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Physiology and treatment of thermal cutaneous burns

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THE PHYSIOLOGY AND TREATMENT OF THERMAL
CUTANEOUS BURNS

SENIOR THESIS

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INDEX

Introduction.....1
Definition.....3
Classification.....4
Pathology.....8
Pathologic Physiology.....18
Treatment.....33
Complications.....53
Conclusions.....55
Bibliography.....56

INTRODUCTION

The treatment of burns dates back to the beginning of civilization. Burns have been a problem of mankind ever since the discovery of fire. Just when or how it was discovered is unknown, although Grecian mythology (5) teaches that the fire was stolen from the gods on Mount Olympus and given to man to differentiate him from all other animals. The purpose has no doubt been fulfilled and along with this fulfillment man has come to suffer that very common injury, namely a burn.

Even today, centuries later, burns offer one of the greatest problems to the surgeon and general practitioner. Statistics (54) show that annually between six and seven thousand people die of burn injuries in the United States alone. Forty-five per cent of these are children under the age of six.

Until recently the treatment has not been well understood and much light may still be thrown on the subject during the next few years. It seems that each new trend in medical progress tends to change the general ideas concerning the treatment of burns. Many of these ideas have been handed down from generation to generation, largely due to lack of better substitutes.

Even today little is known concerning causes of death due to burns, but there have been some very logical theories advanced and at least partially substantiated by experiments.

The fact that the mortality and morbidity rates have decreased tremendously during the last ten years shows us that the treatment is undoubtedly more adequate now.

DEFINITION (3, 33, 34, 45)

A burn, in the true sense of the word, is an injury to the body resulting in tissue gangrene, produced by dry heat, the degree of which is higher than is compatible with the normal integrity and metabolism of the part affected.

A scald is the same condition but is due to moist heat. Since a scald and a burn are practically the same and are treated in the same manner we have come to designate both conditions a burn. However, generally speaking, a scald is usually not as severe as a burn.

CLASSIFICATION

I. The Types of Burns. (3)

We speak of various types of burns. The more common of these are:

1. Ordinary burns, which, as was stated, are due to the application of dry heat.

2. Scalds, which are due to the application of moist heat.

3. Chemical burns, which are due to the application of caustics, such as acids, alkalis, phosphorus, iodine, bromine, etc.

4. Mechanical burns, which are due to friction with some rapidly moving object.

5. Radiant burns, which are due to roentgen ray, ultra-violet ray, or high frequency current.

II. The Degree of Burns.

There are many different classifications of burns, and the ones most commonly used depend upon the country in which the writer has had his training. The more common classifications are:

A. Morton's classification. (45)

Although this classification is attributed to William Morton, who did his work in the latter half of the nineteenth century,

the same classification is also given in Heister's textbook of surgery (31) which was written about the middle of the seventeenth century. The classification is the one most commonly used in America and Germany today. It gives three different degrees:

1. First degree....which is the degree in which there is a mere formation of erythema.
2. Second degree...in which there is formation of vesicles and bullae.
3. Third degree....in which there is formation of eschar and gangrene.

B. Heister's and Callison's classification.(31)

This classification dates back to the middle of the eighteenth century. It is still very commonly used in America, being almost as popular as Morton's classification. It consists of four degrees, which are:

1. First degree....which is the degree of erythema.
2. Second degree...formation of vesicles.
3. Third degree....formation of eschar.
4. Fourth degree...charring of the tissues.

C. Dupuytren's classification. (45)

This classification is most commonly used in England and France. It dates back to the

early part of the nineteenth century and is attributed to Dupuytren, who was one of the pioneers in the study of surgery in France.

It consists of six degrees, which are:

1. First degree....erythema of the skin caused by a temperature of about 140 degrees F.
2. Second degree...vesication caused by a temperature of 160-210 degrees F.
3. Third degree....destruction of the cuticle and part of the cutis vera, the tips of the papillary downgrowths remaining intact. It is caused by a temperature of about 210 degrees F.
4. Fourth degree...destruction of the entire integument and part of the subcutaneous tissue and is caused by a temperature of 210 degrees F. and more, over long exposures.
5. Fifth degree....encroachment on muscles.
6. Sixth degree....disorganization and charring of tissues.

D. Goldblatt's classification. (28)

This is the most recent, recognized classification and is also the simplest. From a clinical standpoint this classification would easily suffice, consisting merely of two types, which are:

1. First degree....erythema and vesicle formation, which heals without scarring.
2. Second degree...includes all other burns of a more severe nature, which usually heal by formation of scar tissue and contractures.

PATHOLOGY

There are various factors which play a part in the severity of a burn and also to the degree of pathology. These factors are:

1. the duration of contact with the heat,
2. the degree of temperature,
3. the susceptibility of the tissues in contact with the heat.

In considering the local histology of the burned area three stages may be noted. These are:

1. the stage of destruction or burning,
2. the stage of inflammation and sloughing,
3. the stage of repair.

For simplification it is best to use Dupuytren's classification in the histological study of burns, as given by Pack (45).

The first degree burn begins with a simple erythema. The vascular reactions are similar to those in any inflammation, consisting of a short period of contraction, followed by a dilation of the arterioles and venules. This local widening of the small vessels, due to direct action of the irritant, is responsible for an increased rapidity of the blood flow to the injured part, causing the area to be warmer and redder. Immediately surrounding this area is a widely spreading irregular margin, exhib-

iting a bright red color, due to the increased flow of arterial blood, which is the result of a local reflex causing a dilation of the arterioles. As the blood vessels widen, the blood flow slows and an active congestion takes place. There is an increased permeability in the vessel walls so that filtering of the plasma in the tissue spaces takes place. Underhill (66, 68) and his co-workers have shown in their experimental work by injecting Methylene Blue into the blood-stream that the permeability is only in one direction. They also showed that the fluid was first extravasated into the periphery of the injured area and that the fluid extravasated was very similar to the blood plasma.

This superficial skin edema is responsible for the low flat wheals of various sizes that are found. Within the blood vessels, there is an increase of leukocytes near the margin of the burned area, with a subsequent migration to the tissue spaces, followed by diapedesis of the red blood cells. The period of edema lasts from twenty-four to forty-eight hours. Within a few days the upper layer of the epidermis separates in the form of scales, or it may peel off which is usually the case in the ordinary sunburn. Any pigmentation which remains disappears in time. The burned area may show an increased redness for a short time, but no scars remain.

In the second degree burn vesication takes place. The epidermal cells undergo a true coagulation necrosis, due to the conversion of their soluble colloids into their insoluble modification. An exudation of the fluid passes from the tips of the papillae into the superficial layers of the skin and the cells which have been killed soon liquify and are dissolved completely. There is then an infiltration of mononuclear leukocytes. The burned area after twenty-four to forty-eight hours oozes slowly. The fluid finally coagulates, becoming jelly-like. Organization then takes place. Fibrous tissue grows into the fluid. The skin becomes thin and prepares to slough. Sloughing occurs and the skin assumes its normal appearance. There is no scarring due to the fact that the corium is not involved.

In the third degree burn, the epidermis is completely destroyed as well as part of the corium, but the tips of the interpapillary processes remain intact. This is the most painful type of burn because the sensitive terminal nerve filaments are left bare and exposed. The papillae of the skin appear as a reticular framework, containing serum, bits of remaining living epithelium, leukocytes and masses of fibrin. The papillae of the injured portion are visible as red points on a white background. The subpapillary plexus of blood vessels and lymphatics

absorb the toxins formed in the burned skin. Occasionally these toxins are taken up from the blood stream by the sweat glands and are excreted in other parts of the body, producing a rash simulating scarlet fever.

The sebaceous and sweat glands as well as the hair follicles are deep enough to remain uninjured so that when the process of healing begins, each of these structures serve as a focus of potential epithelial growth, and the denuded area is quickly covered with new epithelium. This process can well be compared to the restoration of uterine epithelium following menstruation, where the deep glands serve as the potential focus of new epithelium.

The formation of this new skin requires from fourteen days to four weeks. The resulting scar is white, elastic, possessing all of the structural elements of the true skin and does not contract.

A fourth degree burn, according to Dupuytren's classification, is a destruction of the entire integument. In every burn there are two layers to be considered: (1) the destroyed or necrotic tissue, and (2) the injured or damaged tissue just beneath it. In this degree of burn the tissues have been killed. The dead skin forms an eschar, which has a brownish-black color, almost having the appearance of leather, if produced by a flame, or has a white, marble-like appearance if produced by moist heat.

the white color is due to the pressure inflicted by the moist heat and also to its greater penetrative powers not permitting the blood to flow through the corium. The eschar is insensible to touch since the nerve endings have been destroyed.

The eschar is depressed below the level of the skin, which is contracted around it, showing puckered folds radiating from the periphery of the destroyed area.

The area about the eschar gradually fades into an area of third degree burn followed concentrically by areas of second and first degree burns respectively.

In a short time an acute inflammatory process starts around the retracting eschar, and a groove results, intervening between the edges of the dead and living tissues. This is the first step in the process of sloughing, and normally lasts about two weeks unless steps are introduced to speed up the process.

The eschar is often cracked or fissured, especially near the joints, where movement occurs. If the fissures extend deep enough so as to reach the adipose tissue, the fat cells lose their oily contents and the released fat flows out on the surrounding skin.

It is of interest to note that the small arteries and veins, as well as the nerves, sometimes

retain their vitality in the midst of disorganized tissue.

Healing begins soon after the injury but it is not so evident until after sloughing or mechanical removal of the dead tissue. The remaining debris is cleaned up or liquified, partly by autolysis, partly by leukocytic digestion, and the residue either flushed off the surface or absorbed by the lymphatics. At this stage infection very frequently sets in.

The raw surface is covered with a fibrinous exudate, which influences the growth of new tissue cells. The blood vessels become plugged with thrombi, but from these, masses of endothelial cells, accompanied by fibroblasts, grow out along the fibrinous framework and organize it into new tissue, spoken of as granulating tissue. These masses of endothelial cells form new anastomosing capillaries which arch and thereby give the granular appearance to the surface of the new tissue. This tissue is in time covered by a thin bluish film of epithelium, which grows in from the periphery at the rate of about one-eighth inch per week. This new epidermis later becomes thicker and opaque. A good deal of contraction occurs and scarring is inevitable. The scar by its contraction is a common cause of deformity. The granulation tissue has a marked tendency to become overabundant and luxurious, a condition which

hinders epithelialization, and makes the resultant scar irregular, inelastic and contracted. The scar is usually smooth and shiny due to the absence of hair follicles and sweat glands. The border of the scar is irregular, indented and occasionally stellate.

In the fifth degree burn the muscles are involved. It is essentially like that of the fourth degree except that the surface is more deeply charred. The scar is deeper, firmer and immobile. There is greater disfigurement; and since the muscles are involved, a considerable functional impairment may result. The scar has a decided tendency to break down and ulcerate.

The sixth degree burn exhibits a carbonization of tissue to the extent that the tissues are changed to animal charcoal. This type of burn is most commonly seen on the fingers and toes.

So far the histopathology of the burned tissue itself has been considered. It may also be well to consider the tissue changes remote from the burn.

Stallard and Borman (59) have given a very good description of the condition of the various organs of the body as found at autopsy.

The heart muscle was of a pale pink color and microscopically showed an atrophy of the muscle fibers. There was little difference in these findings in the individual cases in which the autopsies

were performed, and the age of the victim seemed to make no difference as to the degree of the heart changes. However they found that in cases where death occurred immediately following the burn the changes were not so marked.

There was also extensive hemorrhage present in the gastrointestinal tract in several cases. This was attributed to the presence of a perforated duodenal ulcer which was found in each of these cases. The presence of a duodenal ulcer complicating burns was first described by Curling in 1842. Maes (40) and McLaughlin (39) have given a very good description of this lesion. It is not unlike an ordinary duodenal ulcer except that it is frequently associated with burns. This will again be discussed when considering the complications of burns.

Stallard and Borman (59) report that they found thrombi in the vascular and lymphatic channels of the ligamentum teres and attributed the ulceration of the duodenum to this. They believed the thrombi to be due to the lodging of small blood clots from the burned area into the blood stream in the minute vessels of the ligament.

There were also extensive firm adhesions between the gall bladder, liver, duodenum and transverse colon. Presence of the adhesions due to cholecystitis was ruled out by the fact that several

of the cases that exhibited the most extensive adhesions were children under the age of five, in which previous gall bladder disease would have been very improbable.

Microscopically the spleen and liver showed a condition of cloudy swelling, the degree of which varied according to the age of the individual. In one of the cases of late death following a burn in a five year old child, the changes in the spleen were hardly noted.

The kidney changes were also very marked. Grossly, red streaks could be seen in the surface of the capsule showing extensive hemorrhage. Histologically there was a diffuse degeneration of the kidney tubular epithelium. The glomeruli were markedly atrophied and contained only a small amount of blood in the tufts.

Weiskotten (73) gives a very good description of the adrenal glands following burns found at autopsy. The changes were constant and rather characteristic. They were markedly swollen and deep red. There was marked edema of the surrounding fat tissue. On section there was evidence of extensive hemorrhage. Microscopically the cells of the adrenal had undergone degeneration, and in several cases this was so extensive the tissue could hardly be recognized. In fact it was almost like a necrosis seen in chlorform poisoning, except that in the case

of a burn the condition is more generalized.

The brain tissue was noted to have undergone slight degeneration. This, however, was not to a very great degree. The blood vessels of the brain were markedly congested in almost every case.

There was also a hyperemia of the lung tissue in almost every case studied by Stollard and Borman (59). However they thought this was due to a terminating brochopneumonia.

PATHOLOGIC PHYSIOLOGY

Many theories have been advanced attempting to answer the question as to the cause of the generalized symptoms and death in extensive burns. None of these seem to answer the question sufficiently well to avoid criticism.

Davidson (21, 22) has given a good description of the clinical course of burns and has pointed out the characteristic generalized symptoms. Promptly after a person has been burned severely, an acute period of depression and exhaustion follows, which is usually designated by the indefinite term "shock". The picture is characterized by blunted sensibility; cold, moist, pallid skin; subnormal temperature; irregular, sighing respiration; rapid, feeble pulse; and finally, a very low blood pressure.

If the patient survives this desperate condition, another syndrome develops, which today is described by the equally vague term "toxemia of burns". In contrast with "shock", it occurs later. The temperature instead of being subnormal is elevated. The blunting of the central nervous system is replaced by increased irritability. The patient becomes restless and responds to all external stimuli. Delirium rapidly follows, and convulsive seizures of varying intensity occur. The unburned skin is

hot and reveals a slight flush. The pulse is generally rapid but parallels the temperature curve closely. It is full and of bounding character unless death is impending. The blood pressure is relatively low but well above the critical "shock" level.

In extensive burns these two distinct clinical pictures are frequently confused. Death may be attributed to toxemia when in reality the organism is rather overwhelmed by massive trauma.

Just how are the above-mentioned generalized symptoms accounted for? Many theories have been brought forward. Three of these stand out and today are given most attention. These theories are (22):

1. loss of skin function,
2. changes in the blood stream,
3. toxemia.

1. Loss of skin function.

The theory stating that the general symptoms are due to loss of skin function is perhaps the oldest. It was brought out by the German writers during the eighteenth century. Davidson (22) after having reviewed the literature gives a good discussion of this theory.

There are various phases of the theory. These are:

1. disturbance of respiration,
2. disturbance of secretion,

3. disturbance of temperature regulation,
4. disturbance of sensation.

These theories have almost all been disproven. Kizanitzin, a German, disproved the idea that the disturbance of respiration and excretion of the skin could cause the generalized symptoms. He gilded a rabbit, thereby destroying any possibility of the skin carrying on the functions of respiration or excretion, and the animal suffered no ill effects.

To disprove the last two points, the French investigator, Welti, found that by keeping burned animals in various temperatures, he could find no optimal temperature. The symptoms developed regardless of the temperature. He found that severing the sensory nerves to the skin and then burning it made no difference to the appearance of the generalized conditions.

Even though, these theories have been disproved, it is still admitted that the skin function of a burned area is destroyed or at least diminished; and although it cannot be the primary cause of these general symptoms and death, it certainly cannot aid the organism in combating the symptoms.

2. Changes in the blood stream.

Baraduc (22), the Frenchman, was the first to bring out the point, in 1860, that there is a tremendous concentration of blood after a burn.

Heister (31) mentioned the fact in 1754 but thought it had little to do with the general conditions.

Recently investigations have been carried out on the blood concentration and changes in cases of burns by Underhill and his co-workers (63, 64, 65). They used rabbits in their experiments.

By using hot plates in burning the animals, they could regulate the extent and also the degree of the burn. Therefore their findings are much more accurate than those obtained from patients. They found that the edema occurs rapidly, reaching its peak in twenty-four hours, and then gradually subsides and is gone at the end of three or four days.

During this time they were making studies of the blood, especially the hemoglobin. Using the arbitrary point 100 as the normal hemoglobin for a rabbit, they found that about four hours after a burn covering about one-sixth the body surface the hemoglobin had risen to about 150. At the end of seventeen hours the hemoglobin had dropped again to normal; and at the end of twenty-four hours it was ninety per cent. Underhill explains the change in this way: The fluid is drawn out of the blood stream to the burned area and after four or five hours the animal compensates by drawing reserve water from the tissues into the blood stream; the hemoglobin concentration then falls until the animal has "over-

compensated". After twenty-four hours, however, the hemoglobin returned to normal and remained that way.

Underhill believes, therefore, that the prognosis of a burn case depends upon the body's ability to compensate for the increased blood concentration. The higher the concentration and the longer it remains that way, the poorer the prognosis. He also pointed out that this is true in humans, judging from a series of burn cases he has studied with Dr. Carrington (64).

Robertson and Boyd (51) have carried out similar procedures and report almost identical results. No one, up to the present time, disputes the fact that there is a tremendous concentration of blood.

Underhill and his co-workers then attempted to measure the actual amount of fluid lost. They did this by killing the animals and then evaporating the tissues to normal consistency. It was found that the fluid reached its maximum about twelve hours after the burn and that at this time the edema fluid represented about twenty-five or thirty per cent of the total blood volume. The greatest loss of fluid noted was seventy per cent. However, these figures were probably not accurate. Calculations were made by measuring the blood of a group of normal rabbits and calculating its ratio to that of the entire body in terms of per cent. This percentage figure then used in the calculation of the blood volume of the

animals burned.

Even though the figure may not be accurate, it is interesting to transpose it to a man's condition. It will be found that the blood volume of a one hundred sixty pound man is about five thousand cubic centimeters. Seventy per cent of this would be thirty-five hundred cubic centimeters; and considering this percentage the fluid loss in a burn involving one-sixth of the body, in a burn involving one-third of the body (or twice the extent of the burn studied in the rabbits) a man would lose one hundred forty per cent of his total blood volume or seven thousand cubic centimeters. Although this is an impossibility, it is certainly indicative of the significance of the fluid loss from the blood stream.

Robertson and Boyd (51) reported similar results from a series of experiments on rabbits and dogs. They studied the exact composition of the blood following a burn and reported that the blood urea nitrogen, rather than free nitrogen, content of the blood was increased twenty to thirty per cent, four to six hours after the burn. This they attributed to a true concentration of the blood.

Blalock (12) did similar experiments and also reported a tremendous amount of fluid loss from the blood stream.

Another experiment by Underhill and Fisk (68)

showed that the fluid produced by the burn had the same composition as the blood serum and therefore could be regarded as such. They found that there was less globulin in the edema fluid than in normal serum; but after checking the globulin in the blood serum, they found that it also was reduced.

Davidson (21) brought out the fact that the sodium chloride content of the blood was reduced. He made his observations on thirty-one cases and found that both the whole blood and the plasma chlorides were greatly reduced. The fall in the blood chlorides was not explained by alteration of the renal threshold, diet, fever, exudation, blood concentration, nor vomiting. There was suggestive evidence that retention of sodium chloride took place. This finding has indeed aided in the treatment of the patient's general condition.

Underhill (69) experimented on the relation of the blood chlorides to the chlorides of the edema fluid. He found that as long as the blood concentration remained normal, the blood chlorides also remained normal. The chloride content of the edema fluid was about the same as that of the blood. Carrying his experimentation still farther he found that the rabbits, with which he and his co-workers were working, would not show diminution of blood chlorides until twenty-five to thirty per cent of the blood volume was lost. He thereby came to the

conclusion that the animal has remarkable powers to compensate for loss of chlorides.

Underhill (70) then decided to learn the composition of the tissues of the body remote from the burned area. He found that these showed little, if any, change in composition and that the chloride loss was mainly from the blood stream and not from the tissues.

Davidson (22) points out the fact that there are morphological changes of the red blood cells which may cause a partial loss of function and thus cause a failure of the blood stream to function properly. However, most writers do not believe that this change is sufficient to play a part in producing the generalized symptoms in burns.

Weiskotten (73) and Stollard and Borman (59) point out that generalized thrombosis frequently occurs. However, when there is such a tremendous concentration of the blood stream, one can plainly see why thromboses are almost bound to occur.

3. Toxemia Theory.

William Cumin is mentioned by Davidson (22) as being the first to report an autopsy of a burn case. This was in 1823. He reported that in cases of early death he found merely a hyperemia, but in cases of late death he observed a well marked generalized inflammatory reaction.

Weiskotten (73) has emphasized the very striking

condition of the adrenal gland found at autopsy. Stollard and Borman (59) have reported changes in the kidney, spleen and liver suggesting evidence of toxemia. Bardeen is cited by Davidson (22) as stating that the alterations of lymphatics observed were nearly identical with those seen in diphtheria, in which there is a definite toxin present. Bardeen published his findings in 1890 and was the first to suggest the possibility of toxemia.

It has already been mentioned that there is a hyperemia of the abdominal and visceral organs. Davidson (22) states that this is similar to the hyperemia found in peritonitis, in which a toxin causes the condition. According to Underhill (63), however, this condition is due to a concentration of the blood stream.

The first experiments carried out to prove the presence of a toxin were done by Reiss (22). He attempted to recover the toxin in the urine. He found a substance similar to pyridine in the urine of a group of eight burn cases. His findings have been confirmed by Heuer and Andrus (32). They also found that by injecting an extract made from the tissue of the intestines, they could recover a similar substance from the urine. This substance not only produced the pyridine-like substance in the urine, but also produced a "shock" similar to that seen in burn cases. An analysis of the substance they injected

showed it to be similar to histamine. They came to the conclusion that since the adrenals were affected following a burn, the animal was much more susceptible to intoxication. This was, therefore, the beginning of a vicious cycle---the toxin causing the adrenal damage, and the greater the adrenal damage, the greater the effect of the toxin on the organism.

Vogt, states Davidson (22), injected blood from burned animals into healthy animals and found that toxic symptoms developed. Robertson and Boyd (51) carried out similar experiments with similar results. Kapsinow, however, states (36) that he believes the shock was not due to presence of a toxin but was due to improper matching of blood in the animals used for the experiment.

Robertson and Boyd (51) have done more experiments regarding the toxemia of burns than have any other investigators. Most of their experiments were performed on young anesthetized rabbits, as were those of Underhill and his associates.

They found that typical symptoms followed the burn in every case. Immediately following the operation there was a period of shock evidenced by a circulatory collapse and a fall in temperature. This lasted from six to eight hours. During the next fifteen hours the animal did not appear to be very ill. After that toxic symptoms began to ap-

pear. The temperature rose to about 105°. Food was refused. The animals were either drowsy or excited or exhibited periods of each. In a few cases convulsions were noted. The non-protein nitrogen of the blood was increased from forty to fifty per cent over the normal amount.

The toxic symptoms persisted for two to three weeks in the nonfatal cases. Eleven of the twenty-five recovered.

They then injected boiling water into the muscles of anesthetized rabbits. They found that although there was a definite fluid shift with a concentration of the blood, there were no signs of toxemia. They therefore concluded that there must have been a toxin formed by burning the skin which was absorbed by the blood stream. This, and not the fluid shift, produced the symptoms.

Their conclusions have been criticized. Underhill (63,64) and Kapsinow (36) believe that the toxic symptoms were not due to a toxin formed by the burn, but were due to a secondary infection. Aldrich (1) believes the same.

Robertson and Boyd (51) decided to do a transplantation of burned skin from one animal to another and see what reaction this would cause. This was done in a series of twelve rabbits. The results were very interesting. If the skin was removed less than eight hours after the burn, the animal from

which the skin was taken would have no signs of toxemia. If the transplant "took", as it did in all except one case, the animal receiving the graft would develop the toxic symptoms in less than one hour after the transplant.

If, however, the transplant was not made until after eight hours following the burn, both animals would develop the signs of toxemia. They also found that if they waited about fifteen hours before doing the transplant, the animal receiving the graft would develop no signs of toxemia.

This, they say, should be definite proof that a toxin is formed. Kapsinow (36) again criticized their interpretations, stating that the signs of toxemia were either due to secondary infection or to a definite sensitiveness of the organism to the skin of the burned animal.

Robertson and Boyd (51) prepared certain extracts of the burned skin and found that on injecting them into normal animals the toxic symptoms would appear. Kapsinow (36) repeated this experiment and got a similar reaction. Still attempting to disprove the toxic theory, he also made extracts of normal skin, which he injected into the normal animals and found the same toxic symptoms were produced.

Morris (43) in 1882 showed that debridement of the burned area often prevented the toxic signs and therefore concluded that the generalized symptoms

were due to a toxin formed in the burned area. Robertson and Boyd (51) also showed this to be true in their work cited above. Bancroft and Rogers (4), Ravdin and Ferguson (50), Goldblatt (28) Duval (5) and Douglas (24) have all come to the same conclusion merely by using the debridement method as a treatment.

Davidson (22) concludes that since coagulation of the burned tissues with some coagulant, tannic acid being the one he used, the toxic symptoms would not appear or at least would be lessened; and that there certainly was some toxin formed which, if not removed or coagulated in some way, would enter the blood stream and produce this condition. Every writer who is an advocate of tannic acid has reached the same conclusion. Underhill and his associates (63, 64, 72) and Aldrich (1,2) still maintain, however, that the tannic acid merely prevents the infection and in this way prevents the toxemia.

Some work has also been done in studying the rate of absorption from the edema fluid produced by burns. Underhill and his associates (66) and Kapsinow (35) injected phenosulphonthalein into the edema mass. They found that it was absorbed very slowly as compared to the absorption of the dye from the unburned skin. They based their calculation of the rate of absorption on the rate of excretion of the dye by the kidney. They found that, when injecting the

dye into the burned area, only ten per cent was excreted in two hours. If the dye, however, was injected into the unburned skin, fifty per cent or more was excreted in the same period of time.

However, in studying the rate of absorption during various intervals following the burn, they found that the dye was absorbed more rapidly after eight hours, although the absorption was still not as rapid as that from the normal skin. Douglas (24) has also experimented along these lines, but his experiments were not extensive enough to be of great value. His results, however, were similar to those of Underhill. The fact that absorption does occur would also support the toxin theory.

Underhill, Kapsinow and Fisk (65) also made studies of the temperature of the body immediately following a burn. This was a repetition of the experiment of Robertson and Boyd (51), who found that the temperature was elevated immediately after the burn. Underhill and his co-workers do not agree with these findings. They state that the general temperature is elevated very little. They also say that the hyperemia found in the abdominal organs immediately after the burn is either due to direct radiation of heat from the burn or due to blood concentration, and not to toxemia.

Thus, it can plainly be seen that no one theory can explain the cause of the generalized symptoms of

burns and in order to make definite conclusions
more experimental work must be done.

TREATMENT OF BURNS

I. Historical Facts

The treatment of burns has been a problem for centuries. Little was understood concerning the treatment until the nineteenth century, and today there is still considerable argument as to the exact procedure to use in the treatment.

Until the beginning of the nineteenth century most physicians were still using the old treatment devised by Hippocrates. Coxe (19) has translated the writings of Hippocrates and Galan. It is surprising to learn that the treatments advocated by them are still the household remedies used by many people today. Galan approved of the use of lard and aromatic oils. Hippocrates did not. He employed the use of arsenic, black hellebore, which is an extremely poisonous root of an herb, and cantharides which is a powder prepared from the dried blister beetle. He also pointed out the advantages of purging and bandaging in certain instances. Aerugo, who lived at about the same time Hippocrates did, advocated using a mixture of sweet wine, honey, resin, myrrh and niter. Hippocrates considered this an excellent ointment for cleansing and removing dead tissue from the burn. Lead, alum, copper and arsenic were also used by Hippocrates. These were used in various ways especially in the form of ointments. This is the first record of the treatment

of burns. Even Julius Caesar, who tells of warfare with boiling oil and hot water, does not leave a record as to how the victims were treated. It is supposed that they were left to die without being treated.

The beginning of the nineteenth century therefore marks the beginning of a new era in the treatment of burns, which, even today, a century and a quarter later is not yet completely understood.

Little was thought about the general treatment in the case of a burn. Cumin, a German, is cited by Barnes (5) as being the first to call attention to the fact that there was a loss of fluid from the blood stream in the case of a burn. This was in 1823. No one paid much attention to this until a half century later, when Baradue became interested and made rather definite and valuable observations. He noted that the viscosity of the blood became remarkably increased immediately following a burn. He therefore advised increasing the fluid intake to overcome this condition. Parker, is mentioned by Barnes (5) as being the first to advocate and urge the treatment of shock. This was in 1844. He however had no idea as to the cause of the shock and merely treated the condition as though it were due to some other cause. He put the patient to bed and maintained normal body temperature, which, of course is quite important.

Passavant and Hebra, in about 1860, are cited by Rose (53) as being the first to advocate continuous baths in the treatment of burns. Rose (53), who published his work in 1906, also advocated the procedure.

Perhaps the most important discovery to affect the treatment of burns was Lister's discovery of the value of antiseptics. After hearing Lister give a paper recommending the use of phenol as an antiseptic, Pirrie in 1867 conceived the idea that perhaps it would be of value in treating burns. Morris, (43) was the first to introduce the use of antiseptics in the treatment of burns in this country.

Copeland (18) was the first to advocate the use of dry air to the burned area in treating burns. This was in 1887. He also advocated cleansing the wound with soap and water, and introduced the use of bismuth subnitrate, sprinkling it on the burned area.

Carron oil is mentioned by Bettman (10) as first being introduced in Scotland during the nineteenth century. Although not a believer in its use, he states that it still is one of the commoner forms of treatment.

Sandfort (5), the Frenchman, advocated the use of ambrine, paraffin being its principal constituent, in 1914 while dealing with burn cases during the world war. It is still advocated by various writers today.

Davidson (22) introduced a form of treatment in 1925 which is accepted by almost every one today. This is the use of tannic acid applied to the burned skin, the purpose being to coagulate the devitalized tissues and therefore prohibit absorption of them, assuming that these are toxic when absorbed by the blood stream. The idea of treating burns by coagulating the toxins is not a new one. Stammel (60) relates that India ink has been the household remedy in the treatment of burns in the Phillipines for centuries. Its chief constituent is gallo-tannate of iron, which coagulates the devitalized tissues the same as does tannic acid. Tea has been used for burns in China for centuries. The origination of the treatment is unknown. Strong tea contains about five per cent tannic acid. This method of treatment is therefore the same as that introduced by Davidson. Other coagulants are being used. Picric acid, probably the most common, was introduced by Sneve (58) in 1906. Ferric chloride, gentian violet, silver nitrate and others are also being used at present.

II. Modern Treatment of Burns

A. The general condition--

As was pointed out in the discussion of pathological physiology, there are various factors to consider in the case of an extensive burn, such as

shock, loss of fluids, loss of chlorides and others.

In the treatment of a burn, one must consider the following general problems:

1. Stopping the pain.
2. Preserving life.
3. Restoring function.
4. Shortening the period of convalescence.

Pain is always alleviated by morphine. Davidson (22) advocates the immediate administration of one-fourth grain. Ravdin and Ferguson (50) recommend painting the burned area with a procain solution immediately following the burn.

The treatment of the general changes caused by the burn consist of combating the following:

1. Shock.
2. Blood concentration.
3. Loss of blood chlorides.
4. Intoxication.

In treating shock various conditions must be considered. Davidson (22) has given a very good description of shock caused by a burn. There is a blunted sensibility; cold, moist, pallid skin; sub-normal temperature; irregular, sighing respiration; rapid, feeble pulse; and a low blood pressure. This description obviously represents a critical condition. The patient must be put to bed and kept warm. A constant temperature of about 85° is most desirable. This may be accomplished by using hot water bottles,

an electric heater, or better still an electric light cradle. Goldblatt (28) states that when using the electric cradle one must be careful not to get the bulbs closer than eighteen inches from the patient.

Underhill (65) has pointed out the fact that there is a tremendous fluid loss following a burn. It therefore is necessary to replace the fluids. Fantus (27) states that in cases treated in the Cook County hospital, if the burn involves more than one-third of the body surface, the patient is given from eight to ten liters of fluid in the twenty-four hours immediately following the burn. This is given by three different routes, orally, hyperdermically and rectally. Normal saline is usually used. Five per cent glucose solution is used in some cases where there is evidence of acidosis.

Blood transfusions are of value in severe cases. Most writers advocate this procedure in every case where the prognosis is grave. Some authorities are firm believers in exsanguination transfusion. Ravdin and Ferguson (50) and Robertson (52) are firm advocates of this procedure especially in children.

Davison (21) also pointed out the importance of giving sodium chloride in the general treatment of burns. Copeland (18) in 1867 and Rose (53) in 1906 also advised the administration of sodium chloride in burn cases but gave no reason for doing so. Duval (25) states that he routinely gives fifteen grains of

sodium chloride, mainly by mouth, during the first twenty-four hours following the burn. Not only does this increase the lowered chloride content of the blood, but it also increases the patients desire to partake of more fluids.

Intoxication is overcome by the forcing of fluids and by the coagulation of the supposedly formed toxins on the surface of the burned area. This procedure will be taken up in greater detail later.

Blair (11) and his associates and Davidson (22) point out the fact that it is very important to keep up the morale of the patient. They should not be allowed to become discouraged and fail to partake of a sufficient amount of food.

Proper position of the patient often is important in the restoration of function. Blair (11) says that this is probably the most important and also the most neglected point in the treatment. If possible the patient should be allowed to move the part freely to avoid secondary contracture. Where this is impossible and the burn involves a joint area, the extremity should always be placed in extension.

The convalescent period may often be shortened by good early care of the case. Skin grafts, where indicated, should be done as early as possible following the burn. Good nursing care is also quite essential.

B. Local treatment of burns--

Numerous treatments have been prescribed, and

extravagant claims have been made concerning the special merit of various remedies. Many of these claims are based upon results obtained in treating burns of minor degree which naturally exhibit the ability to heal spontaneously, irrespective of treatment. It would seem obvious that such observations can be of little value in combating the mortality and the distressing morbidity of major burns.

Trusler (62) states that it should be apparent that there is no single ideal treatment for burns and no one procedure applicable to all phases of the problem. It must be admitted that even with the best of treatment there is unavoidable mortality and unpreventible morbidity attendant upon extensive burns. In general it may be stated that morbidity depends chiefly upon the extent of the burned surface; the deformity or disfigurement depends upon the location and depth of tissue destruction. Admitting these facts, it is true, nevertheless, that much depends upon treatment. Lives may be saved and countless distressing complications prevented or relieved by effective therapeutic measures.

The various accepted treatments will be discussed briefly.

Tannic Acid

Davidson (22) was the first to introduce tannic acid in the treatment of burns in 1925. While working

with phosphotungstic acid, endeavoring to work out a method of coagulating the supposedly formed toxins, he was advised by E. C. Mason, a chemist, to try tannic acid, since it had the same effect as phosphotungstic acid except that it was less toxic. At least this was the first time tannic had been used with a definite purpose in view. A historical study of treatment (60) shows us that it had been used for centuries in China and the Phillipines.

The solution described by Davidson contained two and one-half per cent tannic acid. Almost every other advocate of tannic acid believes that the concentration should be about five per cent. Barnes (5) even recommends using a ten per cent solution. Wells (74) suggests merely adding enough tannic acid to the water to give it a deep, muddy color. This would be about a five or ten per cent solution. Seegar (55) mentions the importance of having the solution at a proper pH. Since autolysis does not take place in a slightly alkaline medium, he suggests adding enough sodium hydroxide to give the solution a pH the same as that of the blood. Fantus (27) suggests the addition of calcium carbonate to arrive at the proper pH.

Methods of application are numerous. Davidson suggested placing sterile gauze over the area and soaking this with the tannic acid solution every half hour until the coagulum is well formed. Mont-

gomery (42) points out the importance of sepsis. Before applying the tannic acid, he believes it beneficial to remove as many gross particles as possible with sterile instruments and wash the burned area with benzene or ether. Montgomery also states that the application of the gauze before applying the tannic acid adds to the difficulty in removing the coagulum. Penberthy (47) suggests washing the area with soap and water before applying the tannic acid. He also states that by spraying the tannic acid solution on the area instead of swabbing the area, reduces the possibility of introducing infection. Wells (74) introduced the method of placing the patient in a bathtub containing the solution. While the patient is in the tub gross tags of skin and the tops of blisters as well as other particles of debris can be removed. The unburned areas are scrubbed with soap and water. As soon as the solution becomes grossly contaminated, the tub is drained and a fresh solution is supplied. As soon as the patient is thoroughly scrubbed the coagulum is usually well formed. The patient is then placed in bed under an electric cradle and sprayed with the solution every hour. At the end of forty-eight hours sufficient coagulum is present so the spraying can be discontinued.

Combined treatment is advocated by various writers. Bettman (9) advocates the use of silver

nitrate following the tannic acid. The tannic acid is applied every half hour. Following this a ten per cent solution of silver nitrate is applied. The advantages of this are: an increase in the rapidity in tanning; an immediate stopping of loss of fluids; a lessening of the possibility of infection due to the added antiseptic; a greater analgesia; and the formation of a thin flexible coagulum instead of the thick coagulum formed by just the use of tannic acid.

Trusler (62) advocates the use of ultra-violet light following the application of the tannic acid. After the coagulum is formed the patient is exposed to ultra-violet light for two minutes the first day and for five minutes each day thereafter until the coagulum is removed. Following the removal, the patient is again exposed to the light for four or five minutes a day until healing takes place or until the area can be covered by skin grafting. If ultra-violet light is not available the patient can be placed in the sunlight. The advantage of the ultra-violet light is that it not only sterilizes the area but also stimulates epitheliazation.

Barnes (5) has used paraffin gauze over which he places boric acid packs on the burned area following the removal of the coagulum. This combats infection which may be, and according to him usually is, present under the tannate covering.

Removal of the coagulum should take place as soon

as it curls up around the edges, according to Davidson (22). This is usually at the end of about thirty-six hours and after any toxic symptoms have subsided. Various writers advocate leaving the coagulum in place for as long as four to six days. Nearly every one agrees, however, that it should be removed at the end of twenty-four to forty-eight hours unless infection should cause a separation of the coagulum, in which case it should be removed sooner, or at least the area under which the infection is located should be removed, an antiseptic applied and the area again subjected to the tannic acid solution. Infection under the coagulum is made evident by the "puffing up" of the coagulum.

Statistics show that the mortality rate has dropped since the use of tannic acid has been introduced. Bettman (9) reports that since using tannic acid for burns he has lost only one of twenty-two cases in which the area involved was over one-sixth of the body surface. McClure (37) has received statistics from various hospitals and finds a drop in mortality ~~of~~ from thirty-two to twelve per cent. Beekman (8) reports a drop in mortality from twenty-eight to fifteen percent and Mason (41) reports a drop from twenty-nine to thirteen per cent.

The advantages, therefore, of tannic acid are:

1. Decrease in mortality,
2. Protection to the patient,

3. Prevention of additional loss of fluid,
4. Inhibition of toxemia,
5. Its mild antiseptic power,
6. Its analgesic property,
7. That it minimizes scarring,
8. That it is inexpensive,
9. That it is non-toxic,
10. That it does not destroy the remaining live tissue.

The disadvantages of tannic acid are far in the minority to the advantages. Goldblatt (28) believes that tannic acid is not sufficiently antiseptic to inhibit infection to any degree, especially anaerobic infections which predispose tetanus. He also believes that plain tannic acid, since it is an acid, would promote rather than inhibit autolysis. Aldrich (1) believes that tannic acid is toxic and will also destroy the living epithelium of sweat glands and hair follicles remaining on the burned surface. He also points out the fact that the coagulum is thick and brittle, so that underlying infection will not present itself until it is so extensive that a great amount of good tissue below the eschar is destroyed.

Gentian Violet

Gentian violet or triphenyl-methylamine, a coal tar derivative, was first introduced for use in the treatment of burns by Aldrich (1) in 1933. In addi-

tion to forming an eschar similar to that of tannic acid, it also has greater antiseptic value.

The solution made up by Aldrich originally was of a one per cent concentration in water. Connell (17) and his associates came to the conclusion that gentian violet jelly was of more value and more practical. This was prepared by adding thirty grams of tragacanth to one thousand cubic centimeters of one per cent aqueous solution of gentian violet.

The technique of applying the aqueous solution introduced by Aldrich (1) was similar to that of applying the aqueous solution of tannic acid. Connell (17) advocated applying the jelly on gauze and then placing the gauze on the burned area. Fantus (27) working at the Cook County hospital also used the jelly in this way with good results. The wound is not cleaned, the gentian violet merely applied over the entire area including the debris. The blebs are opened and the jelly is applied to the raw surface.

The coagulum should be removed after about two weeks and if the burn is deep skin grafting should follow.

Statistics show that this treatment is very satisfactory. Connell (17) and his co-workers have used it in a large number of cases and never yet have seen a rise in temperature during the so-called "toxic period" described by Davidson (22).

Advantages of gentian violet are, in addition to

those described for tannic acid; ~~XXXX~~

1. It is not toxic,
2. It has very good antiseptic powers,
3. The coagulum is thin and pliable.

The chief disadvantage of gentian violet is the fact that it stains everything coming in contact with it.

Picric Acid

Picric acid is today a very popular household treatment for burns. It is usually used in the form of a picrate. It was first used in 1905 by Sneve (58), who reported rather unfavorable results.

Sneve used picric acid in a one per cent aqueous solution. Montgomery (42), although not an advocate of picric acid, states that best results can be obtained using one per cent picric acid in five per cent alcohol. Fantus (27) advises using it in ten per cent alcohol.

It is applied to the burned area by soaking gauze in the solution and placing the gauze on the burn.

Picric acid has not been used to any extent by the medical profession except in cases of minor character. Its action is supposedly similar to that of tannic acid. Its chief disadvantages, however, are its terrific staining qualities, staining everything it comes in contact with, and its extreme toxicity when used in rather extensive burns.

Ferric Chloride

Tincture of ferric chloride was first used as a form of treatment for burns in the latter part of the nineteenth century. It soon fell into disuse, however. Coan (15) however became aroused in 1935 when a man, who later became his patient, fell into a vat of boiling ferric chloride solution. By the time the victim reached the hospital a coagulum had formed. He was given no other external treatment. The coagulum was flexible and the patient was rather comfortable. He made an uneventful recovery even though the burn involved both legs, extending upward beyond the middle of the thighs.

Silver Nitrate

Silver nitrate used in treating burns was introduced by Shillito (57) in 1929. It has also been used in conjunction with tannic acid as described before.

The technique used by Shillito consists of spraying the burned area with a one to five per cent solution of silver nitrate, then exposing the area to ultra-violet rays for five minutes or to sunlight for about thirty minutes. No dressings are used. The silver nitrate forms a coagulum when exposed to ultra-violet light. The chief advantage of this treatment is the remarkable antiseptic value of silver nitrate. Shillito states that he has never seen a burned area

become infected following this form of treatment.

Other forms of fixation methods are described by Montgomery (42). Absolute alcohol causes rapid fixation of devitalized tissues. It demands a great deal of attention because of its rapid evaporation. It produces a minimum of scarring and is especially valuable around the face. Aluminum acetate has also been used. Ten parts of a two per cent alcohol solution of aluminum acetate in addition to one part of methylene blue solution is a very good fixing agent. It is usually applied to the burned area on a light guaze dressing and allowed to dry. Not having been used to any extent, its value is undetermined.

Debridement

Debridement was first introduced in 1882 by Morris (43). The theory on which the treatment is based is, of course, one of toxin formation. Bancroft and Rogers (4) were firm advocates of this form of treatment but now have abandoned it in favor of tannic acid. They reported remarkable results from this type of treatment. Willis (75) found that in cases where debridement was done, the blood changes were less marked.

The procedure merely consists of removing all of the dead and damaged tissue.

The advantages of the treatment are:

1. The source of the "toxin" is removed,

2. The pain is reduced,
3. There is fresh, healthy granulation bed left after removing the dead tissue.

The disadvantages are:

1. The good tissue remaining in the burned area is removed,
2. After treatment is painful and infection often intervenes,
3. Skin grafting must always be resorted to after debridement.

Paraffin-Ambrine

Paraffin used in treating burns was first introduced by Sandfort, a French army doctor, in 1914. Montgomery (42) gives a good description of its use. The original compound was called ambrine and consisted of one part of resorcin, two parts oil of eucalyptus, five parts of olive oil, twenty-five parts of soft wax and sixty-seven parts of hard wax. Collier (16) advocates the use of ambrine until the patient is through the shock stage. The wax is then removed and the area treated with Daykin's solution for twenty-four hours. Skin is then grafted into the area. Either Thiersch grafts or skin flaps are used. If the Thiersch graft is used the graft is covered with the paraffin. If infection does not cause the wax to separate, it is left in place for five days. Collier has had great success in getting "takes" by

using the paraffin compound in this way.

Sodium Bicarbonate

The basis for using sodium bicarbonate for burns is that autolysis does not take place in an alkaline medium and since the "toxin" formed in the burned area is a product of autolysis, its formation would be inhibited. Montgomery (42) mentioned the use of a ten per cent solution of sodium bicarbonate placed on sterile gauze and then applied to the burned area. Goldblatt (28), an advocate of the treatment, uses ~~it~~ either the same strength solution or a paste made of the bicarbonate and water. After ~~applying~~ applying the dressings, they are soaked every half hour with the ten percent solution. They are then removed and the patient is exposed to ultra-violet or sunlight during the day, the dressings being replaced at night. The chief advantages of this type of treatment is that the sodium bicarbonate is always available in the home and therefore is a good home remedy.

Sodium Chloride

Sneve (58) in 1905 pointed out the importance of sodium chloride in a burn case. Not only did he give it internally but also externally,, giving the patient baths in a normal saline solution. Blair (11) and his co-workers advocate the use of a water soluble jelly containing from two to five per cent sodium chloride.

The purpose of the sodium chloride is two-fold. It alleviates the pain and also inhibits the growth of bacteria to some extent.

Various other treatments have been devised. Fantus (27) and Bettman (10) mention the use of scarlet R. This is used in the form of an ointment and is said to stimulate epitheliazation. Carron oil has been a very common treatment of burns. It consists of a mixture of linseed oil and lime water. Bettman (9) states that it should be mentioned only to be condemned because it merely adds to the debris on the area and makes various other treatments almost impossible. Sutton (61) advocates the use of thioglycerol. One part thioglycerol to five thousand parts of glycerine is used. This is applied directly to the burned area and covered with cellophane or rubber. The dressing should be changed once every twenty-four hours. It is supposed to stimulate epitheliazation. It has not been used extensively enough to give an estimate of results obtained.

COMPLICATIONS

Penberthy (47) states that the more common complications of burns are infection, contraction, malnutrition and peptic ulcer.

Infection can be prevented by proper early treatment. Following the burn the involved area is sterile unless it is later contaminated. If, however, infection does occur antiseptics must be used. Fantus (27) states that a solution of chlorinated soda should be applied. The sloughs should be removed with forceps and scissors. Boric acid packs may also be used. Ravdin and Ferguson (50) advocate the use of dichloramine-T for infection. Others advocate the use of merthiolate, Daykin's solution, etc. Tetanus antitoxin should be given to every patient with extensive burns.

Contraction can be prevented by placing the patient in the proper position. If the burn involves a joint, early motility of the extremity is important. If the joint must be stabilized, it should be placed in an extended position. Bettman (9) states that, where skin grafting is necessary, it should be done early to avoid contraction.

Malnutrition of course can be prevented by giving the patient proper attention. Nourishing food should be given and the patient encouraged so that he does not

become disgusted and lose interest in his condition.

Duodenal ulcer, according to Penberthy (47), often remains undiagnosed. The ulcer complicating burns was first described by Curling in 1823. McLaughlin (39) and Maes (40) give excellent descriptions of the condition. It is believed to be due to congestion or even thrombosis in the minute arteries supplying the duodenum. It is usually characterized by pain and tenderness over the duodenum and uneasy digestion. The symptoms may be variable and in some cases may not be present at all. It is most commonly seen in children and reported to be found in from two to fifty per cent of burn cases. It should be treated the same as any other peptic ulcer.

CONCLUSIONS

1. Burns represent a large portion of the general practitioner's and general surgeon's cases.
2. An extensive burn is a major surgical problem and if involving more than one-third of the body surface usually terminates in death.
3. The exact cause of death in a burn case is unknown.
4. The physiology of a burn is not very well understood. The condition present is probably due to shock, blood concentration and toxemia.
5. Early adequate treatment of a burn often prevents complications.
6. No one method of treatment is sufficient in treating a burn.
7. The general treatment of a patient is just as important as the local treatment.
8. The coagulation treatment of burns is very satisfactory, judging from the drop of about fifty per cent in the mortality rate since the introduction of the tannic acid treatment by Davidson in 1925.

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