not receive proper endorsement. (9)

The next great step forward in the proper handling of burns and the burned patient came when it was realized that the burned patient must be treated just as energetically as the local lesion itself. The first to urge this was Parker in 1884, but stress was not laid on this phase until around 1880. The significance of the observations of Cumin in 1823 and of Baraduc in 1862 relative to the increased viscosity of the blood in extensive burns did not receive its just due until the present century. (9) Now the local lesion and the patient are treated with equal vigor. In fact, at the
present time, local treatment is subsident to the
general treatment. In fact, Trusler, Egbert and Wil­
liams state in their paper published in 1939 that "No
local application can be expected to save life after a
large burn" (51) The local treatment of burns has come
to be of importance only in that it has an effect upon
the general effect, which is the fatal factor in burns.

So, the management of burns has developed along
certain lines to reach its present state. The guiding
light of therapy has been the gradually evolving and
as yet, incomplete understanding of the effect of burns
upon the body.
Etiology and Incidence

"A burn is an injury inflicted upon the body by a degree of heat higher than is compatible with the healthy action in the part affected." There are two types of thermal trauma: Burns that are produced by dry heat, and scalds by moist heat. Burns can be caused by any body that radiates much heat. This is commonly due to proximity to, or direct contact with flame or heated solid bodies, super-heated air, gas explosions and inflammable liquids. Scalds are produced by the action of boiling water, or other liquids, super-heated steam and molten metals. (42) According to Wallace (57) however, no distinction should be made between burns and scalds, since pathologically they are the same and the treatment is identical. So, in this paper, no attempt will be made to differentiate between the two, and the term "burn" will be applied to both.

G. T. Pack (43) sums up the incidence of burns well in the following quotation. "About forty-five per cent of deaths from burns occur from birth through the fifth year. Burns are quite common and severe in instances where tactile sensations are subnormal or absent, as in tabes dorsalis, acute alcoholism, paralysis, coma of various types, and the post-anesthetic state. Burns kill more females than males. Female attire is an important factor in increasing the hazard. Only during
late maturity, from the age of thirty-five to fifty-five, when large numbers of men are engaged in industries subjecting them to the hazard of burns and scalds, do as many males die from this cause as do females.

Burns stand fourth among the causes of accidental death, being out-ranked only by automobile fatalities, falls and drownings."
Pathology

A consideration of the pathology of burns immediately resolves itself into two fields—the local and the general. The cause of death is due to the general factor, but the general factor is precipitated by the local factor. The local pathology is well understood and agreed upon. Necropsy findings are also well established. Depending upon the extent and severity of the burn there are very definite lesions produced in almost every organ of the body, but the pathology of the internal organs is not by any means characteristic of burns. It has been noted that the changes were similar to those found in the acute infectious diseases of the skin. (25)

In this section, the local pathology of burns will be described, followed by an outline of necropsy findings and a description of the blood and urine changes in a severely burned patient. This is given so that it may serve as a background for the pathological physiology and for the theories that have been evolved to explain burn-shock.

Local Pathology

The variations in the character of the local lesion in burns depend upon a number of factors, the more important of which are, the intensity, character, and duration of the heat, as well as the extent of the lesions. According to the local changes observed, burns have
been divided into degrees. These are of course, arbitrary and not clearly defined in actual clinical work. For a description of Dupuytren's six degrees of burns, the following excerpt from Gunn and Hillsman (25) is given, the source being the monograph of Pack and Davis (41).

First Degree: This consists of a simple erythema with vascular reactions similar to that of any simple inflammation due to the action of an irritant. It is essentially a reflex and exudative reaction. The reflex phase consists of contraction followed by dilation of the arterioles and venules, resulting in an increased flow of blood to the affected area and a local stagnation of the current. This naturally causes the redness and increased heat invariably associated with this type of burn. The exudative reaction is explained by an increased capillary permeability and consists of filtration of plasma into the tissue spaces, the migration of leukocytes, and the diapedesis of red blood cells. Within a few days the outer layer of the epidermis separates and peels off.

Second Degree: This is essentially the degree of vesication. The epidermis undergoes true coagulation necrosis. By virtue of the increased capillary permeability there is an exudation of serous fluid and filtration of leukocytes from the tips of the papillae,
resulting in the formation of blebs or bullae. In mild cases the exudation occurs within the epidermis and leaves the basal cell layer intact, thus forming the bleb. In the more severe cases the fluid collects in larger amounts beneath the full thickness of the epidermis, resulting in the larger and more serious bullae. In the very severe cases the fluid may resemble plasma and in such cases organization of the fibrinous exudate is more likely to follow than absorption. Such blisters should be evacuated before organization takes place.

Third Degree: In this the epidermis is entirely destroyed as is part of the corium. Its distinguishing factor is that the interpapillary processes remain intact. The papillae appear as a reticular frame-work containing serum, bits of persistent living epithelium, leukocytes and fibrin. These papillae show as red points on a white background. Two things should be noted about this type of burn. First, the exposed nerve ends in the interpapillary processes remain intact and as a consequence this is the burn accompanied by most severe pain. Secondly, the sebaceous glands, the hair follicles and the sweat glands are intact and will regenerate new epithelium providing infection does not destroy their epithelial cell lining.

Fourth Degree: In this there is destruction of
the entire thickness of the integument and complete
disorganization of the skin involved. If the trauma­
tizing factor is dry heat this area will vary from
brown to black in color, be insensitive and leathery to
touch. If on the contrary, moist heat is the agent, the
affected area will be white, insensitive and finger
pressure will not induce the usual color changes.
Surrounding this zone will be areas of lesser degree
burns, shading off from the third degree to the simple
hyperemia of the first degree. An acute inflammatory
process starts very early and the eschar retracts
leaving a groove between the dead and living tissues.
Sloughing of the dead tissue begins and is completed
normally within two weeks. At the same time proliferation
occurs. Fibrinous exudation upon the surface exerts a
chemotactic action upon the new tissue cells. The cap­
illaries proliferate tufts of endothelial cells accom­
panied by new growth of fibroblasts, to grow along the
fibrinous framework after the manner of healing by
secondary intention. The endothelial cells hollow out
to form arches of new capillaries giving nourishment to
the growing tissue. Scarring and contracture deformities
result.

Fifth Degree: This type of burn varies only from
the fourth degree in that the underlying muscles are
involved. The resulting scar is, as a consequence, more deforming and may cause great functional impairment. The scar has great tendency to break down and ulcerate.

Sixth Degree: In this degree the tissues are carbonized and heat may even fracture the underlying bone. It is usually seen in the very severe type of burn involving the digits.

This classification of Dupuytren is considered as being impractical by many workers inasmuch as it cannot be readily applied clinically. A classification of only three is used by many. In the first there is simple erythema of the affected part. The second degree causes both erythema and destruction of the epidermis, with or without vesication. The third causes destruction of at least the whole thickness of the skin, but may be deeper. (57)

The extent of the lesions is also of importance. Dr. S. G. Berkow has perfected a method of estimating the extensiveness of lesions based on surface area proportions. (6) The actual value of this determination lies in the fact that the estimation may be done with exactness and serve as a valuable criterion in judging the prognosis. It is also a practical method of classifying burns when used in conjunction with the depth classification.
In utilizing this method it is essential to know the relative proportions between the surface areas of the component members of the body and the total body surface. The whole is the sum of all its parts. The ratio between the size of the lesion and the member of the body bearing it is easily judged. Berkow states that whereas surface areas per se, vary widely in different persons, the proportions do not so vary. Hence these proportions are sufficiently constant, because disproportional people are few.

Berkow conducts the estimation as follows: "The ratio between parts and the total body surface, arbitrarily corrected to the greater seriousness of lesions of the chest, abdomen, and genitals is: lower extremities thirty-eight per cent; trunk, thirty-eight per cent; upper extremities eighteen per cent; head six per cent. In children the proportions are different, the head and lower extremities vary considerably with age. To ascertain the proportions at a given age, the following rule is proposed: The trunk is forty per cent, the upper extremities sixteen per cent. For the head and lower extremities, subtract the age in years from twelve and add the remainder to the number expressing the adult proportion for the head (six per cent). Subtract the same amount from the number expressing the adult proportion
for the lower extremities (thirty-eight per cent)."

"To estimate a lesion of the head, trunk, upper or lower extremities the number expressing the proportion of that part is multiplied by the fraction expressing the relation of the lesion to the part. For ease in arriving at the latter relation, it should be borne in mind that the hand is one fourth of any upper extremity; the arm three fourths; that a foot is one sixth; a leg two-sixths; a thigh three sixths of a lower extremity. The trunk included the neck; the lower extremities included the buttocks. If more than one part is injured, the lesion of each part is estimated separately."

General Pathology
Necropsy Findings (41)

Central Nervous System: Hyperemia of the brain and meninges with small minute hemorrhages into the brain substance and engorgement and thrombosis of the veins of the arachnoid.

Kidneys: Acute glomerulitis is an early finding with cloudy swelling and fatty degeneration appearing later in the proximal convoluted tubules. Thrombi have been noted in the vessels, particularly in the glomerular tufts. Necrotic areas are found in the more severe degrees of burns and hemoglobin occurs throughout the
renal cortex to give the kidney the brownish-red color seen almost invariably in fatal burns.

Superrenal Glands: The pathologic findings in these organs are interesting because attempts have been made to ascribe the fatal effect of burns to disturbance of their function. The normal weight of the superrenal gland is from four to seven grams and in the severely burned case it often weighs from twenty to twenty-five grams. Experimentally within twenty-four hours there is marked hyperemia and occasional hemorrhages noted in the gland along with disappearance of lipoid bodies, loss of chromaffin affinity, and focal necrosis. The perirenal fat is markedly edematous. The glandular cells are markedly swollen and hydropic.

Spleen: The spleen is enlarged and shows focal necrosis in the germinal centers of the lymph nodules. The endothelial cells proliferate rapidly to occupy the germinal centers and may fuse to form giant cells. Later hyaline degeneration of the lymph nodules may be seen.

Heart: This organ in the late severe cases shows hyaline and fatty degeneration with necrosis of the muscle fibers.

Liver: Parenchymatous degeneration with focal necrosis and hyperemia is usually seen in this organ.
Wilson and his colleagues (60) have done much investigation on the liver and find that at twenty-one hours after injury, fatty degeneration of the epithelial cells surrounding the efferent veins in the central zone of the lobules accompanied by nuclear damage. After fifty-seven hours, they find the liver characteristically enlarged, yellow, soft, greasy, and friable. Cut surface gives the "nutmeg" appearance. Microscopically there is much free fat in the necrotic cells. They found that the destruction of liver tissue was comparable with that of acute liver atrophy but differed in the zonal distribution which was constant and characteristic. It differed also from the majority of bacterial toxemias both in affecting the central instead of the peripheral zone and in the more pronounced severity of the injury to the liver cells.

Lungs: In fatal cases of burns the lungs are congested with thrombi in the smaller branches of the pulmonary artery. Focal pneumonia is usually present.

Gastro-intestinal tract: Hyperemia is noted throughout the tract with petecial hemorrhages into the mucosa. Ulcerations may occur anywhere along the tract but are found most frequently in the duodenum.
Blood and Urine Changes

These changes are given here to complete the picture of the severely burned patient. While it must be remembered that, according to some, the blood changes represent the cause rather than the result of the shock factors in burns, for the time being it will be approached in the sense of observed results. The possible part played in cause will be given later under the discussion of the theories of burn-shock in the section on Pathological Physiology.

Red Blood Cells: The erythrocytes undergo alterations in morphology and function. There is considerable variation in the form of these cells, some being spherical, others bell-shaped and crenated. These changes are possibly due to a partial melting of the lipoid capsule of the cells. (48) Distortion is more common than fragmentation although the red cells may be broken up. Occasional ghost cells are seen (19). Schultze experimented on animal blood, demonstrating that when exposed to a temperature of fifty-two degrees centigrade the red cells broke up into spherical bodies which lost their hemoglobin. (49) There has been noticed a considerable diminution in the vital properties as noted by their lowered resistance to such influences as heat, compression, drying, salt solutions and staining. (34)
White Blood Cells: Immediately following burns, there is a rapidly rising leucocytosis reaching 30,000 in severe cases and to 50,000 in fatal cases. A differential count shows that the polymorphonuclear neutrophils are increased to eighty or eighty-five per cent. Myelocytes are present in small numbers.

Blood Concentration: It has long been recognized that concentration of the blood is one of the characteristics of severe burns. In severe but not fatal cases the increase in a few hours in the number of red cells is from one to two million. In fatal instances there is an increase from two to four million per cubic millimeter. (34)

Blood Chemistry Changes: Davidson, (17, 18) found a significant lowering of chlorides in both the whole blood and in the plasma. He also found a hypoproteinemia with reduction most marked in the albumin fraction. Underhill (53) and his co-workers also found a lowering of blood chlorides. It is pointed out, however, that since the cells normally less chloride than plasma, and since in burns cells are increased, it should be expected that on the basis of this blood concentration the finding of low whole blood chlorides should be expected. However, Davidson found low chloride in plasma studies.
also. Cicalia (13) working experimentally on rabbits, found a lowering of the blood chlorides and an increase in the non-protein nitrogen. He believed that some of the increase of nitrogen was due to histamine-like substances. The creatinine and uric acid increased and he attributed this to renal damage.

Mattina in 1935 (36), reported an increase in the blood magnesium in burned experimental animals. The increase began twenty-four hours following the burn and reached its maximum in ten days.

Numerous studies on blood composition in burns have been made, but the significance of the findings and the interpretations made are not always clear. As Pack, (41) states it "Until it can be demonstrated that the composition of the blood is uninfluenced by the concentration of the blood, it is better to accept the explanation that blood concentration is responsible for the changes noted in composition of the blood rather than to ascribe these changes to other causes."

One other blood finding in burns is given, however, because it plays an important part in one of the theories of burn-shock. Greenwald and Eliasberg (23) found that there was a hyperglycemia in humans and rabbits during the initial period of shock and have attributed this to hyperactivity of the superrenal glands. When toxemia
ensued, with the accompanying degenerative changes in the adrenal glands, they observed the hyperglycemia to change to hypoglycemia.

Urine Changes: In severe changes there is an oliguria. This is attributed to kidney lesions, and to the innability of the concentrated blood to exert an hydremic stimulus on the kidney. The urine is highly colored and often smoky because of the hemoglobinuria. The specific gravity is increased. Albumin is found in varying amounts, the amount varying directly with the severity of the burn. Acetone is frequently found after severe burns.
Pathological Physiology

It is fairly well agreed upon that the phenomena induced by burns is in reality a sequence of events produced by reflex, biochemical and toxic factors, and that this sequence takes place with definite clinical stages. It must be remembered, however, that the stages may overlap and that the sequence may be terminated by treatment. Also, that burns involving less than twenty-five per cent of the body surface may not show this sequence in the clinical picture and may cause little general upset. On the other hand, a patient with a lesion of the face may demonstrate every stage with considerable severity. (57)

The course can usually be divided into the following stages: Primary shock, secondary shock, septic toxemia, and healing. This is the American classification. The British classification is: Primary shock, secondary shock, acute toxemia and septic toxemia. Correlation between the two has been made in the introduction. For the sake of completeness, the British classification will be followed in this paper.

Primary or Initial Shock: This occurs at or shortly after the accident. It is believed to be brought about as a consequence of a severe vasomotor upset caused by the reception of multiple painful nervous impulses from the injured area, together with pain and fright.
It is interesting to note at this point that Bailey (3) in observing burns in the present war, states that the initial shock is infrequently seen in soldiers burned during combat, while it is almost invariably seen in burned civilians. The explanation of this would seem to be more in the field of the psychiatrist than that of the physiologist.

When present, it is characterized by a fall in blood pressure. The pulse is feeble, but the rate is usually unaltered. The skin is pallid and clammy and there is often apathy or anxiety. It is a transient stage resembling syncope from other causes and recovery is usually rapid without specific treatment.

The pain and fright mechanism for this stage has been shown by Underhill, Kapsinow and Fisk (53). In their investigations, they burned animals under anesthesia and showed that these anesthetized animals did not develop primary shock.

This stage may be prolonged and may pass into secondary shock. (57)

Secondary Shock: This stage appears, when present, anywhere from two to ten hours following a burn. This stage is identical with surgical shock from other causes. While emphysis is placed, in this paper, upon a later stage, still this type of shock is frequently seen in
severely burned patients and thus must be dealt with.

- The clinical aspects of secondary shock are readily recognizable. The skin is pale, grayish, cold and clammy. There is a low venous pressure and a low or falling arterial pressure. The pulse is rapid and thready or may be imperceptible. Patients in shock also exhibit thirst, dyspnea and increasing depression of sensitivity. Occasionally there is nausea and vomiting (46).

The etiology of this type of shock is also a subject of controversy as is true burn-shock. J. G. Riley (46) has summarized the major theories as follows:

Vasomotor Exhaustion Theory: Which holds that shock is produced as a result of exhaustion of the vasomotor centers in the cord and medulla by their being excessively stimulated through afferent nerves by tissue insult. As a result of this exhaustion of the vasomotor centers, arterial relaxation occurs and produces a fall in blood pressure and a decrease in the volume of the circulating blood. The evidence cited to support this theory was that persons who die of shock show morphologic changes in the cells of the vasomotor centers of the cord and medulla. This theory is refuted by the fact that, in persons who have died of shock, morphologic changes in the cells, in addition to being seen in the
vasomotor centers, are seen in the parenchymatous organs which do not receive afferent stimuli. Furthermore, autopsies reveal that the arterial system is in a state of maximal contraction in patients succumbing to shock.

The Acidosis Theory: This theory claims that the decreased alkali reserve seen in shock is the cause of the shock. It is true that a decreased alkali reserve is present in shock but the theory that this is the cause is disproved by the fact that the experimental production of acidosis does not produce shock and in addition by the fact that sodium bicarbonate solution intravenously will alleviate acidosis but will not affect shock to any great extent.

The Fat Embolism Theory: In traumatic injuries—particularly those involving the long bones of the extremities—fat globules are released and are arrested in the pulmonary capillaries. This produces a shock-like picture but is not true shock because shock is accompanied by a low venous pressure and fat embolism produces a high venous pressure. The conclusion is that fat embolism is an entity but is distinct from shock.

The Apnea Theory: The explanation of the pathogenesis of shock by this theory is that a failure of the venopressor mechanism occurs as the result of a decrease in the carbon dioxide content of the blood. This is one
of the most satisfactory explanations for the cause of the shock, but subsequent experimentation has failed to substantiate this theory. At the present time it is felt that shock may exist without acapnia and acapnia without shock.

Adrenal Cortex Insufficiency Theory: Adrenocortical insufficiency has many similarities to shock but complete removal of the adrenals in experimental animals does not produce shock in a matter of hours but in days. It is difficult to hypothesize a rapid response to deficiency in one condition and a slow response in another.

The Traumatic Toxemia Theory: This seems the most logical theory but at present there is considerable controversy over the validity of experiments to prove or refute it. In substance, the theory holds that trauma to tissue produces a histamine-like substance that, when disseminated by the vascular system, produces permeability for substances to which they were previously impervious. The ultimate theory of this theory has not been decided.

The above may serve as a brief resume of this stage in the clinical picture. It, of course, does not presume to be complete in any sense. The work that has been done upon this type of shock is voluminous and beyond the
scope of this picture.

**Acute Toxemia:** This is the stage of true burn-shock and its existence is unique to burns alone. Clinically this stage usually starts about the twelfth to eighteenth hour following the burn, unless its inception has been prevented by proper treatment. Once the patient has recovered from the secondary or surgical shock, he is usually fairly comfortable for the first few hours, regardless of the form of treatment which has been used on the burn. Later, if treatment has not been instituted, or if it has been inadequate, the following may be observed: The burned area becomes edematous and oozes fluid. At the same time the blood of the patient begins to show the characteristic concentration. The burn itself looks clean in the sense that there is no obvious infection or necrosis. The patient has little pain and is not actively ill. The temperature in this period is usually normal or only slightly elevated. The pulse is usually of good quality and not markedly rapid. But, beginning about the twelfth to eighteenth hour following the burn, the patient begins to show signs of the so-called toxicity. He starts to grow stuperous, or perhaps restless, the temperature becomes elevated and the patient complains of pain. Nausea and vomiting may occur. From this time to about the seventy-second hour,
a characteristic syndrome occurs. The patient's general condition grows obviously worse. He is nauseated and cannot retain food or fluid by mouth. He grows restless and at times delirious. The face is flushed, the pulse and respirations are increased, and the temperature is elevated in a staircase manner until at about the seventy-second hour it reaches a maximum of between one hundred two and one hundred three. It is usually sustained at this level. (26)

To account for this reaction, many theories have been formulated. Most of these have not stood the test of time and are now of only historical interest. There are, however, several theories that, in the light of experimental evidence, hold an important place in the study of burns. The three major theories held today are: the tissue toxin theory, the bacterial infection theory and the physical or blood concentration theory. A fourth, the adrenal theory, merits consideration due to the fact that in necropsy studies and in studies of blood chemistry, the adrenal glands have been implicated and seem to play an important part in the picture.

The Toxin Theory: The theory of toxemia has been advocated by a large group of investigators. It is based on the assumption that there is formed in the site of the burn, or in the burn itself, a toxic sub-
stance that is responsible for the general reactions of the patient. Many substances have been described by observers to have been found in the blood, in the burned skin, and in the urine. The variety of substances is great, including pyridine, guanidine, peptone, primary and secondary proteoses, histamine and ptomaines. It may, however, be said, that until the present time, at least, no definite toxin has been isolated and proved by different workers to be definitely the toxin of burns.

Pack, one of the supporters of the toxin theory, supports the presence of the hypothetical toxin by citing the work of Vogt (56) who demonstrated that complete excision of burned areas prevented the occurrence of burn shock in that animal, but if the burned skin were transplanted to another animal, the second animal became toxic within an hour. He presents the work of Vogt (56), of Heyde (30), and of Vaccarezza (55) wherein they did altercursive intubation and found that if the blood streams of two animals, one burned and one normal, were mingled, the symptoms of shock were less in the burned animal and present in the normal. Also the burned limb of one animal was totally isolated from that animal and the vessels connected with another animal. The unburned animal whose blood circulated
through the burned limb of the other animal developed toxemia and the other did not. He rules out possible nervous etiology by citing the work of Kotzreff (32) who observed that division of nerves to the burned extremity of a guinea pig did not diminish the severity of the toxemia.

Further evidence in support of the toxin theory is the work of Robertson and Boyd (47) who have presented some of the strongest evidence to prove it. These investigators grafted burned skin from animals onto normal animals who showed toxic manifestations in about eight hours. They also injected whole blood from burned animals into normal animals and found that the blood was highly toxic, whereas blood serum alone was not. They made alcoholic extract of normal skin and of burned skin and injected it into normal animals. They found that the extracts of normal skin was non-toxic while the extract of burned skin was immediately toxic. They isolated and analyzed this toxin and found it to be of two parts; the first thermostable, diffusible and neurotoxic; the second was thermodabile, colloidal, and necrotoxic. They found that the toxin chemically, was made up of primary and secondary proteoses.

The work of Robertson and Boyd has been vigorously attacked by later workers. Underhill and Kapsinow (52)
repeated the work of Robertson and Boyd. They were unable to demonstrate the circulatory toxin in that the injection of whole blood from the burned animals did not produce toxic manifestations in normal animals. They did, however, find the injection of an alcoholic extract of burned tissue to be toxic to normal animals, but they likewise found that the injection of an alcoholic extract of normal skin was also toxic. An analysis of the extract showed enough alcohol to be present to account for the symptoms observed. They injected into normal animals an equal quantity of ethyl alcohol and produced identical symptoms. They concluded that the primary and secondary proteoses of Robertson and Boyd were in reality, ethyl alcohol.

In a later paper, these same men (54) showed that, whereas burns induced an increased capillary permeability, the loss of fluid is external and that the absorption from the burned area does not take place at all for twelve hours and but very slowly for twenty-four hours. In these experiments the injection of methylene blue into the burned area showed no absorption into the blood stream, and strichnine injected in lethal doses did not produce convulsions. But, on the other hand, Mason, Paxton and Shoemaker (37) showed that potassium iodide was easily absorbed from the burned areas.
Adding to the confusion, Harrison and Blalock (29) grafted burned skin onto normal animals and stated that they could find no evidence of toxemia. These investigators also burned animals and found that the shortest survivals were among those debrided. They also confirmed Underhill and Kapsinow's results with whole blood injections from burned animals in that they could demonstrate no toxin.

The Japanese investigators as cited by Harkins have all supported the toxic theory. Naganita (40) found that blood serum perfused through burned limbs of experimental animals showed a histamine-like depressor action. However, the action of the depressor substance was not abolished by histaminase. Inouye (31) stated that the adrenal changes were much the same as those caused by strychnine, barium chloride, and picrotoxin. He recalled the work of Pfeiffer, (44) who attributed adrenal changes to low blood pressure accompanying shock, but quoted Kolisko, Nakata, and Niemeyer as attributing the adrenal damage to the toxin and the adrenal damage in turn killing the animal.

Harkins, Wilson and Stewart (27) made protein-free extracts of normal and burned skin of rabbits using trichloroacetic acid extraction. The extracts of normal skin were found to contain a depressor substance
which was not found to be acetylcholine, adenosine, histamine, or the "P" substance of Eler and Gaddum. (20) Burned rabbits skin was found to contain an apparently identical substance. Wilson, Jeffrey, Roxburg and Stewart (62) in a later paper published similar experiments but this time did not use protein-free extracts. These extracts did not show evidence of toxic action. They found a gradually increasing toxic action and when the edema fluid was collected forty-eight hours after burning it was frequently lethal to healthy animals. The toxicity was independent of bacterial action, they stated, and seemed related to autolytic action in the injured tissues. They noted degeneration of liver cells by the toxin. In a recent paper, (60) Wilson, MacGregor and Stewart describe extensive liver damage (given in necropsy findings) and state that their experimental findings suggest "that the acute toxemia of burns is caused by the action of a specific toxin, of non-bacterial origin, which has been absorbed from the burned area."

Bernhard (7) found that extracts of experimentally burned skin were toxic when injected into other animals.

Fender (21) reviewed much of the earlier work favoring the toxic theory and stated that merely because there is confusion as to the name of the toxin
does not give evidence against its presence. He concluded that "the theory of causation of death by a toxin cannot be said to rest upon adequate experimental work at present."

It was in the conviction that the toxin theory is the explanation of burn-shock that led Davidson (16) to develop the tannic acid treatment of burns. He went on the hypothesis that coagulation of the burned area by tannic acid would prevent absorption of the toxin and would prevent general reactions to burns. It must certainly be said that, toxin or no toxin, the tannic acid treatment has been, clinically, the most important step toward adequate burn therapy that has been made.

Physical or Blood Concentration Theory: This theory has been carefully worked out by Underhill, Carrington, Kapsinow and Pack (52). They demonstrated in twenty-one patients burned in a theater fire, a marked concentration of the blood. They enlarged upon this experimentally, and showed that when an animal is burned there is an increase in capillary permeability in one direction and a decrease in absorptive power in the other direction. In other words, fluid pours rapidly from the capillaries out into the surface whereas no reabsorption takes place from the burned area for twelve hours and then very slowly for twenty-four hours. They have
measured and analyzed this fluid and found that it is so similar, chemically, to blood serum that for all practical purposes it could be considered identical. It is estimated by these investigators that, by comparison to the experimental animal, a man weighing one hundred fifty pounds with one-sixth of his body surface burned will lose 3,500 cc of fluid in the first twenty-four hours and that the fluid lost is directly proportional to the surface area involved. They state that there are no blood chemical changes in the burned animal that could not be due to this fluid loss. They draw the conclusion that whereas the surgical shock stage is due to a relative reduction in the total blood volume, the burn shock stage is due to an actual reduction of the total blood volume.

Blalock (10) confirmed in his experiments the results of Underhill. He determined the amount of fluid lost into the tissues by burning one side of the animal and then bisecting the animal and comparing the weight of the two sides. His series of burned dogs showed that the edema fluid averaged 3.3 per cent of the body weight or about one-half of the plasma volume. He states that plasma was lost to kill them alone without invoking the action of any toxin at all.
Harkins (28) confirmed Blalock's data by repeating his experiments and also by placing the burned animals on a tipping apparatus so that as the burned side grew heavier a kymographic tracing measured the shift. He found that the accumulation began at the time of the burn and continued in the form of a decelerating curve until death. The accumulation of fluid was directly proportional to the increased hematocrit readings.

Davidson (16) stated that in his series of cases treated by tannic acid that there was negligible concentration of the blood. This might be interpreted as the local coagulation preventing the loss of fluid from the burn site and therefore accounting for the absence of toxemia, or it might be interpreted as he did, that the coagulation prevents absorption of the toxin which in turn was prevented from having its systemic effect with concurrent blood changes.

Trusler, Egbert and Williams (51) point out that due to fluid loss, burned patients are thirsty and drink enormous quantities of water which leads to "water intoxication" superimposed on the burn shock. They emphasize that the fluid loss is plasma loss and that intravenous saline and glucose solutions only produce waterlogging of the tissues and lowers the chloride level. They confirm their observations with animal experiments.
Infection Theory: This is the latest of the theories concerning the effect of burns. It was presented by Aldrich (1) although he credits Dr. W. M. Firor with the conception of the idea. These two men discount the tissue toxin theory entirely but admit the probability of Underhill's blood concentration theory as having a part in the phenomena. They believe that infection accounts in part for the symptomatology of burn-shock and entirely for the stage of septic toxemia.

Their theory had its inception in their observation of the obvious infection in the burned areas after the first twelve hours following the burns. Before their investigations they had tried out all of the generally accepted treatments and had compared them, on the same and on different patients. They state that the tannic acid treatment was superior to all others. They were impressed with the lack, in the literature, of studies on the bacteriology of burns, and this, coupled with the infections observed, led them to do their studies and thence to evolve their theory.

They found, by taking cultures from the burned areas and of the fluid under the blebs, at short intervals following admission of the patient to the hospital, that cultures were sterile for the first
twelve hours. A few contaminants were found—staphylococcus aureus and albus and B. coli—but the growths were slight. After the first twelve hours they found that in one hundred per cent of the severely burned patients and in a large majority of minor burns, there could be grown from repeated cultures the beta hemolytic streptococcus. They found the concentration of these organisms increased with the obvious signs of sepsis and the beginning of the toxicity of the patient, until after forty-eight to fifty-six hours pure cultures of the streptococcus could be obtained, having out-grown all other organisms.

They reasoned that when a large surface of the body has been destroyed by a burn and becomes involved with streptococcus infection, that the characteristic bad effects of the burn could be ascribed to this. They gave as additional evidence the fact that in fatal cases, the hemolytic streptococcus was found in the heart-blood and in the lungs in instances of terminal pneumonia.

From this, they decided that the best form of treatment would be one wherein the burned area could be sterilized. They felt that if they could show, clinically, that no toxic symptoms developed when the burns
remained sterile, that it would be logical to assume that the infection was the essential factor in the production of the toxic symptoms. They explain the success of the tannic acid treatment of Davidson not in the fact that it rendered the toxic substances non-absorbable, as was Davidson's contention, but because they felt that the tannic acid eschar over a practically sterile burn sealed the burn and prevented fluid loss and infection. They felt that if a more bacteriocidal substance could be used to form the eschar that the hazard of infection could be avoided. So they used gentian violet because it is almost specific for gram positive organisms and because the pH reaction, together with the contained methyl radical, it reacts with the burned flesh to form a thin, light, tough and flexible eschar. The dye also produces almost instantaneous analgesia.

They found, under this regime, that the fever of the burned patient was moderate and not of the septic type. They found no indication of nephritis or unusual blood changes. They could find no evidence of developing anemia and the blood chlorides remained up.

Wilson, MacGregor and Stewart (60), however, state that they believe that a bacterial toxin could not be responsible because of their findings of specific liver
damage which could not be caused by bacterial toxins. They were unable to demonstrate the streptococcus as described by Aldrich. In another paper (62) they state that "There is little doubt that acute toxemia is a condition distinct from septic or bacteriogenic toxemia."

Clark and Cruickshank (14), supporting Aldrich's theory advocated the use of scarlet fever antiserum for the burned patient.

Harkins (27) states that many of the toxic manifestations present after the first two days as well as some of the complications of burns may be explained on the basis of a septicemia, although this is not conclusively proved. He states that marked infection is not obvious during the first twenty-four to forty-eight hours which is the time when shock with blood concentration occurs. He says that death from true secondary shock occurs in the first day after a burn, which is earlier than infection becomes active.

In 1937, when Aldrich brought out a modification of his original treatment (2), he states "I am certain that the conception of a burn as an infected surgical lesion is correct, and that the infection rather than the absorption of a split protein is the cause of death in burns. For, let me add again, where there is no infection, there is no toxemia"
Adrenal Theory: This theory will be mentioned only briefly. Interest has been directed toward it since the time that the characteristic adrenal findings were noticed in severe and fatal burn cases. Weiskoten (58) emphasizes the fact that in cases in which death occurs, greatest injury seems to involve the adrenal glands. Both the medulla and the cortex have been indicated separately and several authors have attributed death to the adrenals alone.

Freeman (22) and his coworkers have evolved a theory of shock that consists of the hypothesis of overexcitation of the adrenals producing much circulating adrenaline in the attempt of the cardiovascular system to maintain the blood pressure despite a lowered blood volume. When the blood pressure falls farther, the increased adrenal output can no longer compensate and collapse occurs. If fluids are administered, then shock continues due to hypofunction of the adrenals as a result of damage inflicted on them during the period of hyperactivity. To bear up this theory is the work of Greenwald and Eliasberg (23) who found in humans and in experimental animals that there was a hyperglycemia during the initial period of shock and later, during the toxemia, there was hypoglycemia. They too, att-
ribute the toxemia to adrenal damage in part.

Brooks and Blalock (11) however, have thrown doubt on the adrenal theory of burn shock. They presented evidence to show that somewhat similar adrenal changes will follow hemorrhage and trauma to muscles, indicating that they are not specific to burns.

Other writers have accused the cortex as being the site of injury with resultant hypofunction and death. Lowden (35) supports this view and advises the use of cortin, in the form of desoxycorticosterone. He states that in experimental animals, adrenalectomy results in a fall in blood pressure, concentration of blood and other shock-like characteristics. Injection of cortin returns the animals to a normal state.

Many other theories have been advanced over the years. The ones that have been discussed, however, have been founded upon actual findings and have been backed by experimental evidence.
Fourth Stage of Burns--Septic Toxemia: This is a later developing stage and one that is not always present. It is generally agreed that this stage is due to bacterial infection. Aldrich (1) considers that this stage is only a more full-blown manifestation of the burn-shock. Other authors look upon it as being a complication of burns. With the modern methods of treatment and with the present chemotherapeutic measures this stage is infrequently seen. (6)

Wilson, MacGregor and Stewart (60) state that the incidence of infection is intimately related to the depth of the injury. They state (Aldrich notwithstanding) that local signs of bacterial infection of burns were rarely present before the fifth day, and even in fatal cases, hemolytic streptococci did not appear in the blood until the ninth day.

When this stage is seen, the patient presents the typical picture of toxic manifestations due to an infected wound. The burn suppurates and is foul-smelling, there is regional lymphadenopathy and fever and leucocytosis is septic in type. (43)

Bailey (3) states that with present day treatment this stage should never be seen.
Treatment of Burns

"Every surgeon should understand that it is not he who heals, but the balsam within the body is that which heals, and that wherein thou art a good surgeon is that thou offerest to nature defense and protection in that wounded part".

Theophrastus--1493 A. D.
(From Pack)

In this section, the treatment of burns will be approached from the standpoint of the immediate and emergency treatment to severely burned patients--treatment through the first several days following injury. Thus emphysis will be given to treatment of secondary shock and acute toxemia, along with the local treatment to the burned area. Since it is the effort of this paper to present the emergency aspect of burns, the treatment of the stage of septic toxemia will only be lightly touched upon, and the late sequelae and complications as well as the plastic aspects of late treatment--such as skin grafting--will be omitted entirely.

General Treatment

Wallace (57) states that the first step in treating any burned patient is to control shock. Local treatment should never be undertaken until the immediate threat of shock (traumatic shock) is controlled. He states that the burned area should be covered with a
a sterile dressing and for the moment, be forgotten; that application of any substance locally does not help the patient and may give the surgeon considerable more difficulty in carrying out his treatment.

Treatment of Primary Shock: There is not much found in the literature upon treatment of this phase. It seems to be so transient an affair that the physician is seldom called upon to treat it, recovery usually taking place before his arrival. Wallace (57) recommends only warmth and morphine.

Treatment of Secondary Shock: The treatment of this type of shock when seen in burned patients, is similar to that used to combat surgical or traumatic shock due to other causes. In general, fluids, warmth, sedation, and in some cases, stimulants are advised. (26) Pack (43) states that "It is impossible to establish a line of management for all patients, insomuch that each merits individual attention. No particular routine will prove equally valuable for all cases" So, the individual case will call for individual measures.

Sedation is usually accomplished by giving one-fourth grain of morphine as soon as the patient enters the hospital, or as soon as seen by the physician. Pack (43) warns though, that if shock is extreme, morph-
ine should either be withheld or given cautiously. He states (41) that for the average severely burned adult opiates may be given as the system is extremely tolerant to them under these circumstances. He also suggests the use of ten grains of chloral hydrate with twenty grains of sodium bromide given in cold water every three hours as an alternate method of sedation.

Warmth may be given in several ways. Welles (59) advocates the immersion, clothes and all, into a bath of warm water containing a very dilute solution of tannic acid. This, he states, relieves the primary shock by increasing the body heat and stops pain by protecting the burned areas from cold air and by the analgesic action of the dilute tannic acid. The clothing may be soaked off carefully while the patient lies in the water. Other authors furnish heat to the patient by putting him to bed without undressing and use hot water bottles and blankets. Some (57) use electric light "cradles" which keep the bed clothes from coming in contact with the patient. When the patient is burned so that he must lie on a burned area, the bed clothes should, of course, be sterile. (41)

Fluids, to bring up the blood volume, are most valuable. Underhill et al. (52) showed that there was
there was a relative reduction in blood volume in this stage. The type of fluid and the method of administration has been a moot question for some time.

The earlier authors, working on the idea that there was a relative blood deficiency and that hemoconcentration was present, advised giving large quantities of saline intravenously or by hypodermoclysis. (35) Other writers, however, have reported that large quantities of intravenous saline may actually be harmful to patients in shock. (15) They state that in animal experiments that large injections of crystalloid solution tend to lower the osmotic pressure of the blood column even more and that the crystalloids pass out of the blood stream through the vascular bed and carry with it protein. So, more protein is lost from the blood than would have been if no treatment of this type were given at all and the vascular condition is made worse. These workers advise giving of plasma to increase the protein content of the blood and thereby increase the osmotic pressure. If plasma is not available, they state whole blood should be used. Harkins (26), commenting on the use of whole blood, makes the observation that although the patients blood is concentrated already, the blood of the donor is
relatively dilute and that the common objection to giving whole blood on the basis of increasing the concentration is not entirely valid.

The use of stimulants is valuable to increase the falling blood pressure. Pack (41) advocates the use of caffeine sodium benzoate as well as hot black coffee enemas. The use of adrenaline therapy is controversial. Riley (46) advises its use only in incipient shock when the vessels are dilated and against its use in fully developed shock when the vessels are in a state of constriction. Greenwald and Eliasberg (24) believe that there is a hyperactivity of the adrenals in this stage and so adrenaline should not be given. This is backed by their experimental observations of hyperglycemia in this type of shock. Rack (41) however, states that it is a popular drug and an efficient one to use for shock.

Local Treatment

The local treatment of burns is a very important aspect of the treatment of the burned patient. Proper local treatment, given as soon as primary shock is controlled, serves as a preventive measure against burn-shock or acute toxemia of burns. In reality then, it comes under the heading of treatment of the acute toxemia.
The local treatment has changed markedly in the last twenty years, since it has been in this period that most of the research has been done on the etiology of burn-shock. And, although the actual process of burn-shock is not at all settled, still the theories that have been prominent and the treatments that have been conceived as a result of these theories have brought about great strides in adequate therapy.

In this section, the most accepted present-day modes of treatment will be given. Other methods will be mentioned. The treatments will be given in the order of acceptability rather than in any chronological order.

Tannic Acid: This mode of therapy is one of the most important contributions to treatment of burns that has been made during the twentieth century. (41) Most of the recently devised methods of burn therapy have been offshoots of the tannic acid method. (26) It was originated by Dr. Edward C. Davidson of Detroit. Davidson, acting on the theory that the toxemia of burns is due to some product of protein decomposition which occurs in the burned areas, attempted to find a substance which would diminish pain and seal the lymphatics, thereby preventing toxic absorption and acting as a moderate anesthetic. After studying a number of acid protein precip-
itants, he came to the conclusion that tannic acid is the agent of choice. He states that tannic acid precipitates proteins, lipoids, some glucosides, and salts of heavy metals. It forms a more or less stable compound with protein constituents of body fluids. When applied to a burned surface in dilute solution, its further penetration into the deeper protoplasm is apparently prevented by its action, and the effect seems to be limited to the most superficial layers. If, however, it be applied first in concentrated solution, the acid may penetrate deeply before superficial coagulation has occurred and a deep caustic action may result. The precipitated proteins on the coagulated surface act as a protective coating or barrier against chemical, bacterial and mechanical action. The coagulum splints and protects the injured tissues from trauma and pressure.

Davidson originally recommended a two and one-half per cent solution applied by means of packs. Montgomery, (39) recommends a five per cent solution. Wilson (62) recommends the use of a twenty per cent solution. The use of a spray to apply the acid is now generally used instead of the packs. This method was suggested by Beck and Powers (5) and is used now except in the vicinity of the eyes. The spray is applied every half
hour until the surface becomes a mahogany brown. Drying is accomplished by a bed tent and warm air blower. When the patient must lie on a burned area, sterile sheets should be put under him.

It is very interesting to note that the supporters of all three major theories of burn-shock attribute the success of tannic acid therapy in the light of their particular theory. The supporters of the toxic theory maintain that absorption of the toxin is prevented by tanning. Supporters of the bacterial theory say that infection is prevented by tanning the sterile burned area. The physical theory exponents explain it by saying that it prevents loss of fluids from the body.

Taylor (50) objects to the use of tannic acid so generally. He states that from his studies he finds that the tanning process kills the islands of epithelium that may be left in the hair follicles and sebaceous glands, and in so doing, prevents these areas from serving as points of regeneration of the epithelium. He says tannic acid should only be used where the condition of the patient is one of life or death. He also objects to it on the grounds that the formed eschar is stiff and cracks, thus forming a portal for infective agents. W. E. Lee states that it should never
be used on burned fingers as the eschar contracts and produces an ischemia of the terminal digits with resultant sloughing. Along this line, however, H. G. Pinker (45) states that he believes the crippling end-results of fingers is due to the severity of the burn rather than due to the tannic acid. However, he recommends that on the fingers, tannic acid be only applied to the anterior and posterior surfaces.

Modifications of the tannic acid used have been suggested. Mitchiner (38) recommends the use of a one to two thousand mercuric chloride solution to make up the tannic acid solution, thus bolstering the weak bactericidal properties of the tannic acid. Wilson (61) advises using one to one-hundred acriflavine with the tannic acid for the same reason.

Bettman (8) made a valuable modification of the tannic acid treatment when he suggested using a combination of tannic acid and silver nitrate. This method does away with many of the objectionable aspects of straight tannic acid. Only one application must be made, thereby eliminating the loss of body heat that goes with frequent sprayings, the loss of fluids is stopped immediately, the silver nitrate prevents infection, and the thin coagulum is flexible and not so likely
to cause constriction as is the heavier tannic acid membrane. His mode of application is; after the skin has been thoroughly cleansed and blebs opened and loose skin removed, to apply five per cent tannic acid with a swab followed by application of ten per cent silver nitrate in the same manner. The patient is then placed in a tent heated by electric lights and the burned area dried.

Gentian Violet: This was first used by Aldrich (1) and came as a result of their observations and experimentation. They chose gentian violet because it exerts an almost specific bactericidal action on gram positive organisms, infection with which, he held, caused toxemia; and because of its pH reaction together with the contained methyl radical, it reacted with the burned flesh to form a tough, light and flexible eschar along with analgesia. The dye was found to be of negligible toxicity by Churchman and Herz (12) who found that rabbits showed no ill effects although a concentration of one to two thousand was circulating in their bloodstream.

In 1937, Aldrich brought out the modification of his gentian violet treatment wherein he advocated the use of a combination of three dyes--gentian violet,
acriflavine and brilliant green. We found that this combination was very effective against both gram positive and gram negative organisms. It is highly bacteriostatic and will not injure living tissue. The mixture of dyes is used in the same way as is gentian violet alone.

The technique of application is quite similar to that used in tannic acid. The surrounding skin is cleansed as much as possible, the blisters are punctured or removed, and for the first twenty-four hours the wound is sprayed every two hours with a one per cent aqueous solution of gentian violet. Following this period the wound is sprayed every four to six hours as long as seems advisable to the surgeon.

Picric Acid: This substance has been used for some time, but Pack (41) states that it is of value only in minor burns as it is quite toxic and gastro-intestinal, nervous, circulatory or urinary symptoms may occur. It should never be used over large surfaces. Wallace (57) concurs with this and states that it gives good analgesia and satisfactory sepsis in second degree burns but that it should not be used for more serious burns due to its toxic reactions. It is commonly used in a one and two-tenths per cent solution applied on a
sterilized gauze and covered by a pad of dry absorbent cotton and a light bandage.

Paraffin: This is described in the monograph by Pack and Davis (41). It should only be used if the surface is aseptic. It gives symptomatic treatment and prevents pain as well as splinting the injured tissues. The biggest objection to it is that it has no bacteriidal action and infection is a big danger if the burn is extensive. Surgical paraffin is melted and used at a temperature of about one hundred twenty-seven degrees Fahrenheit. Vesicles are not opened. The burn is covered with a thin layer of cotton and the paraffin is applied with a brush or as a spray. More cotton is applied and then another layer of paraffin to cover and seal the entire dressing. At later dressings, the vesicles are punctured and dead skin cut away.

Oils: This mode of treatment has been used for centuries as it gives symptomatic relief from pain by shutting off air from the wound. It is not a good method though, as nothing else is accomplished but the relief of pain when neutral oils are used. Recently, however, cod liver oil has been recommended for burns. Lichtenstein (33) has done investigations upon this, and found that cod-liver oil killed suspended bacteria
in seventeen to twenty-four hours and has, therefore, a definite bactericidal power. It is non-irritant to tissues, a property possessed by few bactericidal preparations. In addition, the tissues are supplied with vitamins and probably other forms of nourishment. The bactericidal power of the oil is enhanced by irradiation with ultra-violet rays from a mercury vapor lamp. Dressings should not be reapplied more than twice weekly.

In general, however, no oil can adequately prevent the loss of fluid from the burned area and so is undesirable regardless of whether it is bactericidal or not.

Sulphydryl Solution: This is the work of Mellon (37a), who has used it in the treatment of one hundred fifty cases of burns. He points out that the nutritional value of glutathione, a normal component of most human tissues, and in particular the skin, is dependent upon the reduction of its oxidized or SS sulphur to the sulphydral or SH form. The use of the SH form in the form of "Hydrosuphosol" he believes, stimulates tissue growth. In this treatment there is an early febrile effect which, he believes, also plays a part in the success of the treatment. He states that
scarring is minimal. The sulphydryl compounds are liberated from the solution on dilution with water, or on contact with the tissues. Simultaneously, a colloidal form of sulphur is precipitated, which results in the formation of a dry, sterile and flexible eschar.

He states that in his cases there has been no clinical evidence of infection nor any evidence of toxicity. The solution is also sufficiently bland so that it may be employed in diluted form in burns of the eye.

In his words, "In the cases so far studied, this solution appears to fulfill the several requisites of a successful local treatment of burns more completely than any other single treatment with which we have been familiar. There is incomplete evidence that certain components of the solution tend to counteract shock."

As has been said before, the treatments that have been described in this section are only the methods that are generally used at the present. A complete list of all the treatments that have been used would be beyond the scope of this paper.
General Treatment in Acute Toxemia: The striking feature of this type of shock is the same as that of the stage of secondary or traumatic shock—circulatory failure. If adequate local treatment has been given, this stage will not develop. The mechanism is not precisely clear, but local treatment of the proper sort will prevent its development.

If the patient is first seen at this stage, the same general treatment should be given as was outlined under secondary shock—sedation, warmth and fluids. After the shock is controlled, then attention should be given to the local lesion and it should be cleaned thoroughly and the treatment of choice applied (57). If local treatment has not been previously instituted and infection is probable, Wallace (57) recommends the use of one of the sulphonamide drugs in small doses and following with large doses at the first signs of sepsis.

Robertson and Boyd (47) recommend the "Exanguination-Transfusion" method of treatment. Their idea is that this is a good method to remove the toxin from the blood. Pack (41) does not believe this is necessary as he states that, in his opinion, the toxin is eliminated from the kidneys and the dilution and diuretic effect of saline and glucose is to be preferred.
Lowden, (35), recommends the use of cortin in the form of desoxycorticosterone. In burn-shock, there is a fall in the sodium and a rise in the potassium content of the blood, and cortin mobilizes the sodium from "an unknown source" and enables the sodium to be retained in the blood serum. Wallace (57) states that urgent indications for the use of cortin are a marked fall in blood pressure and a failure of peripheral circulation.

On the basis of the bacterial theory of the burn-shock, vaccines and serums are advocated by some (41). But it is stated that it is a safer policy to inactivate the antigen by coagulation than it is to attempt to find and use the proper antibody.

Treatment of Septic Toxemia: When this stage of sequence has developed, the treatment is the same as for any infected wound. To prevent this stage, adequate local treatment should suffice. Late blistering is a source of infection and the tan, however produced, should be constantly inspected for fresh bli and for cracking. When found, these areas should be
as irrigations. Mercurochrome or acriflavine have also been recommended. The use of the sulphonamide drugs greatly improve the prognosis in this stage. (57)
In this paper, the pathology, both local and systemic, which results from a severe burn has been given. The theories of the pathological physiology which attempt to explain the clinical manifestations have been discussed.

The first dangerous stage that is encountered in a severe burn is secondary or traumatic shock. This seems best controlled by administration of plasma or, if plasma is not available, of whole blood. Additional therapy is helpful, but plasma is the essential.

The second dangerous stage is burn-shock or acute toxemia. Each of the three theories that have been advanced as to the etiology of this stage is backed by considerable experimental evidence. The clinical applications of these theories in the material form of logical therapy based upon each conception give further evidence of the credence of each.

The conclusion is therefore drawn, that the condition of burn-shock is a state in which the physiological mechanisms contained in each of the three theories plays its part. The loss of fluid from the blood in the form of plasma will upset the biochemical balance of the blood so that the normal function is hindered to the extent that shock is produced. That a specific toxin is produced has not been definitely proved, but
experimental evidence points strongly to its presence. The absorption of dead and necrotic tissue from the injured area might well be the "toxin" of burns. That bacterial activity plays a part is very probable. Any large area so exposed to infection and so deprived of the natural barriers would furnish an excellent culture media. Therefore, all three must be borne in mind in selecting proper local therapy.

The treatment by local coagulation is by far the most satisfactory. This treatment serves as a logical application of each of the three theories. It serves to fix any tissue toxin, it restores the continuity of the skin and prevents loss of fluid, and it forms a barrier against infection.

be superior to others as it gives not only a coagulum but it also gives the highest bacteriostatic action of any of the precipitant dresings.

Proper local treatment will, then prevent the development of burn-shock.

The features of greatest interest to the physician
64.

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