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Cutaneous burns: with especial reference to their physiology and treatment

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CUTANEOUS BURNS; WITH ESPECIAL REFERENCE
TO THEIR PHYSIOLOGY AND TREATMENT

By

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Presented to the Faculty
of the University of Nebraska in
partial fulfillment of the requirements
for the Degree of Doctor of Medicine

UNIVERSITY OF NEBRASKA
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INTRODUCTION

"Grecian Mythology teaches that of all the animals, man, was the last race created and in consequence thereof, was most poorly endowed with physical gifts. So Prometheus stole fire from the hearth stone of the Gods on Mount Olympus and bestowed it as a gift which would set man apart from all other animals. And so it has. But the sword with which civilization was founded is a two-edged one, and since time immemorial the followers of Aesculapius have sought to bring relief to those luckless mortals who have felt its sting." (Barnes 1933).

This quotation, although it refers to Grecian Mythology, is very true in its statement that fire was the basis of the development of civilization. Throughout the Ages, fire has been one of the most useful physical elements which man has had. This is even more true in our present day life, because without it, industry could not exist. Although fire is essential for modern day life, it can do much destruction if not kept within its normal bounds. Likewise, it is capable of inflicting one of the most serious and painful injuries if it comes in contact with the human body.

It is this type of tissue injury, that I have chosen to discuss, so that I might familiarize myself with the more minute body physiology, and thus to be better able to chose the type of treatment which will bring about a cure.
DEFINITION AND CLASSIFICATION

A burn in its analysis may be stated as being an injury inflicted on the body by a degree of heat higher than is compatible with healthy action of the part affected.

The results of burns vary according to the several factors which play a part and which may be listed as follows.

1. The degree of temperature.
2. The nature of the exciting agent and its capacity for heat absorption.
3. The duration of contact.
4. The susceptibility of the part acted on and the condition of the patient.

Burns are classified according to several different methods. They may be classified as burns or scalds according to the type of heat injury, according to the area of skin surface involved, or according to the depth of tissue injury. The last method is a pathological classification, and is used by most men. However, in giving a prognosis on a burn case, they also consider the relative amount of skin injured, as the greater the proportion of the burn to the body surface the poorer is the prognosis.

In classifying the types of burns, we find several different variations taken from the Pathological study of burns. In America and Germany 3 degrees are usually discussed, while in France and England 6 degrees are discussed.
The American Classification includes the following:

A. According to Heister and Callison. (Pack 1926).

- **First Degree**: Erythema of the skin
- **Second Degree**: Formation of vesicles
- **Third Degree**: Formation of an eschar
- **Fourth Degree**: Charring of the tissues


- **First Degree**: Erythema
- **Second Degree**: Formation of vesicles and bullae
- **Third Degree**: Formation of an eschar or gangrene

We see from this that the classifications are essentially the same except in the latter classification a third degree burn includes everything, showing more destruction than simple vesicle formation.

Goldblatt (1927) discusses burns as being of two types. The first showing erythema and vesicle formation, and heals without scarring. The second type includes all other burns of more severe nature, and which heal by the formation of scar tissue and contractures.

The English and French Schools of Thought discuss burns according to 6 degrees. A good classification of this type is that of Dupuytren (Pack 1926).

- **First Degree**: Erythema of the skin caused by a temperature of about 140° F.
- **Second Degree**: Vesication caused by a temperature of 160° - 210° F.
- **Third Degree**: Destruction of the cuticle and part of the cutis vera, the tips of the papillary
down growths remaining intact;
Caused by a temperature of 210°F.

**Fourth Degree** Destruction of the entire integument and part of the subcutaneous tissue; caused by a temperature of 210°F or more over long exposures.

**Fifth Degree** Encroachment on muscles.

**Sixth Degree** Disorganization and charring of the tissue (carbonization).
The discussing of the minute pathology of burns will be taken up according to the last classification or that of Dupuytren and the data is that taken from Pack (1926).

In a First degree burn there is a simple erythematous flush. The vascular changes are similar to those seen in any inflammation and consists of a momentary contraction, with a subsequent vasodilation of the arterioles and venioles. This local widening of the capillary bed, due to the direct action of the irritant, is responsible for the increased rapidity of blood flow to the injured part which therefore accounts for the reddness and warmth of the injured area.

Microscopically, there is an active congestion of the part and a locally increased permeability of the walls of the minute blood vessels so that a filtering of plasma occurs out into the tissue spaces. Within the blood vessel margination of leucocytes occurs with a subsequent migration into the tissue spaces followed by a diapedesis of red blood corpuscles. The period of edema lasts ordinarily from 36 to 48 hours. Within a few days the upper layer of the epidermus separates in the form of scales or occasionally peels off.

In burns of the Second degree, the epidermal cells have undergone a true coagulation necrosis due to the conversion of soluble colloids into insoluble "gels". An exudation of fluid passes from the tips of the papillae into the epidermal layers, where the cells have been killed or injured by the heat and have become swollen and will
soon dissolve completely.

The serous infiltration is accompanied by an infiltration of mononuclear leucocytes. The rapidity of the exudation is dependent on the intensity of the stimulus, the sensitiveness, and the vascularity of the affected part.

When the blisters rupture due either to an accident or to a loss of elasticity with spontaneous breaking, a continuous discharge of serum occurs over the burned surface. Scarring does not result if the corium is not involved and if infection is absent.

The burned area in this type and in those of the first degree may remain pigmented and red for some period of time afterwards.

The Third degree burns show that the epidermus is completely destroyed and also part of the corium, but the tips of the inter-papillary processes remain intact. This is the most painful type of burn as the very sensitive terminal nerve filaments are left bare and exposed. The papillae of the skin appear as a reticular framework, containing serum, bits of persistent epithelium, leucocytes and masses of fibrin. Healing following this kind of burn occurs by the formation of a white elastic scar. This requires from 18 to 24 days to grow and does not result in contracture.

Fourth Degree burns are those in which the entire integument is destroyed, leaving two layers of skin, that destroyed and that which is only injured. The dead skin forms an eschar which is insensitive to touch and which fades off into hyperemic zones in which there are burns of the third, second and first degree. An acute inflammatory process starts around the retracting eschar and a groove results
between the living and dead tissues. A slough occurs usually taking about two weeks for completion. Fat may be liquified and may flow out from under the eschar.

The raw surface, after sloughing has occurred, is covered by a fibrinous exudate which exerts a chemotactic influence on the growth of tissue cells. The exposed blood vessels close by the formation of thrombin and from the capillaries grow out endothelial cells accompanied by fibroblasts and form granulation tissue which after a varying length of time becomes covered by epithelium from the edge of the wound.

A Fifth degree burn shows encroachment of the muscles by the destructive process. The pathology is essentially similar to a Fourth degree burn except that the burn is deeper, the surface is more deeply charred, the scar formed is deeper, more firm and immobile, and the disfigurement resulting is greater.

The Sixth degree burn is one in which the tissues are charred and carbonized, being converted into animal charcoal. Loss of a part may result from this type of burn and the disfigurement associated from the healing process may be great.
PHYSIOLOGY

The conception of physiological processes associated with burns seems to be changing rapidly especially during the last few years. Although there have been several recent advances along this line, the whole process is not definitely known.

It has been the belief of many authorities that the symptomatology of burns was due to a toxin liberated at or near the site of injury. Various men have had slightly different conceptions of this process. Ravdin and Ferguson (1925) have reviewed the literature and have enumerated the various theories which have been brought forth to explain these symptoms.

They discuss these under the following heads.

I Malfuction of the skin

A. Respiratory function lost with consequent asphyxia as believed by early writers.

B. Excretory function impaired with retention of poisonous substances (Billroth).

II Alterations in the Vascular System

A. Blood concentration (Underhill and his associates).

B. Dilation of peripheral vessels with rapid loss of body heat and general cooling of the body (Falk).

C. Diffuse thrombosis as the basis (Silberman and Savioli).
III Over excitation of the Nervous System

A. Whole condition one of shock which is followed by a period of reflex exhaustion with a loss of muscular tonus (Sonnenburg and Tachmarke).

IV Changes in the blood itself

A. Destruction of red blood corpuscles by heat and its consequences (Max Schulze).

B. Loss of function of red blood corpuscles (V. Lesser and Silberman).

C. An absorption of toxic substances into the blood.

1. Cotiana attempted to show the formation of hydrocyanic acid in the skin of burn cases.

2. Kijanitzen believed the toxins were due to an altered metabolism.

3. Lustgarten believed that toxins were due to absorption products of bacterial action. (Recently Aldrich and Firor have attempted to show that infection is the cause of the characteristic syndrome).

4. Boyer and Gunnard and more recently Robertson and Boyd believed the action of heat on the tissues caused the formation of some toxin which was absorbed.

It can be seen from the above that there have been many
different ideas as to the causation of the abnormal physiology and
due to this fact we must examine the experimental data and draw our
own conclusions from the evidence discussed.

Of the more widely accepted and discussed theories, the
Toxin Theory of Robertson and Boyd, and the Blood Concentration Theory
of Underhill and his associates, seem to have the most experimental
data to back their conclusions. Their conclusions contradict in a few
instances and it would seem necessary for more experimental work to be
done before accepting one or the other conclusively.

The Toxin Theory has numerous adherents and among these are
Davidson (1925) and Ravdin and Ferguson (1925). The Blood Concentration
Theory is championed by Underhill, Kapsinow and Fisk from experimental
work done at Yale University and the New Haven Hospital. Its principles
are being applied to a certain extent by various men and the experimental
work which they discuss seems to throw great evidence that their con-
clusions are correct.

Robertson and Boyd base their ideas on experiments which they
performed on rabbits, dogs and guinea pigs. The first part of the
experiment consisted of burning anesthetized rabbits by the use of hot
metal plates and then observing the animals. They found that such
animals showed an immediate period of primary shock which was evidenced
by a circulatory collapse and a fall in temperature. This lasted for
6 to 8 hours. During the next ten to twenty-four hours the animals did
not appear very ill. In 24 to 36 hours after the burn, their so-called
toxic symptoms appeared. The temperature rose to 104° or thereabouts
(normal for rabbits is 100° to 102° F.). Food was refused. The animal
appeared drowsy or excitable or exhibited alternate periods of each. Convulsive twitchings and in a few cases general convulsions were noted. Some animals had diarrhoea with mucus in their stools. Two had melena. Hemoglobinuria lasting for 2 or 3 days was present in three cases. They also found the non-protein nitrogen of the blood to be increased 40% to 50% due chiefly to the greater increase in urea nitrogen. Eight cases proved fatal. In two cases death occurred in the first 8 hours and was attributed to primary shock. In five cases it occurred in from 36 hours to 5 days after the burn, and they attributed these deaths to toxic shock. One case died 3 weeks after the burn from secondary infection. They next burned the subcutaneous tissue on a different group of rabbits, after the skin flap had been dissected away, and obtained similar results. This also held true for muscle burns.

The next experiment was the transplantation of skin from one animal to the next, and they found if the burned area was removed within 8 hours, the burn toxemia would not develop. They also found that the normal animals on which the burned area was transplanted showed toxic symptoms within one hour.

An experiment similar to this was carried out by Vogt, at an earlier date, which is referred to by the above authors. It was one in which animals were connected together by the use of a vascular parabiosis and then separated at different intervals after the one was burned. He found that if they were separated within 12 hours no toxic signs developed in the unburned animal.

Robertson and Boyd also performed a series of experiments to
try and find out the nature of the burn toxin by the following procedures.

1. They injected blood serum from children suffering from burn toxemia into guinea pigs and rabbits and found no toxic symptoms developed.

2. Citrated whole blood was injected intraperitoneally into rabbits and guinea pigs. When the blood from normal rabbits was used no symptoms developed but when that of burned animals was used toxic symptoms and sometimes death resulted.

3. Extracts of ground burned skin were prepared by using normal saline or distilled water, allowed to stand for 12 hours, put in a canvas bag and the fluid squeezed out in a press. Four volumes of absolute alcohol were added to the fluid and then filtered. This extract was concentrated and the alcohol removed by evaporation in a vacuum at a temperature not exceeding 40°C. This was filtered through a Berkfeld filter and injected into animals. Extracts of normal skin and skin burned postmortem were also made and injected into animals. They found that the extract of burned skin proved toxic while that of normal skin or skin burned postmortem was not.

They concluded from their experiments the following points.

(a) Death from burning occurring within the first 24 hours is practically always due to primary shock; later than this it is due to toxic shock.

(b) The toxic substance is produced in larger quantities in skin burns. This circulates in the blood either in or absorbed by, the red blood corpuscles and causes the symptoms seen in bad superficial burns and in some cases death.
(c) Extracts of burned skin are toxic. Extracts of skin burned postmortem are innocuous.

(d) There is increased protein catabolism in burns as evidenced by the increase in non-protein nitrogen and urea nitrogen of the blood.

(e) The contents of burn blisters are not toxic.

(f) Whole citrated blood from burn cases is toxic. The blood serum is not. Solutions of the corpuscles properly treated are more toxic than whole blood.

(g) The toxic substance consists of two portions, one of which is thermostable, diffusable, and neurotoxic; the other is thermolabile, colloidal and necrotoxic. Chemically the toxin consists of primary and secondary proteoses.

(h) No evidence of the production of antibodies against the burn toxin could be found.

From their conclusions, one would suppose they had proven their points quite well. However, we find that some men do not get the same results. Kapsinow (1932) has repeated certain parts of Robertson and Boyd's experiment and comes to a very different conclusion. He prepared an extract of skin in the same manner as they did, and he found that the extract of both normal and burned skin gave toxic symptoms similar to those described as toxic by the two experimenters. Numerous repetitions of this experiment conclusively proved that the results were not positive as in some instances, the extracts of the burned skin proved non-toxic, whereas that from normal skin was toxic, conversely, the opposite was found to occur in some cases.
In other cases neither was toxic. Different temperatures were used to extract the alcohol, such as 100°C and 37°C with similar results. Since the elusive toxin was considered to be volatile when heated on the water bath, and inert when heated to dryness, the loss of toxicity was considered due either to the effect of heat upon the active agent or to volatilization.

Kapsinow then took a toxic preparation and heated it in a reflux for 30 minutes and after this time the preparation was still found to be as toxic as before. He then analyzed a potent toxic extract as used previously and found it to contain 9.8% (by volume) of alcohol. Experimenting with various dosages and strengths of solution, he found that the minimal effect was produced with 10 c.c. of a 10% solution. At this concentration, no odor of the alcohol was detectable. When 15% solutions were used the reactions were rapid and severe.

Harrison and Blalock (1932) experimenting with dogs, attempted to see if there were toxic substances produced at the sight of a burn. They transplanted burned skin from one dog to a normal animal. This was done aseptically in 10 cases, and in none were they able to get evidence of viability of the transplanted skin. Neither was there evidence of toxemia shown by the recipients.

They also burned anesthetized animals and using one group as a control, they experimented by removing the burned area at varying intervals after the burn. Six such experiments were done, and in all but one, the animal whose burned area had been debrided, lived for a shorter time than the other which had been left alone. In the sixth case, the two animals lived the same number of hours.
The same investigators transfused blood from burned dogs into normal dogs at varying intervals after the burn, this interval varied from $\frac{1}{2}$ to 91 hours.

In 12 of the animals no ill effects resulted. In one broncho-pneumonia developed, another died and an autopsy showed thrombosis of the right femoral vein and a pulmonary infarct (femoral veins were used for transfusion). A third died immediately following transfusion, apparently from shock or an anaphylactoid reaction.

We see from this latter group of experiments that the various investigators cannot substantiate Robertson and Boyd's conclusions so further work must be done before their conclusions can be accepted.

In support of the theory of Blood Concentration, as a cause of the so-called toxic symptoms, Underhill and his associates have performed a rather extensive series of experiments.

This idea of the physiology of such injuries arose from the association and treatment of lethal gas poisoning during the World War. In these cases they found that there was extensive pulmonary edema, which resulted from the irritation of the gas. This edema was very severe and as a result of this a very definite blood concentration occurred. They found that in such cases that if fluids were forced to such an extent that severe concentration could be averted, that some of the patients could be saved.

A very similar out-pouring of fluid occurs in burns. Considering this fact, Underhill, Carrington, Kapsinow, and Pack (1923) made a series of observations on a group of patients brought in to the New Haven Hospital following a local theater fire. Their
observations were to determine the extent of blood concentration and to see the results of therapy directed toward eliminating this as much as possible.

They divided the cases into two groups; those severely burned and those less severely burned. The patients were given first aid treatment and later treatment by paraffin film dressings, with subsequent skin grafting where necessary. The local treatment will not be discussed here, but I will attempt to summarize their findings on these cases from a physiological viewpoint.

The blood concentration was judged by the hemoglobin content of the blood. The first readings were not taken until 3 P.M. of the day following the accident. They had noticed previously, due to observations on laboratory animals that a blood concentration of 140% of the normal value if kept up for any period of time, was incompatible with life and a concentration of 125% indicates a precarious situation.

They found that in the cases of clinically severely burned patients, the hemoglobin was unusually high. In this first group the range was from 137% to 209%. They considered normal as about 110%. The patients belonging to the less severely burned class had a hemoglobin ranging from 129% to 150%. They then observed the cases when fluids were forced. The fluids given were water by mouth and small quantities of sodium bicarbonate. The amount taken by the individuals varied, but in general was from 4 to 8 liters per day. The hemoglobin values were studied regularly during the period of convalescence, and they found that during the first few days, there was considerable fluctuation. They attributed this fluctuation to the effect of a very unstable equilibrium of the water balance mechanism, and that the water
given during this time was used in saturating the badly dehydrated tissues. Following this period of fluctuation, there was observed a steady, fairly rapid fall in the hemoglobin content. Coincident with this decrease in blood concentration, improvement in the patient's condition was observed.

Out of the series of 21 cases so studied, 2 ended fatally. Of these cases, one died too soon for the forcing of fluid, and in the second case insufficient fluid was administered to change the blood concentration significantly. The first case died on the second day after the injury and the only hemoglobin reading taken was 200%. The other case showed a hemoglobin of 205% at 3 P.M., 203% at 7 P.M., and 197% at 10 P.M. This patient died the next day. Two more of this series of cases ended fatally, but not due to the burn itself. One died of a pneumonia 8 days after the injury, and the other died from the same cause several weeks later. The last hemoglobin readings taken on these patients were 118% and 94% respectively. The second reading was taken some period of time before death.

Doctor Underhill recognizes the lack of control patients in this study, however, he sites a patient who was burned in this same fire, and treated at home under a management in which the fluid intake was not forced. This case he says was especially badly burned and presented the typical signs and symptoms characteristic of intoxication from burns, chief among these was an active delirium and in this case measures had to be taken to keep the patient in bed. This was succeeded by a period of collapse and unconsciousness and death was anticipated. After a period of 8 days a consultation with the physician in charge
resulted in the active forcing of fluids. Previous to the administering of the fluid, the hemoglobin value was 163%. She was given 2 liters of 0.7% sodium chloride solution subcutaneously, and after a few hours she regained consciousness and was rational and was capable of cooperating in the taking of fluid. The blood concentration fell rapidly and the patient went on to recovery. Doctor Underhill emphasized the point that on the eighth day the patient had a hemoglobin as high as some of their cases which were seen on the first day and that in none of their cases did the hemoglobin content stay as high for as long a period of time.

Underhill and his associates also observed the blood pressure in these cases and found that in cases of severe concentration, that the pulse pressure was low and that this condition probably was one of impending shock. They also noted that with the forcing of fluid, the pulse pressure increased with the decrease in blood concentration.

To study the effect of burns on the body and the general body reaction to such an injury, Underhill, Kapsinow and Fisk (1930) and Underhill and Fisk (1930) did a series of experiments on laboratory animals.

The first experiment was to study the formation of edema following burns. They anesthetized rabbits and then burned the rabbits with hot plates. They found that there was a rapid development of cutaneous edema which reached a peak at the period of approximately 24 hours. They also found that this edema was sufficient to cause a marked blood concentration. The reabsorption of the fluid into the body was slow and not completed until the 5th or 6th day. They also
found that the heat might penetrate the interior of the body to a degree sufficient to lead to local circulatory changes inducing the formation of ulcers, hemorrhages etc., which from time to time have been ascribed to the effect of a burn toxin. Under experimental conditions they found the general body temperature to be little affected, and that if after a period of 24 hours, there was a rise in temperature, it was probably due to infection of the wounded area.

The same experimenters also observed the effect of burns on the capillary permeability. By the use of methylene blue and trypan blue intravenously, they found that for a period of 2 to 24 hours the transportation of edema fluid containing the dye was greatest. They measured this by an analysis of the edema fluid in which large amounts of dye could be recovered. They also noticed that if the dye was injected 8 hours or more after the burn, the edema fluid did not show its presence. They suspected from this observation, that the capillaries had reattained some of the normal properties of withholding some colloidal substances, and that permeability was decreased.

They also observed the reabsorption from the burned area by the use of phenolsulphonephthalein and strychnine. They found that if the two substances were injected into the burned area that absorption took place very slowly. When strychnine was used they found they could inject the lethal dose for rabbits into the burned area and if later than 1 hour following the burn, little ill effects developed. In only one case of a series of 10 rabbits did convulsions occur under these circumstances.

Kapsinow (1933) confirmed these results. He used the minimal
lethal dose of strychnine in most of his series. This was found to be .6 milligram per kilogram of body weight for a normal rabbit. In a few cases 5 times this dose was injected into the burned area with no harm resulting.

His statement concerning this experiment in relation to the toxin theory of burns is as follows: "The presence of an unknown toxin that would have a more rapid absorption rate and that would be more potent than strychnine as indicated in these experiments is absurd and to suppose such a toxin is but to muddle the solution of the burn problem".

From the experiments sited above, Underhill, Carrington, Kapsinow and Fisk concluded that the capillary permeability in the direction away from the capillaries was increased and permeability to the capillaries decreased. These findings, as shown by them would obviously tend to disprove the theory of their being a toxin produced at the site of the burn unless it is much more toxic and much more easily absorbed than is strychnine or phenolsulphonephthalein. This possibility, it seems to me would be quite remote.

The same investigators also determined the amount of edema fluid formation induced by a superficial burn which involved about 1/6 of the body surface of the rabbit. They found that the greatest water loss occurs in the first 36 hours and the most they observed was 70% of the total blood volume. This, if the same qualitative and quantitative figures be applied to a man weighing 65 kilos and with a total blood volume of 5,000 c.c. would mean a fluid loss of 3,500 c.c., which would greatly handicap the body in
maintaining the normal blood relationships. They found that the degree of burn did not play the same relative factor as did the extent of body surface involved. If this same type of reasoning were used in man, we can understand why on a basis of fluid loss that a burn of 1/3 of the body surface has been considered a fatal burn.

After a chemical analysis of the edema fluid, the same men concluded that it must be considered as blood plasma. A similar conclusion was reached by Beard and Blalock (1931) after their analysis of the fluid escaping at the sight of the injury.

Underhill and his workers also found that the blood chloride level remained about normal as long as the blood concentration was maintained at a normal value, and that if increased, the chlorides were reduced in amount. In rabbits, it was found that the equivalent of 36% of the sodium chloride content of the blood could be lost without altering its content in the blood stream.

Working with various types of dehydration, Underhill and Fisk (1930) concluded that through water deprivation or by an increased osmotic power of the blood, the tissues of the organisms could be made to lose their essential water with death resulting as an invariable consequence.

Blalock (1931) experimented with anesthetized dogs and after burning a portion of the body he observed the blood pressure and the amount of edema formed. The edema was measured by sectioning the animal in half and weighing the two sections of the whole area. In most cases about 1/3 of the body surface was burned.
He found there was a marked drop in the blood pressure and that the blood concentration was increased. If 100% hemoglobin is considered as normal, the average percentage at the end of the experiment was 148%. The average weight of the two halves of the body in the 18 animals showed the burned half to be the heavier. This amounted to 3.34% of the total weight of the dog.

Concerning the theories of burns, Blalock thinks that there is a great amount of good evidence which indicates that a toxin exerting a deleterious effect, is formed. However, he later states: "In the human being, the factor of loss of fluid after burns may be even more important than in the dog, since there is frequently copious weeping from the injured skin in man, and this is not encountered in dogs. It is entirely possible that such agencies as tannic acid and epinephrine exert their beneficial effects by preventing loss of fluids rather than stopping the absorption of toxins".

Fender (1933) feels that the burn toxin theory has not been adequately proven. Aldrich (1933) from a group of experiments in conjunction with Firor has attempted to prove that infection is the basis for the toxic symptoms. He also takes into consideration blood concentration and institutes treatment to eliminate this. A more detailed discussion of their work will be made later.

It is to be seen from the above experiments and discussions, that although the toxin theory is held by some, they still feel that it has not been adequately proven, and some have even gone so far as to state that the whole theory is unfounded, which theory is correct, if any, may be shown in the next few years after more experimentation.
McIver (1933) has made an interesting study of the blood picture of burned patients.

After making this survey of a group of patients he drew the following conclusions.

1. The white count is elevated in all cases of burns and in the more severe cases the count can reach an extraordinary height within a short time. In making a comparison of the fatal and non-fatal cases a high white count in general indicates a poor prognosis.

2. Blood concentration is an important factor as shown by the high red cell counts.

3. Slowing of the sedimentation rate occurs. This is due somewhat to blood concentration, but possibly infection in the burned area also plays a part.

4. The plasma protein remained somewhat near the normal levels and is probably related to the disturbances in water balance.

5. Normal non-protein nitrogen values were found in most cases. However, in the fatal cases it rose except in one instance. In two of the fatal cases it reached high values, being 140 milligrams percent in one and 130 in the other.

6. The blood sugar in the majority of cases showed a marked increase, reaching as high as 266 milligrams percent in one case. These values returned to normal within the first 24 to 48 hours. In some of the less severely burned patients there was no rise.

7. The carbon dioxide combining power on the whole was normal. The same was true of the blood calcium and phosphate values.
8. After an analysis of the blister fluid, his conclusions may be shown by this quotation from his article, "The loss of fluid is not simply a loss of water content of the blood, but a substance that closely parallels whole plasma. One would not therefore expect great changes in the individual constituents of the plasma following burns, but rather a lowering of the total volume of plasma with a resulting concentration of the blood".

In this series of cases, he observed the fluid intake and the urinary output. In the fatal cases the diminished output and the low excretion of chlorides were striking findings.

Davidson (1926) explains this reaction on the basis of a lowered blood chloride content below the renal threshold. Underwood, on the other hand, thinks the cause is a blood concentration so high that the circulation through the kidney is greatly diminished with a resultant inability of removing the urinary products from the highly viscid blood.
TREATMENT

The treatment of burns has never been a fixed routine, and most procedures have been quite unsatisfactory. As a result of this, we find that many different forms of therapy have been recommended by different men. A large portion of these procedures have been the use of protective ointments and lotions which would protect and soothe the injured surface.

Hippocrates as far back as 435 B. C. treated these injuries by the use of bee's wax with myrrh and certain balsams of an antiseptic nature. This type of treatment has continued in modified form in Europe throughout the centuries (Mitchiner 1933).

Another treatment of burns which was advocated by the physicians of China some 5,000 years ago was the use of tea. This method is quite universally used in China even to this modern period. (Mitchiner 1933). Although tea had been used for centuries in China, it was not until 1925 that the principle implied in this treatment was conceived by the modern practitioners of medicine. This was inaugurated by Davidson in the treatment of extensive cutaneous burns by the use of tannic acid.

Throughout the 19th Century we find various types of treatment and medication used with about equal results.

Cumin (1823) used vinegar locally and wine and anodynes at bedtime. Turpentine dressings were also used, Pirrie (1887) used carbolic acid solutions and was one of the first to use the stronger antiseptics as a dressing for burn injuries. Morris (1882) likewise used more or less sterile precautions. He opened the blebs, which
formed and then applied iodoform powder, cheese cloth with vaseling, and then a sheet moistened with carabolic acid (2%) and the whole enclosed in gutta percha tissue. Copeland (1887) was one of the early advocates of the open air treatment of burns. He left the injured surface free to the air under a box covered with cheese cloth.

From this time on there were newer methods of treatment. Sir Cuthbert Wallace in 1899 introduced the use of Picric acid and this according to Mitchiner (1933) materially cut down the death rate. Previous to this time some were removing the infected material and applying local antiseptics.

The use of carron oil and the paraffin wax dressing were also recommended by some, but on the whole these procedures have given way to newer types of therapy. It was at a little later period that different investigators began to experiment, attempting to analyze the nature of a toxin which some thought to be present in the burned individuals. The toxin theory of burns was not a new theory at this time, however little was known about its nature and due to this, numerous methods of treatment were instituted attempting to correct or prohibit such a toxic state.

According to Davidson (1925) it was Wiener in 1905, who found that the intracellular proteoses acted only in a faintly acid medium, and that by changing the hydrogen ion concentration, the effects of the proteoses which they thought were responsible for the toxic symptoms, could be prohibited from exerting a deleterious effect. Due to this idea, the use of sodium bicarbonate compresses and baths was instituted by some.
Another method of procedure which was directed toward eliminating the so-called toxins of burns was the use of continuous baths in the hope that this would wash away the toxin or so dilute it that the amount absorbed would be minimal. This according to Davidson was known as the Hebra method of treatment although Rose (1906) records the method as being introduced by Passavant in 1857.

Vogt in 1913 (Davidson 1925) showed that the early removal of the affected area would decrease the toxemia or keep it from developing. Thus Broger in 1913 used this as a method of treatment by removing as much necrotic tissue as possible under general anesthetic or large doses of morphine and then washing the bleeding surface with gasoline.

Douglas in 1923 applied epinephrine in a dilution of 1 to 10,000 to the burn hoping to so constrict the blood vessels that the rate of absorption would be so slowed that the concentration of the toxins would be decreased beyond a point that no symptoms would develop.

Ravdin and Ferguson in 1925 proposed a similar treatment in which novocaine, adrenalin and dichloramin was used.

Another possible means of limiting absorption of the toxins from the wound was then conceived. It was the use of some chemical which would coagulate the burned skin and thus not allow the toxins formed to be taken up by the blood stream. For this purpose some men had used bichloride of mercury, and others had used phosphotungstic acid (Davidson 1925). These chemicals, however, were
found to be somewhat dangerous to use, because absorption of the chemical would occur with harmful results.

Davidson was looking about for some non-toxic chemical which would coagulate tissue. It was suggested to him that he try tannic acid since it was found to coagulate proteins similarly to phospho tungstic acid.

Chemically tannic acid is a non-nitrogenous, amorphous powder, which is readily soluble in water, glycerine and alcohol. It precipitates proteins, alkaloids, some glucosides, and the salts of the heavy metals. It forms a more or less stable compound with the protein constituents of the body fluids and cells. When applied to a burned surface in dilute solution, further penetration into the deeper lying protoplasm is apparently prevented by the coagulating action, and the true astringent effect appears to be limited exclusively to the more superficial layers of the tissue.

Due to these properties, Davidson (1925) used tannic acid on a series of cases of burns, and concluded that this method of procedure lessened the toxemia.

It might be well to include here, that by this time, the period of burn injury had been divided into 3 stages. The first stage was the period of shock which lasts up to the first 24 hours. (Robertson and Boyd 1923). The second stage was the so-called toxemic stage, which lasted from 24 to 48 hours and according to some men, a longer time. Following this there is a third stage, which is the infection stage, and if death does not occur, is followed by repair period.

The treatment of the first stage is much the same, no matter
what the subsequent treatment is to be. This includes the use of heat in the form of hot packs or hot water bottles, warm fluids by mouth or rectum, and possibly normal saline or glucose solutions by hypodermoclysis. Associated with this treatment enough morphine is given to control pain and to quiet the individual. In addition to this some men recommend the use of atropine.

It is during the second and third stages that the above methods of procedure were found to be the most useful. Davidson recognized this fact and experimented with the use of tannic acid. He found that when applied to a burned surface, tannic acid was also analgesic, and thus eliminated much of the pain which consequently reduced the amount of shock. He also concluded that this treatment when followed by open air method, causes a minimal trauma and promotes the general comfort. The local astringent effect also reduced the fluid loss and formed a coagulum which greatly reduced the amount of infection by not providing a favorable nidus for bacterial growth. He also noticed that scar tissue formation was less after this treatment than following the use of any other method since the protective layer of coagulated tissue formed a scaffold for the growth of the young epithelial cells over the denuded surface.

Since Davidson was the first to use the tannic acid treatment of burns, it might be well to give his method of procedure and to discuss other methods as variations from this.

Davidson states that as soon as the patient has been seen, he should be given a large dose of morphine sulphate hypodermically (for an average adult $\frac{3}{4}$ grain) to alleviate the intense pain. The
burned area is then covered with dry sterile gauze pads, which are held in place by sterile gauze bandages. These dressings are then soaked with 2.5% aqueous solution of tannic acid. He thought this strength was the most desirable after the use of various concentrations from .75% to 5%. It is essential that the tannic acid solution be made up fresh just before use, because it deteriorates upon standing with the formation of the far less astringent gallic acid. He states that to prevent a deep caustic tissue injury, small sections of the dressing should be opened for inspection at the end of 8 - 12 and 24 hours. As soon as the part assumes a light brown color, all dressings are removed. The dressings are first moistened with fresh tannic acid so that they may be removed without pain. The wound thereafter is left exposed to the air, but is carefully protected from mechanical injury, chilling, and bacterial invasion by a suitable cradle draped with sterile linen. In more serious cases, artificial heat has been supplied, by placing within the cradle, so prepared, one or more ordinary light bulbs.

In a few cases he used a 5% tannic acid ointment made by using equal parts of vaseline and lanolin as a base. This was substituted in place of the aqueous solution, and although it had a definite beneficial effect, it was far less efficacious than the solution. He says the chief value of the ointment is in the use about the eyes, where the astringent solution cannot be used with entire safety.

To quote from his article concerning fluids, we find he advocates forcing by any means which is necessary. He states,
"One of the most essential features of the management of all burn cases is that of keeping up the fluid balance in the body. This is accomplished by forcing fluids by mouth, where possible, or by hypodermoclysis, proctoclysis or intravenous infusions according to the special indications in each case. Blood transfusion has been employed in some of these cases, apparently with favorable effects".

Numerous surgeons and general practitioners since 1925 have adopted the tannic acid treatment. Many use the 2.5% strength recommended by Davidson. Among these are: Bledsoe (1932), Christopher (1928), Ghormley (1933), and Wilson (1933). Others use 5 - 10% strengths of the solution and among this group some of them are: Glover (1932), Hunt and Scott (1932), Strauss (1932), Wells (1933), and Lee (1928).

Christopher (1928) has reviewed the current literature concerning the treatment of burns and he then gives his method of treatment. This is very similar to that of Davidson and includes morphine, heat and fluids to prevent the primary shock. For the toxic stage he uses tannic acid locally and the pushing of fluids, so that 4,000 to 5,000 c.c. of fluid are given by hypodermoclysis, proctoclysis or infusion. Sodium bicarbonate is given by mouth and if the tissues become water-logged, he gives magnesium sulphate. If there is evidence of adrenal deficiency, he thinks epinephrine is indicated.

The same author discusses an interesting method of tanning the skin by the use of tea, which was advocated by Shen in the China Medical Journal. By this method Shen found that an infusion of tea was nearly as effective as the ordinary tannic acid solution. His
directions for preparing such a solution are as follows:

One gram of tea leaves in 100 cc of boiling water is considered a 1% infusion. The infusion to be effective for protein precipitation must be 8%. Thus a 15 minute 8% infusion is prepared and sterilized by boiling and can be used locally over the burned area.

It may be well to state that not all men use tannic acid in the form of packs, but use an atomizer and spray the solution on at various intervals until the area is coagulated. Between intervals of spraying, the burned area is left exposed to the air. For this type of procedure Glover (1932) uses strengths of 5 to 10%. Glover also recommends the active forcing of normal salt solution or 5% glucose which may be given according to the rule of 100 c.c. per kilogram of body weight per day. This rule is more or less flexible, and one should adjust the fluid intake according to the individual case, as this amount of fluid may cause edema in a few patients.

In the tannic acid treatment, after about 6 or 8 days, and exudate may form under the coagulum, when this occurs Glover uses Dakin's solution to remove the crusts. He also says that at this time, the toxic state, which sometimes occurs, resembles very closely the toxemia of a mild acute infection or a sapremia. He also states that the Dakin's solution tends to control this condition.

Seeger (1932) has reported results of experimental work on the use of tannic acid in which he changed the pH by the use of sodium carbonate. He states that ordinary tannic acid is strongly acid and highly astringent, which tends to cause swelling and edema.
of the tissues and a too rapid fixation of tannin at the surface. He then neutralized solutions to different pH values, and his conclusions were that the best results occurred when the pH was somewhere near that of the blood. He was not able to show any reduction in tanning power by this neutralization. He also gives a formula, which makes a 5% solution with a pH of about 7.4. This formula is as follows:

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure anhydrous sodium carbonate</td>
<td>3.975 gms.</td>
</tr>
<tr>
<td>Pure tannic acid</td>
<td>25. gms.</td>
</tr>
<tr>
<td>Water</td>
<td>500. c.c.</td>
</tr>
</tbody>
</table>

Within the past year or so a slightly different treatment for burns has been suggested. The principle is very similar to that of tannic acid. The method is the use of gentian violet as a spray or jelly. This treatment was first suggested in the literature by Aldrich in 1933. He gives Firor of Johns Hopkins Hospital the credit for conceiving their theory of the cause of toxic symptoms and the subsequent use of gentian violet.

Due to the characteristic symptomatology associated with the so-called toxemia of burns and the amount of infection, which is present in many cases, Firor decided to study burns from a bacteriological standpoint. He and his associates cultured burns as soon as the patients were taken in and before treatment had been started.

He also cultured the areas repeatedly for a period of several days following the burn. They found that during the first 12 hours, the areas were practically sterile. They did find, however, a few contaminants such as Staphylococcus aureus, albus, and Bacillus coli.
After this period of time they found in 100% of the severely burned patients, and in the majority of minor burns that there could be grown from repeated cultures the beta hemolytic or the gamma streptococcus. They state that the concentration of these organisms increased with the obvious signs of sepsis and the beginning of the toxicity of the patient, until after 48 to 56 hours, when pure cultures of the streptococcus could be obtained. Coincidentally with this, the characteristic bad effects of the burn were shown by the patient. According to their reports, the general condition of the patient, and the characteristic chart of the fever would be one which coincided with that of any wide-spread streptococcus infection. Additional evidence was obtained by taking blood cultures from patients after the temperature had started to swing in the picket fence curve. These cultures were positive for the invading streptococcus. Another bit of evidence in favor of the infectious theory is that in fatal cases the hemolytic streptococcus was found in the heart blood and also in the lungs in cases of terminal pneumonia.

Wells (1933) was not able to confirm the report of Aldrich and Firor that the streptococcus outgrows all other organisms, and so we must leave this point more or less unproven.

However, since Firor found the streptococcus to be so commonly present in burns, he thought that if he could use some substance which would kill the organisms, possibly he could show that no toxic symptoms would develop.

Tannic and picric acid while slightly antiseptic are not
specific for the streptococcus. Also after the use of tannic acid the eschar formed would prohibit any penetration and thus no action on the underlying infection could be obtained.

Aldrich and Firor did not agree with Davidson that the beneficial results of the used tannic acid was due to the rendering of the toxins non-absorbable by the precipitation, but that the effect was due to the sealing off of the burn which would prevent fluid loss and cut down infection. Both of these latter factors, they considered to be the important elements in the causation of the so-called toxemia of burns.

There are also several disadvantages to the tannic acid eschar according to Aldrich and these may be listed as follows.

1. The eschar is brittle and will crack if exposed to strain.

2. The eschar is quite dense and fluid proof so that any underlying infection will not present itself until quite extensive.

3. Minute islands of unburned epithelium such as those found in practically all third degree burns are not preserved but are destroyed by the infection beneath the eschar.

Due to these factors and to the fact that they wanted a chemical, which was more or less specific for the streptococcus, they hunted for some new compound.

Aldrich states that Churchman in 1912 proved that gentian violet was practically a specific for the gram positive organisms
and this led Firor to the use of it in burns. The effect of gentian violet on organisms was rechecked by Firor and confirmed. They found that a dilution of 1 to 1,000,000 would inhibit growth, and that a concentration of 1 to 10,000 would kill the gram positive organisms. It was also shown that gentian violet could be used intravenously in $\frac{1}{2}$ to 1% solution with no untoward symptoms.

Gentian violet or tri phenyl methylamine which is a coal tar derivative, is slightly alkaline and is quite soluble so that a 1% aqueous solution can easily be prepared.

It was also found that gentian violet, due to its PH reaction, together with the contained methyl radical, will react with the burned flesh to form a thin light eschar, which is tough but flexible. This eschar when situated in the folds of the body will bend but not crack. Thus when it is used on a burned surface it causes a tough firm eschar which is antiseptic and in addition to this gentian violet produces a practically instantaneous analgesia, which also tends to decrease the amount of pain and shock.

From the discussion of Aldrich, it would seem that we have an almost ideal drug to use in treating burns. They used this in the treatment of several injured patients with very gratifying results. Their method of application is as follows: "When a patient is admitted with a fresh burn, unless it is covered with oils or grease, there is no need to do any preliminary cleaning. The dye in 1% aqueous solution is sprayed on the burned area immediately. The usual procedures for combatting shock are carried out and the patient sent to the ward with the burned surface uppermost. No dressings are used, but the burn is
covered with a cradle containing electric lights and the temperature maintained between 84°F and 88°F.

For the first few hours gentian violet is sprayed on the burned areas every 2 hours. This seals the burn by a light eschar and the wet oozing areas become dry and tough. Further infection is thus kept out and the loss of fluids through the serous ooze is eliminated. Likewise by the time the preliminary sedative has worn off, pain has ceased. After the eschar has formed, the patient is sprayed every 4 to 6 hours during the day. Any blebs which form are opened and the unstained portions then presenting are sprayed. This treatment continues until healing is complete.

Aldrich states that in practically none of the burns, unless actual charring has taken place, is all the epithelium destroyed, but that there exist many small islands throughout the lesion which will live and spread rapidly if not destroyed by sepsis. He also says that if a patient presents an old burn which is already septic, the gentian violet treatment can be started immediately without an initial clean up. An eschar of pus and necrotic tissue forms which is irregular, in pieces and floating, and can readily be removed every day and the freshly exposed area sprayed immediately.

Aldrich also cautions one that in very extensive burns, the whole area cannot be left upwards and open to the air. These areas must then be considered as potentially infected. He says that each day the eschar should be examined for softening, which may be due to the secretions of the body, liquified and necrotic fat or infection, and that when this occurs the area must be opened and the
exposed area sprayed in the usual manner.

The patients they have treated did not feel bad, but usually lie quietly in bed in no apparent pain, are not stuporous, have no fever and are usually willing to take foods and fluids by mouth. Throughout their series of cases they forced fluids and helped maintain a normal blood concentration. In less extensive burns the patient feels quite well and they allow him to sit up and many times walk to the bathroom.

In Aldrich's discussion four burn cases are discussed which had been treated by the gentian violet method. I will give a brief synopsis of one of their cases which turned out fatally.

The patient was a 9 year old white female, whose clothing had caught fire and were practically burned off. She was found to have a 3rd degree burn of the entire thorax, entire circumference of the neck, occipital region of the head, both arms from the shoulder to the mid-lower arm, both axillae, the left thigh on its lateral aspects for about \( \frac{3}{4} \) its surface and both ankles. Their estimate of the burn was somewhat over \( \frac{3}{5} \) of the body surface. As soon as admitted the patient was put in a sterile sheet and then blankets and a cradle placed over the bed and hot water bottles put about her. Normal saline was given in the right thigh and in 1 hour intravenous saline and glucose was started. About 4 hours later the burned areas were sprayed with gentian violet. This was repeated every half hour. At no time did the patient complain of pain and one day later she was entirely out of shock. Her temperature which on admission had been 96°F had now risen to 102°F. The patient was forced to lie on her
back and to keep this part sterile, she was placed on large pads soaked in hexyresorcinal. Repeated transfusions were done. As the eschar softened it was cut away. Her temperature continued to run in a picket fence manner, ranging from 100° - 103° F. After 2 months the thorax, arms and left thigh appeared ready for skin grafting. However, on the 80th day, the child showed evidence of cardiac fatigue and in spite of the fact she had been digitalized, she died on the 84th day of decompensation. Up until her death, the blood showed no changes other than a leucocytosis. Repeated blood cultures were negative, and autopsy showed only passive congestion of the liver, kidneys and spleen.

Since the original article of Aldrich was published, there have been several other men writing their results with the use of gentian violet.

Penick (1933) discusses its use and stresses the importance of forcing fluids in addition. He thinks that 100 c.c. of fluids in the form of normal saline or 5% glucose solution, per kilogram of body weight per day, is a good starting point and one should vary the amount according to the individual patient's needs.

Connell, Fatheree, Kennedy, and McSwain (1933) report a series of 15 cases treated with 1% gentian violet in aqueous solution alone or with 3% tragacanth with very favorable results. They used the jelly form in 5 cases of comparatively minor burns with excellent results. They concluded that the gentian violet treatment gave excellent end results and from their small series of cases, they preferred the jelly form to the aqueous solution.
Warthen (1933) also discusses the use of gentian violet and gives statistics showing how since the advent of its use the mortality rate from burns has been reduced at the Johns Hopkins Hospital.

It is rather unfortunate for this discussion that the tannic acid treatment had not been used over an extensive series of cases so that a comparison of the two methods could be made. This type of treatment apparently had been used only during the latter part of 1925.

A table showing the comparative results of the different types of treatment is inserted here and the change in mortality rate after the use of gentian violet is very evident.
<table>
<thead>
<tr>
<th>YEAR</th>
<th>NUMBER OF PATIENTS</th>
<th>TREATMENT</th>
<th>NUMBER OF DEATHS</th>
<th>MORTALITY RATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1921</td>
<td>14</td>
<td>Vaseline gauze, boric acid compresses, continuous tubes or carron oil, fluids by mouth, subcutaneously, or rectally, occasionally small amounts of 5 and 10% dextrose were given intravenously. Saline or Dakin's solution compresses to granulating wounds and later Thiersch or Reverdin grafts.</td>
<td>6</td>
<td>42.8</td>
</tr>
<tr>
<td>1922</td>
<td>16</td>
<td>Same as 1921 with occasional picric acid and ambrine sprays.</td>
<td>7</td>
<td>43.7</td>
</tr>
<tr>
<td>1923</td>
<td>36</td>
<td>Same as above with occasional blood transfusions.</td>
<td>14</td>
<td>38.8</td>
</tr>
<tr>
<td>1924</td>
<td>33</td>
<td>Same as 1923 with occasional blood transfusions.</td>
<td>14</td>
<td>38.8</td>
</tr>
<tr>
<td>1925</td>
<td>33</td>
<td>Vaseline gauze or continuous tubes, for first half tannic acid compresses with dry heat, fluids forced.</td>
<td>14</td>
<td>42.8</td>
</tr>
<tr>
<td>1926</td>
<td>22</td>
<td>Dry heat alone exsanguination followed by transfusion, intravenous glucose more frequently than before.</td>
<td>10</td>
<td>45.4</td>
</tr>
<tr>
<td>1927</td>
<td>13</td>
<td>Same as 1926.</td>
<td>4</td>
<td>30.7</td>
</tr>
<tr>
<td>1928</td>
<td>13</td>
<td>Same except 1 patient received convalescent burn serum.</td>
<td>11</td>
<td>35.4</td>
</tr>
<tr>
<td>1929</td>
<td>31</td>
<td>Firor's use of Gentian violet.</td>
<td>4</td>
<td>12.9</td>
</tr>
<tr>
<td>1930</td>
<td>30</td>
<td>Forcing of fluids by mouth, reaction under the skin or intravenously. If necessary a canula was kept in the vein continuously through which saline or 5% glucose was given.</td>
<td>3</td>
<td>13.0</td>
</tr>
</tbody>
</table>
The preceding table shows the only comparative studies of gentian violet and other methods. However, there are numerous statistics comparing the tannic acid treatment with older methods. Of these some of the more outstanding are: Bancroft and Rogers (1926) report that the use of tannic acid is a distinct advance in the treatment of burns which is substantiated by their mortality rate, which was reduced from 40% to 50% down to 20%.

Barnes (1933) has shown that by the use of tannic acid that some burn cases which would probably have died by the use of older methods have been saved.

Herzfeld (1929) has a reduction in mortality rate from 38% to 9%.

Beekman (1929) from 27.8% to 14.9%.
Glover (1932) from 14% to 9.6%.

Mitchiner (1933) of St. Thomas Hospital in London shows a reduction from 9.4% to 2.4%.

Wilson (1932) Royal Hospital for Sick Children at Edinburgh shows a reduction from 35.4% to 8.8%.

Donald (1930) does not view the effects of tannic acid in the treatment of burns as such a great advance as he states the mortality rate had been dropping for several years. He, however, does concede that tannic acid does have a place in the treatment of burns. Although Donald does not view the use of tannic acid as a great aid in the treatment of burns, it seems that after observing the change in mortality rate we must concede that the modern treatment is a distinct advance over the older methods.
Whether this is due to the local treatment or whether it is due to some other factor, it is hard for me to say. However, it is the opinion of most of the modern writers that the local treatment is responsible for many of the favorable end results.
1. Burns may be classified according to several different methods. That used by most men is dependent upon the depth of tissue injury. For this classification the American surgeons divide burns up into 3 degrees. The first shows erythema, the second vesicle and bullae formation and the third includes any cutaneous injury involving a depth greater than just the epidermis.

2. The Pathology associated with these injuries varies from a simple erythematous flush due to a vasodilation of the peripheral arterioles and venules to a coagulation or charring of the devitalized tissue. Injuries of this latter type usually show necrosis or char-ring in the center with the periphery showing vesicle formation. This in turn gives way to erythema and then normal skin. With the more severe injuries there is some thrombosis of the peripheral vessels and leucocytic infiltration into the surrounding areas. From the exposed papillary projections, serum in varying amounts is discharged. If the area involved is quite large, this might result in a serious fluid loss. As healing occurs, granulation tissue is formed and this may form dense fibrous connective tissue and lead to a serious deformity.

3. The so-called "toxemia" of burns is accepted by many as the cause of the characteristic symptomatology. Robertson and Boyd did some very good research on this subject and they concluded that the toxin was formed at the site of the tissue injury and consisted of two parts. One part was a substance which was thermostable,
diffusible and neurotoxic, while the other part was thermolabile, colloidal, and necrotoxic. They stated that the toxin consisted of primary and secondary proteoses.

Several physiologists have rechecked certain parts of the experiment of Robertson and Boyd and have not been able to confirm their conclusions. Some of these men are inclined to take a very different opinion concerning the so-called "Toxemia" which is present in the more severe burns.

4. Underhill and his associates believe this characteristic symptomatology is due to a concentration of the blood which has been shown to occur in severe burn injuries. They also stated that when this reaches a certain degree and remains there for any length of time, death will result. They have found that for certain animals a concentration of 125% of the normal indicates a precarious condition and one of 140% if maintained for any period of time is incompatible with life. They have also shown that the fluid lost from the tissues is practically identical in its chemical and physical nature to blood serum. For this reason some have considered it as such.

5. The blood count after a severe burn shows a marked increase in red blood corpuscles which is the result of the blood concentration. The white count rises quickly to very high values and according to McIver the higher the white count the more grave is the prognosis.

6. The treatment of burns must be divided up into 3 stages. The first is the primary shock period and lasts usually not over 24 hours. It is treated by the use of heat to make the patient warm,
fluids to keep up the blood volume and morphine in sufficient doses to relieve the patient of pain and to do away with anxiety and nervousness. The fluids should be given by mouth, hypodermoclysis or by intravenous infusion. Those commonly recommended are normal saline and 5% glucose in normal saline solution.

7. The so-called "toxic" period is treated by several methods. The tannic acid treatment of Davidson has been found to be very good and is recommended by many surgeons. In this type of treatment, a fresh tannic acid solution is prepared in a strength of 2.5% to 10%. The weaker solutions are usually applied in the form of packs while the stronger solutions are sprayed on at frequent intervals by the use of an atomizer. This type of treatment is continued until a brownish coagulum is formed and then the burned area is placed under a cradle kept at a constant temperature by the use of electric light bulbs. At this same time fluids are forced by any method that is found necessary. Some have recommended as high as 100 c.c. of fluid per kilogram of body weight per day as the amount necessary to treat the blood concentration.

8. Another more recent form of treatment which has been recommended, is the use of 1% aqueous solution of gentian violet. This is sprayed on the burned area at intervals of one half to 2 hours during the acute stage. After 24 to 36 hours the interval is lengthened so that the sprays are given every 6 to 8 hours. Also along with this treatment, fluids are forced as in the use of tannic acid. It has been shown that gentian violet precipitates the burned tissue so that a flexible coagulum results. It has also
been found to be antiseptic and analgesic which is very important in reducing the amount of infection and pain.

9. The treatment of burns by the use of carron oil, picric acid, paraffin wax dressings, and the like have apparently given way to the more recent methods such as tannic acid or gentian violet.

10. The infectious period in the tannic acid treatment is controlled by the use of Dakin's solution compresses or some other antiseptic which will loosen the coagulum when this is thought to be necessary. When gentian violet is used, the sprays are continued through-out this period. All blebs are opened as soon as noticed and the exposed surface sprayed.

In both methods of procedure great precautions should be taken to prevent contamination and infection from the body secretions or excretions.

11. When there is a large area of unepithelialized surface, skin grafting may be necessary.
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