Thermal burns: their pathology, physiology and treatment

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THERMAL BURNS: THEIR PATHOLOGY, PHYSIOLOGY AND TREATMENT

by

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CONTENTS

I. Introduction .............. 1
II. Pathology .................. 4
III. Pathological Physiology 10
IV. Clinical Course .............. 23
V. Treatment ..................... 28
VI. Summary ..................... 54
VII. Bibliography .............. 55
THERMAL BURNS: THEIR PATHOLOGY,
PHYSIOLOGY AND TREATMENT

Introduction

The problem of cutaneous burns has been with us for centuries. In reviewing the literature it is interesting to note how the pendulum swings, at times, with certain advances in therapy interest becomes paramount, and at other times when interest decreases, the question of burns falls off and the burned patient suffers accordingly.

The mechanical age has brought a much more serious type of burn into existence. High explosives, the increased use of oil and gasoline, industrial injuries, and the use of electricity has created a type of burn most difficult to treat and cure.

Almost every clinician has some sort of an idea on the treatment of burns. It is therefore, highly essential for the individual to know something about the background in the history of these conditions, so that he may be better able to understand the logic and principles which are used in our present day treatment.

In the beginning of the nineteenth century, clinicians were still using various procedures which were not far removed from the lards and aromatic oils of Hippocrates. We see there was an early attempt to use some precautions in the treatment of all wounds. Pirrie (43) of Aberdeen, after listening to Lister on the value
of antisepsis, began to use phenol in the dressing of burns. The antiseptic principle was also advocated by Morris (33) in this country.

During the nineteenth century more attention was being given to the general treatment of the patient, and it was realized that this was just as important or even more important than the local treatment. In 1823, Cumen (16) and in 1862, Baraduck (5) pointed out the presence of the increased viscosity of the blood in burns. In 1844, Parker (38) pointed out the importance of treating the shock which accompanied this condition, even though this was not universal until 1880. Several decades ago, much work was done on the value of blood letting. With this knowledge, Dr. Bruce Robertson (49) of Toronto established his life saving measure of exsanguination-transfusion, a method which is used to this day in burn toxemia of grave character.

The local treatment of burns seems to have been inadequate in the past since each author described so very many different applications and stressed none of them.

First we had the introduction of the continuous submersion bath to replace the older traditional methods of occlusive dressings of all kinds of oils and ointments. In 1887, the open air treatment was popularized, forming enclosures about the patients. In 1914, parrafin wax or "ambrine" was introduced as a dressing for burns. In
1925, Davidson (18) of Detroit published his method of treating the burned area with tannic acid. This method seems to have revolutionized the treatment of burns, so that today coagulation of the burned area seems to be the most logical principle of treatment, although there are many modifications to Davidson's original method.

In order that the problem of thermal burns be adequately understood it will be necessary to bring out their pathology and physiology to show that the phenomena induced by thermal burns are in reality a sequence of events and should be treated as such. If this point of view is taken we have a knowledge of the principles which lie behind the various methods of treatment and management of burns.
Pathology

The examination of post-mortem material shows conclusively that the pathological changes induced by severe burns are not confined solely to the local and obvious lesion. There are very definite lesions produced in almost every organ of the body, depending upon the extent and severity of the burn.

The variation in the character of the local lesion in burns depends upon a number of factors, the most important of which are, intensity, character, and duration of the heat. Burns may be either caused by thermal, electrical, or chemical means, but in this paper only the thermal burns will be discussed—those due to fire and boiling liquids. According to the local changes observed heat burns have been divided into degrees. Some authors describe only three degrees while others describe as many as six. Thermal burns are best classified in six degrees to denote the various depths of tissue invaded or destroyed (37).

Local Pathology.

First degree: This consists of a simple erythema with vascular reactions similar to that of any simple erythema due to the action of an irritant. It is essentially a reflex and exudative reaction. The reflex phase consists of contraction followed by dilatation of the arterioles and venules, resulting in an increased flow of blood to the affected area and a local stagnation of the
current. This explains the redness and increased heat always associated with this type of burn. The exudative lesion 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. There may be a slight degree of desquamation of the superficial epithelium a day or two following the subsidence of the hyperemia.

Second degree: Dupuytren's (19) description is as follows: "If the exposure to heat is prolonged, blisters may result. This constitutes a second degree burn. The blisters consist of spaces in the epithelial layers which are filled with acellular exudate of clear fluid which does not coagulate. These spaces are usually just above the germinal layer of cells in the Malpighian layer."

Third degree: In this the epidermis is entirely destroyed as is part of the corium. The papillae appear as a reticular framework containing serum, bits of living epithelium, leukocytes and fibrin. 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 the 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 epithelium.
Fourth degree: "In this there is destruction of the entire thickness of the integument and complete desquamation of the skin involved. If the traumatizing 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 capillaries proliferate tufts of endothelial cells accompanied 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." (22).

Fifth degree: Fifth degree burns expose structures deeper than the subcutaneous tissue including muscle. The scar is usually quite deforming and has a tendency to break down and ulcerate.

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

General Pathology.

The pathology of the internal organs is not by any means characteristic in burns. It has been noted that the changes were similar to those found in the acute infectious diseases of the skin. At necropsy the following systemic changes have been noted.

Central nervous system: In early cases little is seen beyond the congestion of the brain and meninges. Later there are evidences of toxemia with 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. Areas of focal necrosis have been described in the kidney after severe burns and hemoglobin occurs throughout the renal cortex to give the kidney the brownish-red color seen nearly always in fatal burns.

Spleen: The spleen is enlarged and shows focal necrosis in the germinal centers of the lymph nodules. Later hyalin degeneration of the lymph nodules may be seen.

Adrenals: Elliot (20) described hemorrhage or focal necrosis of the suprarenals as the most common and
characteristic post-mortem finding in fatal burns. The normal weight of the suprarenal 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 glands. The perirenal fat is markedly edematous. Later the glandular cells are swollen, hydropic, and frequently show necrosis.

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

Lymph nodes: The usual lesion seen in the lymph node is central necrosis of the follicles with endothelial proliferation.

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

Gastro-intestinal tract: Hyperemia is noted throughout the gastro-intestinal tract with petechial hemorrhages into the mucosa. Ulcerations may occur anywhere along the tract, perhaps due to the action of the toxins absorbed, perhaps to emboli (11).

Blood: Some of the most interesting changes are those in the blood. In severe cases there is an immediate apparent increase in the number of red cells, owing to the concentration of the blood caused by the great
outpouring of fluid. There is some distortion and fragmentation of the red cells. There may be an increase of 1,000,000 or 2,000,000 per cubic millimeter. The leukocytes are increased, sometimes to 30,000. Thrombosis is common. The urine is decreased in amount, is often smoky because of hemoglobinuria, and contains albumin (11). With concentration the sodium chloride of the blood is lowered. Further examination reveals an increase in non-protein and urea nitrogen and sometimes a rise in creatinin and sugar content (55).
Pathological Physiology

That the first ill effect of a severe burn may be a reflex shock phenomenon due to pain and fright, has been adequately proven by Underhill, Kapsinow, and Fisk (59, 60, 61, 62). These investigators noted that in animals burned under anesthesia the primary shock seen in a clinical case did not occur. Underhill (58) and his colleagues also showed that the primary shock occurred too quickly for it to be caused by an actual reduction of the total blood volume, and that the loss of power of absorption from the burned area in the first twelve to twenty-four hours following a burn precludes the explanation of a protein absorption product. That the primary shock could be due to a bacterial toxin is not very logical since the time element is too short and there is a lack of absorptive power in the burned area, in this early stage.

The factor which determines the onset and degree of this primary shock is the extent of the burn rather than its depth. It appears within a few minutes after the accident and is recognized by the usual signs—rapid, feeble pulse, subnormal temperature, low blood pressure, pallor, sweating, and thirst. It is usually over with in twelve hours, and, therefore, if it is prolonged beyond this time we are dealing with something other than surgical shock (45). Pusitz states that rather a small per centage of the mortality due to burns is caused by
primary shock (45). However, Barnes (6), in a review of two hundred and five cases, found that 67.5 per cent of the deaths were from primary shock. Again it must be stated that the severity of the burn would determine the mortality of primary or surgical shock.

It is a known fact that the cause of death in severe burns is due to general and not local phenomena. As early as 1868 Wertheim (66) advocated the idea of a toxin circulating in the blood as a prime factor in burn mortality. Pontick (44) advocated the same idea in 1876. The theory that the burn decreased the respiratory function has been definitely disproved. Welti (65) in 1889 disproved the theory of death being due to failure of the heat-regulatory mechanism by protecting the burned animal from such heat dissipation and showing that there was no reduction in mortality. In a series of experiments Markusfeld and Steinhaus (32) demonstrated that the interference with the nerve supply of the burned animal had no effect upon the ultimate result, while division of the blood supply to the burned area prevented the constitutional reaction.

Following the primary or surgical shock a clinical symptomatology occurs which could likewise be called shock. It is concerning the etiology of this secondary shock that most of the controversy occurs. Up to the last few years it was generally accepted that the ill effects of burns during this stage was due to toxins absorbed into the circulation. Davidson (18) was one of the leading proponents
who, in 1925, summarized the evidence in favor of this hypothetical toxin.

Several men have presented experiments to uphold the tissue toxin theory. Reiss (47) in 1904 reported a toxin in the urine of burned cases which was toxic upon being injected into the laboratory animal. Pfeiffer (42) in 1905 reported the isolation of cleavage products from the burned skin which were neurotoxic and necrotoxic. Robertson and Boyd (50) in 1923, in one series of experiments, removed the burned skin of young rabbits within eight hours and grafted it on unburned animals, while the skin of the latter was grafted to fill the defect in the burned animals. Toxemia developed in the rabbits receiving the burned transplants, while the others escaped. If, however, the transplants were made after eight hours, both burned and unburned suffered. Robertson and Boyd further showed that if they injected blood from burned animals into normal animals they found the whole blood to be highly toxic whereas blood serum alone was non toxic. The injection of alcoholic extracts of normal skin were found to be non toxic to normal animals while the injection of alcoholic extracts of burned skin was immediately followed by toxic symptoms. The toxin chemically consisted of primary and secondary proteoses. From these experiments the idea of debridement and exsanguination transfusion evolved.

It is believed by many investigators that the toxic symptoms in extensive burns are indeed due to protein de-
composition. Just how this toxin acts on individual organs is not yet quite clear. Histamine may be liberated and responsible for the shock (11). Neuda (36) has lately stated his opinion that symptoms are indeed due to protein decomposition, but that the true toxin has not been discovered. From his experiments he believes that cholin or its ester may be responsible. Neuda has also observed an enormous dilatation of the capillaries causing great congestion of the periphery.

Ravidin (46) summarizes the facts which seem to be proved at the present time as the result of experimental work as follows:

1. "The toxin which gives rise to the toxemia in burns is developed by the application of heat to living tissue----.

2. "That the toxin is carried by the blood stream is shown by ligation of the major vessels of the burned part which prevents the development of the toxemia---. The injection of blood removed from toxin-burned patients into healthy individuals results in a transitory toxemia.

3. "The toxin is carried by the red blood corpuscles.

4. "The toxins of the burns are formed very rapidly, for the removal of the burned area more than eight hours after the burn has occurred does not prevent the toxemia."

Recently Underhill, Blalock, Wilson and their colleagues (10, 25, 59, 60, 61, 62, 63, 64) working indepen-
dently, have obtained results so convincingly harmonious that a rapidly increasing group now deny the existence of the tissue toxin entirely, and a still larger group point out the lack of unified opinion among the early workers as Reiss (47), Lustgarten (30) and Spietschka (53). Furthermore, very few of these investigators have been able to check the results of the others. Underhill and Kapsinow (64) repeated the work of Robertson and Boyd exactly. They were unable to demonstrate a circulatory toxin. In the opinion of these workers an analysis of the alcoholic extract of burned tissue showed enough alcohol to be present to account for the symptoms observed. They concluded that, in reality, the ethyl alcohol was the toxin which Robertson and Boyd had used in their experiments.

By grafting burned skin onto normal animals Harrison and Blalock (25) could find no evidence of toxemia. They also found that the burned animals which were debrided survived the shortest period. They also confirmed Underhill's and Kapsinow's results with whole blood injections from burned animals in that they could demonstrate no toxin.

Perhaps the most convincing evidence against the tissue toxin theory, however, are the experiments of Underhill and his colleagues (10, 25, 58, 59, 60, 61, 62, 63) with burned tissue. They demonstrated that whereas burns caused 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 more convincing, strychnine injected in lethal doses did not even produce convulsions.

Another factor which may be of greater importance is concentration of the blood, which rapidly occurs in superficial burns owing to a great outpouring of fluids through the damaged capillaries into the subcutaneous tissue. Underhill and his associates (59, 60, 61) claim and present experimental evidence to show 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. Thus fluid pours out rapidly onto the surface, whereas no reabsorption takes place in the burned area for twelve hours and then very slowly for twenty-four hours. Underhill (59, 63) and his co-workers have shown that in animals burned over one-sixth of the body surface the amount of fluid lost was over seventy per cent of the total volume of the blood. Furthermore, analysis of the fluid showed it is similar chemically to blood serum. Also it is shown that there are no blood chemical changes in the burned animal which could not be due to this fluid loss. Further evidence is found in the fact that the blood pressure follows exactly the curve of the fluid loss.
Blalock (10) confirms, in his experiments, the results of Underhill. Harkins (23) in his investigations points out that the bleeding volume decreases more rapidly than the blood pressure, and that its rate of change is more closely related to the cardiac output, blood concentration, and local fluid accumulation.

These phenomena are entirely due to physico-chemical forces; the water equilibrium between the blood stream and the tissues is the resultant of two osmotic forces—the osmotic pressure of the tissue colloids and that of the blood colloids. As the plasma of the blood escapes into the burned area the amount of colloid per unit volume is increased in the blood stream. This results in the water being drawn into the bloodstream to re-establish the blood volume; and the result is a dehydration of the tissues. This fluid, however, is again poured out onto the surface of the burned area and the blood is again concentrated. Finally, when the body tissues become dehydrated and the viscosity of the blood increases, circulatory failure follows.

After twenty-four to thirty-six hours, the capillary openings close, and if enough fluid has been supplied the fluid equilibrium is re-established. The total plasma proteins and the total plasma volume drops during the first twenty-four hours.

Underhill (62) concludes as follows: "Marked concentration of blood means a failing circulation, an in-
efficient oxygen carrier, oxygen starvation of the tissues, fall of temperature, and finally suspension of vital activities." The secondary shock or toxemia according to Underhill and his colleagues is due to an actual reduction of the total blood volume.

The very latest of the theories concerning the deleterious effects of thermal burns is reported by Aldrich (2) who gives Firor credit for his early work on the idea of bacterial infection in burns. He states, "Ignoring previous theories and regarding the picture speculatively, one is impressed with the fact that there is enough obvious infection present on the burned area to account for all symptoms and physical signs exhibited by the patient. It was with this in mind that Firor decided to investigate burns from a bacteriological standpoint.

"A review of the literature revealed the fact that no work had been done on the bacteriology of burns. Without any precedent to go on, it was decided to begin bacteriological studies on the patients as soon as they were admitted to the hospital, and follow these right through. The procedure was simple. At the time of admission, before treatment, cultures were taken of the burned area and of the fluid under the blebs, if there were any. Repeated cultures were likewise obtained at short intervals. For the first twelve hours, these areas were practically sterile, as was to be expected. After the first twelve-
hour period it was found that in 100 per cent of the severely burned patients and in a large majority of the minor burns, there could be grown from repeated cultures the beta-hemolytic or the gamma streptococcus. 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 the other organisms. Coincidentally, the characteristic bad effects of the burn were shown by the patient.

"It seems reasonable to suppose that when a large surface of the body has been destroyed by a burn, and becomes involved with streptococcus infection, certain ill effects should result. Furthermore, the general condition of the patient and the characteristic chart of the fever would be one which coincided with that of any widespread streptococcus infection. Additional evidence has been obtained by taking blood cultures from patients after the temperature has started to swing in the picket fence curve. These cultures were positive for the invading streptococcus. Another bit of evidence in favor of the theory is that in fatal cases the hemolytic streptococcus was found in the heart-blood and in the lungs in instances of terminal pneumonias." (2)

It is difficult to classify this theory of bacterial toxicity in either the second or third stage of the burn
phenomena, as Aldrich (2) claims it accounts partly for the symptomatology of the second stage and entirely for the symptomatology of the third stage. He cites in proof of the existence of this bacterial toxin in the stage of secondary shock that burned patients succumb during this period even when the concentration of the blood has been restored to within normal limits. However, as Brandson and Hillsman (12) have pointed out, the restoration of the blood to normal concentration of red cells in the second stage is usually accomplished by injecting isotonic saline intravenously and such therapy does not replace the serum protein deficit. The lowering of the serum protein content of the blood alone is sufficient to cause shock-like symptoms and death, even in the presence of a normal concentration of the blood as estimated by the red count and hemoglobin index (54).

It is not exactly known just to what extent the chloride depletion in the blood and the epinephrine deficiency caused by adrenal damage in severe burns contributes to the secondary shock.

The third stage of burn symptoms is generally admitted to be due to bacterial infection and, is not invariably present. This is comparatively late stage and the complications of burns usually appear during it. This stage of sepsis overlaps in its onset somewhat the end of the toxemic period. Bacterial infection is such a common sequel in burns of severe degree that it is usually anti-
cipated after twelve hours. The principal infecting organism has been shown to be streptococcus hemolyticus (15).

The very character of the trauma naturally produces a lesion which is at first sterile. However, in a burn involving any great part of the body surface it is manifestly impossible to prevent subsequent contamination. It is certain that a high percentage of severe burns do reach the actual stage of infection and a goodly proportion actively suppurate. Locally, the onset of sepsis is evidenced by a foul-smelling, purulent discharge and this is usually accompanied by fever. Septicemia may occur and terminate fatally. This septic state lasts until all the sloughs have separated and free drainage is established. This usually occurs about the end of the second week. Occasionally the sepsis persists even after separation of all the sloughs. In such cases amyloid disease is prone to occur. After toxemia, sepsis is the most important cause of death. To keep bacterial contamination at a minimum and to take every precaution against its occurrence constitutes one of the chief aims in the therapy of burns.

In summarizing the deleterious effects of thermal burns the following sequence of events seems to be generally recognized by most investigators, although the cause of the stage of toxemia is considerably debated at the present time.
1. Primary or Surgical Shock.

This occurs within the first twelve hours usually much sooner, depending on the intensity and severity of the burn. Primary shock is apparently due to a relative reduction of the total blood volume. The exact nature of surgical shock is still unknown, although it has been shown that reflex shock is one of the contributing factors causing the initial shock.

2. Secondary Shock or Toxemia.

It is concerning this secondary shock that most controversy has arisen. The various theories as to what causes toxemia have been thoroughly discussed in this paper and briefly summarized are as follows:

a. Tissue toxin theory - shock due to the toxin absorbed from the burned area.

b. Physical theory - shock due to the local loss of fluid from the blood into the burned tissues.

c. Bacterial toxin theory - Aldrich believes that there is enough obvious infection present on the burned area to account for all symptoms and physical signs exhibited by the patient. There has been some question raised as to whether the bacterial toxin accounts for all the symptomatology of the second stage or whether it explains the symptomatology of only a part of the second stage.

3. Sepsis.

As has been stated the stage of sepsis overlaps
in its onset somewhat the end of the toxemic period. It has been shown by Aldrich and others (2, 15) that bacteria can be found in the burned area after twelve hours and the principal infecting organism has been shown to be Streptococcus hemolyticus. It is during this period that the complications occur.
Clinical Course

There certainly is no set clinical course which a burned patient will follow, but there are certain symptoms and findings which tend to occur rather typically during each stage of the patient's condition.

The early symptoms following a burn are primarily those of shock with a profound disturbance of the circulatory and heat regulatory mechanism, and, in all probability, equally serious interference with many other normal functions of the body. Shock may appear a few minutes after the accident and is recognized by the usual signs, pallor, sweating, restlessness, subnormal temperature, rapid feeble pulse, low blood pressure, rapid respiratory rate, and often evidence of great pain. If the patient is a child, vomiting, and convulsions are common. The expression is anxious, the pupils are dilated. Barnes (6) believes that primary shock is the main cause of death from burns, and that the outstanding clinical characteristic of shock is a failing circulation.

About twelve hours after the burn certain clinical phenomena usually begin to appear—symptoms of toxemia. Increasing fever and tachycardia are always present and sometimes delirium and convulsions. Death often occurs at this time especially in children (67).

Two types of clinical reaction to burns have been observed following shock. In one the patient is quiet and apathetic, and does not complain of pain. In the
other he is restless, excited and in agonizing pain. Fever is a constant symptom. It may appear on the first day, reaching its maximum on the fifth or sixth day and then gradually subside. In severe cases temperatures up to 40° C. are not unusual. If the maximum is not reached by the sixth to the eighth day, this is taken as an ominous sign. The pulse in severe cases may average 48 to 52, sometimes even 80 to 100. A fall in blood pressure is noted by the second day, with return to normal as the temperature falls to normal. The respiration is accelerated.

Aldrich (2) describes the patient's course following shock in the following manner, "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 stuporous, or perhaps restless. The temperature becomes elevated, the patient complains of pain, nausea and vomiting set in. Except for a slight increase in hemoglobin, leukocytosis and a moderate increase in the blood sugar and lactic acid; there is not much in the normal blood picture, provided fluids are being forced adequately. From this time up to about the seventy-second hour, a characteristic syndrome occurs. The patient's general condition obviously grows 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 respiration are increased, and the temperature becomes
elevated in a curve that is characterized by staircase increases, until at about the seventy-second hour it reaches a maximum of between 102½ and 105° where it is sustained for a time, plateau-like. This plateau remains constant for a number of hours, when the typical picket-fence fluctuation begins. The appearance of the burn during this period also follows a marked and characteristic change.

After the first twelve hours, signs of early sepsis present themselves. The fluid over the burned areas, at first clear and odorless, becomes turbid and malodorous. The turbidity increases to the stage of frank pus and the pathognomonic odor of a burned patient is apparent. This effect also reaches its peak at about the seventy-second hour. During this time, the usual complications and sequelae such as albuminuria, extreme exhaustion, secondary anemia, and delirium take place. This sequence of events during the first three days in an extensive superficial burn is remarkably constant."

The clinical course of the patient may become complicated by complications of various kinds. These nearly always follow bacterial invasion and only too often cause death.

In the paper only a brief discussion will be made of the complications following burns.

Septicemia: This complication, while reported rarely, may well occur more frequently than is realized.
Erysipelas and Tetanus: Both of these conditions complicate burns, but fortunately occur in only a small percentage of cases.

Hemorrhage: Hemorrhage occurs only in a small percentage of cases and is usually met with only in dirty, sloughing, badly infected burns. Since the burned patient is usually in poor condition when this complication occurs it is quite serious.

Bedsores: Bedsores are seen in burned cases among the aged and those cases who are bedridden over long periods without proper prophylactic attention.

Pneumonia: This condition is one of the most common complications in fatal cases of burns. In those cases dying late in the course of the sequence, pneumonia or nephritis are the usual causes of death.

Nephritis: Kidney damage occurs early in the course of burns. In fact, this condition may be taken as a part of the phenomena of burns and not classed as a complication. The early occurrence of kidney damage warns us that unguarded intravenous fluid therapy after forty-eight hours may be dangerous. The early kidney damage may be accounted for by any of the theories advanced, but the late stage is undoubtedly due to bacterial toxicity.

Amyloid degeneration: Amyloid degeneration is not a peculiarity of burns, but is found in this condition, as elsewhere, after long continued suppuration.

Curling ulcer: Although ulcers are found all along
the gastro-intestinal tract, the more common location is in the duodenum. It was first described by James Long (27) in 1840 and later by Curling (17) in 1842. Perry and Shaw (41) reported duodenal ulcers as being routinely found in 0.4 per cent of all cases coming to autopsy and in 3.3 per cent of all persons autopsied after severe burns. Upon investigation it has been found ulcers rarely become chronic, but either perforate or heal.

The mechanism of its production is still unknown. Curling (17) believed hyperactivity of Brunner's glands to be responsible. Hunter (27) thought emboli to be the etiologic factors. Certain investigators (20, 31, 35) believe that suprarenal damage in some manner explains the ulcers. The question still remains unsettled.
Treatment of Thermal Burns

After surveying the literature on the treatment of burns it was decided that the following outline would adequately and completely cover the treatment and management of thermal burns.

1. General treatment of the individual.
   a. Shock.
   b. Toxemia.
   c. Infection and Secondary Anemia.

2. Local treatment of burned areas.
   a. Immediate treatment of burned area.
   b. Treatment of granulating surfaces.
   c. Skin grafting.

1. General Treatment.
   a. Shock - The immediate treatment of the patient in shock is similar to that of any patient in surgical shock. This treatment should also be used to prevent shock if it has not as yet occurred. Clothing should be removed with as little trauma as possible, and if necessary the patient first placed in a warm sodium bicarbonate bath to make removal less painful to the patient. Heat in the form of the electric crib or by means of hot water bottles is used to combat heat dissipation and to restore normal temperature. Morphine or codeine should be given freely for the pain. The ideal treatment must exclude all confusion and excitement in the room. Every effort to obtain the patient's confidence should be made,
at the same time allaying apprehension. The room should be kept very warm, 90°F. or over. The foot of the bed should be elevated. An intravenous injection of isotonic saline is given to combat the relative reduction of the total blood volume. Bancroft (4) recommends a hot intravenous infusion or hypodermoclysis of 5 per cent glucose in normal saline. The amount of saline injected intravenously during the stage of primary shock should be a quantity equal in amount to the patient's total blood volume. In a man weighing 150 pounds it is thought this would amount to 5,000 c.c. By rapidly injecting this amount of saline intravenously the relative blood volume is apparently restored. When the tone of the capillary bed recovers, the circulation will have an excess quantity of fluid present to combat the primary shock. Blood transfusion at this stage is of unestimable value. Caffeine or epinephrine for stimulation if indicated. These measures should successfully combat the primary shock.

b. Toxemia - The relation of fluid and sodium chloride depletion to secondary shock or toxemia has already been thoroughly brought out and the physiology will not be repeated.

The restoration of the fluid lost from increased permeability is merely a question of the quantity and kinds of fluid one should give the patient. The quantity is determined in two ways, first, the surface area involved, and, secondly, the variations of the red blood
cell count and hemoglobin index. By following the red blood cell count and the hemoglobin index, taken each time from the same site, one has a simple way of determining the degree of blood concentration and the patient's fluid requirements. As a general rule fluids should be forced until the blood concentration is restored to normality. During the first forty-eight hours following a burn, when it can be reasonably assumed that the patient's heart and kidney are functionally normal, there is no danger of forcing isotonic fluids in any quantity. This fact has been proven by Smith and Mendel (52) who have shown that the excess fluid is eliminated by the kidneys. The intravenous method is the most common method of supplying the fluid but the oral and rectal routes should not be neglected.

The kind of fluid used is very important. It has been shown by Underhill and his associates that the fluid lost is almost analogous to blood serum. Thus, the ideal fluid to give would be blood serum if this were possible. Normal saline solution approaches nearer the nature of blood serum than any other fluid commonly used. The water necessary to restore blood volume is present in this solution and it also contains the sodium and chlorine ions so necessary to the maintenance of the acid-base equilibrium and the electronic concentration.(28). Bancroft (3) believes a blood chloride determination should be made every third or fourth day if possible. He recom-
mends giving subcutaneously 2,000 to 3,000 c.c. of normal saline during the first four days and if the patient shows any evidence of vomiting, 500 to 1,000 c.c. of a 2 per cent saline solution or 500 c.c. of 3 per cent saline given very slowly intravenously and repeated if vomiting recurs.

But in order that the colloidal content of the blood stream will be sufficiently high to prevent the saline injected from diffusing out into the tissue spaces the serum protein lost on the granulating surface must be restored (23). Reduction of serum protein content of the blood has in itself been proven to be dangerous and often fatal (24). The simplest way to restore serum protein lost is by blood transfusion. About 1000 c.c. of blood should be given to adult patients in the first twenty-four hours and, in the severe burns, more should be given if necessary.

Because experimental work has shown the suprarenals are badly traumatized and for several days after the injury there is a marked decrease of adrenalin in the blood, some men recommend the intravenous administration of epinephrine. This measure is of more importance if the patient remains in continued shock and if the blood pressure remains low. A prophylactic dose of tetanus antitoxin is usually indicated (26).

c. Infection and Secondary Anemia - In the general treatment of burned patients the infection and secondary
anemia must be considered. This is especially important if there are extensive granulating areas since then there will be marked loss of fluids and red blood cells, and infection is very likely to be present. Small transfusions, frequently given, help to develop the resistance of the individual and, also, to overcome any secondary anemia. About the only method which has had any results in the saving of life after severe toxemia has set in, is that of exsanguination-transfusion. This method reduces the mortality from toxemia by one-half (45). Its disadvantages are that it does not modify in the least the production of the toxin, and it requires a lot of blood. At this time good food and fresh air are essential. If the patient shows evidence of a nephritis in three or four weeks, probably due to toxin absorption and infection, it is advisable to force fluids to aid the condition.

2. Local Treatment.

a. Immediate treatment of severely burned areas-

It should be apparent that there is no single ideal treatment for burns and no one procedure applicable to all phases of the problem. In reviewing all the pathological physiology of burns it is logically concluded that we must apply a substance on the burned area which will tend to do the following:

1. Reduce shock.

2. Tend to prevent the absorption of toxic material.
4. Prevent infection.

There are at present various methods which are used in the local treatment of severe burns. Although there is considerable controversy as to what substance should be used locally the various methods used all tend to meet the above requirements. The clinics advocating the use of coagulants admit the possibility of infection and in using the coagulating substance to combat the absorption of the tissue toxin and also to prevent the escape of plasma and water, they consciously or unconsciously practice the debridement taught by the bacterial toxin school. On the other hand, the clinics which believe in the bacterial toxin theory use various bactericidal agents as gentian violet, mercurochrome, and silver nitrate, and in so doing precipitate protein which forms an eschar. This eschar is likewise chemical debridement and would also retard the absorption of a tissue toxin should it be present. If the clinician believes the secondary shock stage in burns is due to fluid loss the application of a coagulant or any of the above mentioned bactericidal agents would still be satisfactory because of the following factors: Firstly, either of these procedures will decrease pain. Secondly, it is easily possible that the precipitation of protein to form the eschar may lessen fluid loss by the early establishment of the electronic balance. Therefore, ir-
respective of which type of toxin one believes in, the principles of treatment are carried out by the use of coagulants and the use of various antiseptic and bactericidal agents.

It is evident that either of the types of the above solutions causes chemical debridement and thus lessens the possibility of bacterial contamination and the third (septic) stage of burns.

Unfortunately, many patients who have been given first aid treatment for burns have had some type of oil or ointment applied over the burned area. While this tends to relieve the initial pain, it does not prevent the absorption of toxic products, nor does it in any way hinder infection. Further, it is a serious detriment if one wishes to use other methods of treatment.

First Degree Burns: These may be treated in almost any manner; wet dressings of any type—sodium bicarbonate, tannic acid, or picric acid—are efficacious. Many types of ointment help to relieve the initial pain.

Second Degree Burns: The burn is of a very superficial type. The blisters are punctured by a sterile needle introduced into the side, followed by a compression bandage, which, if not disturbed for three or four days, allows the epidermis of the blister to become vascularized with complete and rapid cure.

The early local treatment of severe burns is nearly the same no matter what method of treatment is later
adapted. Should the patient not rapidly recover from his shock he should be immediately immersed in a continuous hot (90° - 100°) saline or boric acid solution bath. The clothing should be cut away after the patient has been immersed and not previously.

Regardless of time, the patient should be suspended in the bath by means of a sheet until he has fully recovered from his shock and should be reimmersed immediately should he, on removal, show signs of recurring shock. It is suggested that the salt solution furnishes nutrition to the devitalized cells that cannot be reached by the blood current because of the occluded superficial capillary circulation (13).

The area is next cleaned under general anesthesia. Nitrous oxide and oxygen is the anesthetic of choice, but ether can be given with safety. The burned area should be cleaned of all foreign matter and mechanical debridement carried out as far as is compatible with the patient's general condition. The raw surface is gently swabbed, first with ether or alcohol and then with a 1:1,000 corrosive sublimate solution. Avoid vigorous rubbing or scrubbing. The area is now ready to be treated with some coagulating or bactericidal agent.

The following types of local treatment are the more common present day methods of treatment of burned surfaces:

1. Tannic Acid Coagulation.
2. Ferric Chloride Coagulation.
3. Mercurochrome Treatment.
4. Gentian Violet Treatment.
5. Tannic Acid - Silver Nitrate Treatment.

The various advantages and disadvantages of each treatment will be discussed and a technique of application of each treatment will be given.

Tannic Acid Coagulation.

Davidson (18) acting on the theory that toxemia from burns is due to some product of protein decomposition which occurs in the burned area, attempted to find some substance which would diminish pain, seal the lymphatics, thereby preventing toxic absorption, and act as a moderate anesthetic. Having had his attention called to the similarity of tannic acid and phosphotungstic acid in precipitating proteins, he started investigation along these lines, and proposed the use of tannic acid as the primary treatment of the burned area.

Tannic acid 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. It has been shown that if tannic acid is applied in concentrated solutions the acid may penetrate before superficial coagulation occurs, and in such event a deep caustic action may result. The eschar formed by the precipitated proteins provide a protective coating.
against chemical and mechanical action and act as a splint for the growth of epithelium arising from the hair follicles. Tannic acid is easily soluble in aqueous solution and a 5 per cent solution is the most satisfactory.

Davidson summarized the advantages of the tannic acid regime as follows: (1) it lessens toxemia, (2) is analgesic, (3) minimizes trauma, (4) conserves body fluids, (5) limits secondary infection and consequent scar formation, and (6) forms a scaffold for growth of young epithelial cells.

Technique of Tannic Acid Treatment: The burned area having been cleaned with ether and alcohol as previously described, dressings saturated with a freshly prepared 5 per cent aqueous solution of tannic acid are loosely applied. These are kept saturated with the solution for twenty-four hours, or until the burned area is tanned a mahogany brown. At the present time it is the practice of most men to spray the tannic acid over the burned area every fifteen minutes for the first four to six hours. The time required for the spraying depends upon the depth of the burn and the reaction of the tissues to the tannic acid. Barnes (6) uses a 10 per cent tannic acid in the effort to penetrate to the bottom of the burn and to secure at once a sterile scab, a sealed wound and a dry, noninfected and non-infective field. No dressings are required when a spray is used. A tannic acid jel
should be used for facial burns.

McClure and Allen (34) use the following method of procedure in the treatment of a burn case:

1. "The grime of work is removed without undue scrubbing, using ether or benzine.

2. "The surrounding unburned skin area is thoroughly cleaned with soap and water.

3. "The blisters are widely opened under careful aseptic technique, using sterile instruments and sterile gloves, and the loose areas of skin are removed. The extent of the debridement and scrubbing must depend on the patient's condition. Too much manipulation may aggravate or produce shock.

4. "The patient is placed in bed on sterile sheets.

5. "The burned area is sprayed with a 5 per cent aqueous solution of tannic acid every fifteen minutes until a dark mahogany coagulum is obtained. This usually requires from ten to eighteen hours.

6. "A cradle to support the bed covers is placed over the patient.

7. "Sufficient electric lights are introduced between the covers to provide a temperature of 90°.

8. "The greatest care is taken in the handling of these patients in their rooms and so far as technique is concerned one must treat them exactly as one would treat a surgical incision.

9. "The coagulum is allowed to remain in place
until it frees itself and curls up at the edges as healing takes place, at which point it is cut away.

10. "Should serum collect underneath, as may occur when there has been complete destruction, or should infected fluid cause its loosening, the coagulum should be removed sufficiently to allow drainage and treatment.

11. "The denuded areas which are left after loosening and removal of the coagulum has taken place, should be immediately prepared and early skin grafting carried out."

In the typical case of first and second degree burns, the tanned membrane usually begins to curl at the edges after about two weeks, and it is seen that epithelialization has already been initiated. If infection is not present, the membrane gradually separates having beneath it newly formed epithelium and granulating areas of relatively small extent, dotted here and there with patches of epithelium.

In burns of the third degree the membrane is lifted by the serum after a time and must be removed. The granulating surface then presents the same problem as it does after any type of treatment.

The presence of infection is evidenced after four or five days by one or more collections of fluid under the tanned membrane. It is generally agreed that the best way to handle these collections of fluid is by making incisions and irrigating the area with some solution.
after the detached membrane has been excised. Firmly adherent membrane should not be disturbed. Lee (28) recommends cross-cut incisions through the infected area, dividing it into two-inch squares to permit drainage around the edges.

Various solutions have been used to wash out these infected patches. Barnes (6) says the open areas should be covered with paraffin gauze over which dressings moist with physiologic solution of sodium chloride or boric acid are laid. Strauss (55) believes that warm compresses of warm potassium permanganate or Dakin's solution should be applied as compresses after the precipitate is removed. Aldrich (1) has obtained his best results by using one per cent gentian violet. Bancroft (3) believes that when a patient has a high temperature and there is redness around the periphery and tenderness beneath the epithelium, it is advisable to anesthetize the patient and excise the membrane as far as possible. Wet dressings are then applied to the granulating area.

Results of Tannic Acid Treatment: In a series of 114 cases at the Lincoln Hospital the mortality was reduced from 50 per cent to 20 per cent. Previous treatment consisted of picric acid, electric cradle, oils, ointment or debridement. McClure and Allen (34) found no decrease in mortality after treating 476 patients by the tannic acid method. Their series of patients extended over a period of ten years. Barnes (6) in speaking of
the tannic acid treatment says that tannic acid treatment saves more extensively burned patients than any other treatment. Pemberthy (39) states that the use of tannic acid has materially reduced the mortality and has resulted in the saving of many lives. Aldrich (2) after using all the more generally accepted treatments of burns has shown the tannic acid technique superior to others.

Ferric Chloride Coagulation.

Slack (51) has reported the use of tincture of ferric chloride on burns as far back as 1891. At the present time Coan (14) is one of the most enthusiastic advocates of the use of ferric chloride as a coagulant. He recommends the use of 5 per cent aqueous solutions, made by dissolving commercial anhydrous ferric chloride in distilled water, and filtering. In some cases he has alkalized the solution with sodium hydroxide.

In technique of application there is no essential difference between tannic acid and ferric chloride solutions. A pledget of cotton on a wooden applicator is sufficient for small areas. In more diffuse areas atomizers are preferred. Infected areas are treated by the application of some antiseptic as gentian violet or mercurochrome.

Coan (14) compares the relative merits of the ferric chloride and tannic acid solutions as follows:

1. Ferric chloride solutions are more stable and rather less irritating.
2. The consistency of the coagulum is such that early mobilization of joints is usually possible without disturbing a ferric coagulum; this has not been possible with tannic acid.

3. It is possible to discover infected areas much earlier under the coagulum formed by ferric chloride than under that formed by tannic acid.

4. There is a tendency for ferric chloride to stain the hands and skin.

5. With ferric chloride one sees wrinkling of the coagulum with movement of joints, and after swelling subsides, without any loosening. This is the greatest advantage of ferric chloride over tannic acid.

Coen (14) has reported excellent results with all his cases. In reviewing the literature it has been noted that the use of ferric chloride is not as yet very extensively used.

Mercurochrome Treatment.

Turner (57), a British clinician, in search of a drug which would have strong antiseptic and bactericidal properties and at the same time help bind the crust of the burned area, selected mercurochrome to possess the necessary qualities. He gave the drug a trial for a period of one year and made the following observations.

1. Mercurochrome does not precipitate protein, and can therefore be claimed as an effective antiseptic in the presence of protein.
2. The crust formed is thin and transparent.

3. Bed linen is not in any way destroyed.

4. It is non-irritating to the tissues.

5. A 2 per cent aqueous solution is stable for an indefinite period.

Technique and Results:

1. Complete the general toilet and then swab over with a 2 per cent aqueous solution of mercurochrome. Then dry the surface with some form of dry heat.

2. The following routine is recommended: first day, four applications; second day, three applications; third day, two applications.

3. If any area of infection occurs—easily seen through the semi-transparent crust—remove the crust and swab the area with normal saline and reapply the mercurochrome treatment.

Conclusions: Turner (40) concludes as follows: "Our results have been most encouraging. The rapidity and reliability with which desiccation has taken place have been iminently satisfactory and with adequate preliminary treatment we have been able to reduce sepsis to a minimum. On a general survey of our cases it would appear that patients treated with mercurochrome have less general reaction during convalescence than those treated with other agents, while the amount of pain and discomfort compare well with any other method. On no occasion have we seen any toxic effects following the use of this drug."
Gentian Violet Treatment.

Aldrich (1, 2) believing that all the symptoms of the patient, excluding shock, could be explained on the bacterial infection, aimed his treatment at killing the organisms. He used the analine dye, gentian violet, and developed the gentian violet treatment of burns.

Gentian violet has two valuable characteristics which answer the problems offered by a burned patient; it is a specific antiseptic for the invading organism and because of its pH reaction, together with the contained methyl radical, it reacts with the burned flesh to form a thin, light eschar, tough but flexible. This eschar when situated in the folds of the body will bend but not crack. The burn is thus sterilized and protected from further infection by an almost ideal protective eschar. The dye also produces instantaneous analgesia.

Technique of the Gentian Violet Treatment: "Unless the burned surface is covered with oil there is no need for any preliminary cleaning. The dye in one per cent solution is sprayed on the burned area immediately. For the first few hours the gentian violet is sprayed on the burned area until a light eschar is formed. The wet oozing areas rapidly become dry and tough, further infection is thus kept out and the loss of fluids through the serous ooze eliminated. After the eschar is formed the patient is sprayed every four to six hours during the day. Any blebs that have formed are opened and the un-
stained portions then presenting are sprayed. This is continued until healing is complete.

"Any epithelium which has not been destroyed will continue to live and spread rapidly under a gentian violet eschar. If the burn is so deep and extensive that skin grafting will be necessary, the eschar is allowed to remain on for about three weeks after which time it can be softened and removed by warm compresses of sterile salt solution." (2) Fatherree, Kennedy, and McSwain (21) recommend a gentian violet jel for facial burns.

If a patient presents himself with an old burn already septic, the treatment can also be instigated with no initial clean up. The necrotic matter and pus is likewise converted into an eschar, but this eschar is irregular, in pieces, and floating. This eschar is usually removed every day and the area sprayed immediately afterward. Otherwise the treatment is the same as for fresh burns.

This treatment is simple, not characterized by painful daily dressings. The patient does not feel bad, has no apparent pain, is not flushed or stuporous with fever. The fever, if any, is moderate, and not of the septic type. There is no evidence of increasing anemia since there is no blood destruction. Fluid loss is at a minimum and less intravenous supportive treatment is necessary. The burn remains clean and thus the difficulty of handling a malodorous patient in the open ward
does not arise. If any infection does occur the softening is seen much sooner under a gentian violet eschar than under the eschar of tannic acid. Gentian violet has little, if any, injurious effect on living tissue and, therefore, it may be used freely without running any risk. (7).

Tannic Acid-Silver Nitrate Treatment of Burns.

Bettman (7) recently introduced a new method of treating burns which he believes to be an improvement on the tannic acid method. The new method consists in the use of a 5 per cent tannic acid solution, followed by a 10 per cent silver nitrate solution. There is an almost instantaneous sealing of the burned areas, the fluid loss is stopped almost at once and the treated burned tissues are made insoluble and non-absorbable. At the same time an antiseptic covering is supplied and the patient is made comfortable.

Technique: After the burned area has been adequately cleaned a 5 per cent tannic acid solution is applied by means of cotton swabs to all burned areas. This area is next sponged over with a 10 per cent solution of silver nitrate. Drying and keeping the tanned areas dry is very important. Any blebs or areas of infection are opened after twelve to twenty-four hours after the first treatment and treated as before. The coagulum is removed at the earliest possible moment. There is no danger of argyria.
Bettman (7) has treated 21 cases by this method and has had very gratifying results. He gives the superior points of the Tannic Acid-Silver Nitrate Treatment as follows:

1. A more rapid method of tanning.
2. Immediate stopping of the loss of body fluids, thereby preventing the concentration of the blood.
3. Prevention or very definite minimizing of shock because of the rapid formation of an eschar.
4. Immediate prevention of the absorption of toxic product.
5. Prevention of infection by the short period of application of moisture and the early drying.
6. Less injury to the kidneys and other organs from the effects of fluid concentration and the absorption of toxins and infection.
7. Greater comfort for the patient.
8. Patient is safely carried past the first twenty-four hours which is the most critical period following a serious burn.
9. Avoids the second critical period, that of infection and absorption of toxic products.
10. Simplification of the nursing problem.
11. Formation of a thin, flexible coagulum.
12. Speedy healing.
13. Prevention or minimizing of heavy contracting scars by early rapid healing in the absence of infection.
14. Lessening of the amount of skin grafting and secondary corrective surgery necessary.

The use of paraffin and various oils in the primary treatment of burns are rapidly being discarded. Mechanical debridement, as a primary measure has no place in the treatment of burns. Picric acid has been replaced by the more popular tannic acid, although butesin picrate is still used by many clinicians for superficial facial burns.

Of all the various methods of immediate local treatment of burns which have been discussed in this paper, the tannic acid treatment seems to be the most extensively used at present. However, the use of such agents as gentian violet and the silver nitrate-tannic acid combination may prove to be improvements on the tannic acid treatment of burns in the future.

After the mechanical removal of the tannic acid membrane in infected cases, or after the natural separation of the membrane in deep third-degree burns, the treatment of the granulating surfaces becomes a surgical problem. Each clinic has its own particular method but the main methods of treatment may be briefly stated as:

(1) The use of wet dressings.
(2) The use of ointments.
(3) The use of pressure.
(4) Some form of paraffin.
(5) Ultra-violet ray.
The aim of the treatment is to clear the infected granulating tissue if it is infected, and then to stimulate the granulations. Blalock (10) advised treatment with 1:5000 neutral acroflavin. The dressings are loosely applied and kept saturated with the solution. The granulations beneath then become red and clean, and epithelialization occurs rapidly. If infection has been prevented, epithelialization occurs rapidly from the healthy islands of epithelium and from the hair follicles even before the coagulum is completely removed.

Many men advise the use of paraffin as a dressing. Paraffin gauze seems to be the simplest and most efficient. First several layers are applied over the granulating surface, then pads or surgical gauze wet with boric acid solution, and finally either rubber or sea sponge. These sponges are held in place by a pressure gauze bandage, and the whole dressing is kept wet constantly with the boric acid solution. This dressing promptly converts exuberant, pale, boggy granulations into firm, bright pink ones. (6).

If the granulating surfaces begin to become pale and healing is not proceeding rapidly, they may be stimulated by ultra-violet rays. Trusler (56) recommends daily exposure of infected granulation tissue to ultra-violet ray with intermittent soaking in normal or hypertonic salt solution with one-half per cent chorazene or other mild antiseptic solutions. He believes that if
the granulations are in a bad state the ultra-violet ray should be applied at close range for as long as five minutes, the time being reduced one or two minutes daily as the surface improves. The granulations should become red, vascular and free from exudate. Small transfusions frequently help the individual to improve his own repair. Granulation tissue continues to form fibrosis at its base until covered with epithelium. Grafting should be done as soon as healthy granulations form, to prevent fibrosis.

Skin Grafting.

In a general way, it may be said that the best treatment for granulating surfaces, after the separation of the slough, if the surface area is large, is skin-grafting. The question of the type of graft used and the technique of applying it will be described only briefly in this paper.

Many failures in skin-grafting occur in areas in which infection of the granulating surfaces has persisted for a considerable time. If an exuberant granulating tissue persists, scar tissue forms beneath the superficial granulations. Thus, unless the scar tissue area is removed, the skin-graft will not derive sufficient blood supply to persist. Therefore, all scar tissue should be excised along with the granulations before grafts are applied. The earlier the skin-grafting is done the less contraction will be found in the resulting scar.
Where appropriate, thick split skin grafts are used in preference to the full thickness grafts, because of the greater assurance of their "take", the shorter time necessary for the operation for healing, and the lesser deformity of the donor area. Such grafts may be cut almost full thickness if desired, but even the thinner ones have enough derma to give the needed anchorage that is lacking in spontaneous epithelialization of a defect. While full thickness grafts make better repairs than split grafts, their growth on anything but a sterile field is too uncertain to warrant their use in the presence of even a mild infection. Full thickness grafts are used only when the best possible early bearing surface and cosmetic result is desired, unless it is necessary to use pedicle flaps. (9). Brown and Blair (13) recommend a graft which includes from one-half to three-fourths of the thickness of the skin, utilizing grafts as large as 15 by 5 inches if possible.

In children with extensive burns who have not much normal skin that can be used for grafting, pinch grafts can be inserted 1 cm. apart, and epithelialization will occur from each graft and bridge the defect. It is better to attempt small areas at a time, repeated operations being performed. It is best to graft near the advancing edge of the epithelium, for if pinch grafts are put in the center of the granulating area they may be choked off by the surrounding granulating tissue.
If the granulating area is rather small Thiersch grafts imbedded in the granulations are advocated by Strauss (55).

The care of the grafted area requires a simplified method of applying a pressure dressing and keeping it moist if necessary. If the area is quite free from contamination a sponge pressure dressing is applied with a few layers of grease gauze over the graft. If there are any reasons to fear a degree of infection that might damage the graft, a wet saline dressing with irrigation tubes incorporated in it is put on and pressure is obtained over the area with sea sponges bound on firmly with heavy gauze rolls. The dressings are kept constantly moist for four days at which time the first dressing is changed.

Burns of the hands deserve special attention because every effort should be made to prevent the deep infection that will rapidly fix tendons and joints. The first treatment is active surgical drainage. Next active movement encouraged (8); the fingers should be dressed apart, and the whole hand kept in position of function. The use of splints has proven valuable in the care of burned fingers.

In all instances where function is important the extremities should be in such a position as will prevent contractures and adhesions. Early movement is advisable, unless there is danger of breaking down the grafted surface.
When patients have healed deformities, it is necessary to determine the extent of the original loss and the tissues available for repair. To overcome the deformity complete relaxation of the tissues must be attained by removal of the binding scars, and the resulting raw surfaces covered with tissue of suitable thickness.
Summary

In this paper the author has attempted to show that the phenomena induced by thermal burns are in reality a sequence of events and should be treated as such. Having covered the physiology and pathology, together with the clinical course of the burned patient, several methods of treatment were presented with a rather thorough explanation of the various principles involved. The following material was covered in this paper on burns:

1. A brief history of the problem of burns.

2. The pathology of burns, including local and general pathology.

3. The pathological physiology, covering the problems of (1) primary shock, (2) toxemia or secondary shock, and (3) sepsis.

4. A few of the complications of burns were discussed.

5. The treatment of burns was discussed covering both the general and local treatment. Special emphasis was placed on the immediate local treatment of the burned area.
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