thermal burns

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THE PROBLEM OF THERMAL BURNS: 1944

HENRY N. HARKINS, M.D., PH.D.
BALTIMORE

Before 1939 an average of 5,000 to 7,000 burn deaths occurred in the United States every year.[1] The present war has resulted in an unprecedented number of thermal burns, so that this subject is now in a phase of especial importance.

Burn treatment has always been somewhat empiric. The peculiar nature of the burn wound, representing as it does an actual destruction of tissue, renders no treatment completely effective. Because of this fact literally hundreds of burn remedies have been suggested. It is only recently that some order has come out of all this confusion. Possibly the two chief advances that have led to this are (1) the introduction of plasma therapy by Weiner, Rowlette and Elman [2] in 1936 and (2) the application of the method of aseptic pressure dressings in local burn therapy by Koch [3] in 1941. It is interesting to note how these two advances dovetail, indicating the essential unity of all burn therapy. The use of plasma is essentially a general treatment and pressure dressings are a type of local treatment, yet part of the benefit of the latter comes from reducing plasma loss and aiding the general condition of the patient. In fact it may be said that no good local burn treatment will neglect the general condition of the patient and vice versa.

The problem of thermal burns is essentially twofold: mortality and morbidity; the patient and the burn wound; general care and local management. Both of these are important and, as Pack [4] has so aptly said, "it is penny wise and pound foolish to consume invaluable time in applying perfect local dressings while the patient is sinking into irrecoverable shock." This symposium attempts to cover both of these aspects of burn treatment as completely as is possible in the space provided.

The following outline shows the division of the subject into seven articles:

1. The Problem of Thermal Burns: 1944. Henry N. Harkins, Johns Hopkins Hospital, Baltimore.

2. The Chemical Aspects of Burn Treatment. Oliver Cope, Massachusetts General Hospital, Boston.

3. The General Care of the Burned Patient. Conrad R. Lam, Henry Ford Hospital, Detroit.

4. The Present Status of the Tannic Acid Method in the Treatment of Burns. Walter E. Lee and Jonathan E. Rhoads, Pennsylvania Hospital, Philadelphia.

5. Surgical Cleanliness, Compression and Rest as Primary Surgical Principles in the Treatment of Burns. Sumner Koch, Northwestern University, Chicago.

6. The Early Plastic Care of Deep Burns. Fraser B. Gurd and John W. Gerrie, Montreal General Hospital and McGill University, Montreal.

7. The Late Plastic Care of Burn Scars and Deformities. John Staige Davis, Johns Hopkins Hospital, Baltimore.

From the Department of Surgery, Johns Hopkins University, School of Medicine.

This paper, in a symposium on "The Treatment of Burns," is published under the auspices of the Section on Surgery, General and Abdominal.

1. Harkins, H. N.: The Treatment of Burns, Springfield, Ill., Charles C Thomas, Publisher, 1942.
2. Weiner, D. O.; Rowlette, A. P., and Elman, R.: Significance of Loss of Serum Protein in Therapy of Severe Burns, Proc. Soc. Exper. Biol. & Med. **34**: 484-486 (May) 1936.
3. Koch, S. L., cited by Mason, M. E.: Local Treatment of the Burned Area, Surg., Gynec. & Obst. **72**: 250-253 (Feb.) 1941.
4. Pack, G. T., cited by Harkins.[1]

The present article outlines the important questions pertinent to the subject at the present time, many of which are answered or clarified in the remaining articles. Newer knowledge of the underlying chemical changes in burns on which all treatment, local and general, is based forms the thesis of Dr. Oliver Cope's paper. The practical application of this knowledge to the all important subject of the general care of the burned patient is delineated by Dr. Conrad Lam. Drs. Walter Lee and Jonathan Rhoads discuss the present status of the treatment with tannic acid, which, after a meteoric rise in popularity following its introduction in 1925, was probably the most widely used remedy until its recent displacement by the pressure dressing method. The latter method is described by its originator, Dr. Sumner Koch. The early plastic care of granulations by skin grafting, without which the treatment of no extensive third degree burn is complete, is covered by Drs. Fraser B. Gurd and John W. Gerrie of Montreal. Finally the subject of the late plastic care of burn scars and deformities, which will be a very important one in the immediate postwar period, is outlined by Dr. John Staige Davis.

GENERAL CARE

Early General Care.—This involves the prevention and treatment of shock, toxemia and sepsis. During the first forty-eight hours shock is of prime importance and the use of plasma is the chief item in its treatment. Since its introduction in the treatment of clinical burns by Weiner, Rowlette and Elman [2] this method has become so widely adopted that arguments in its favor are no longer necessary. It is enough to say that since the chief element in the pathology of burns is an extensive plasma loss, both to the outside in the form of weeping and into the burned tissues, it is of great importance that this plasma should be replaced. In very serious burns the patient may lose several liters of plasma within a few hours. If plasma is not available, albumin or even whole blood may be given although the contained cells in the latter are not necessary.

Plasma administration in burns should be on a quantitative basis just as is insulin administration in diabetes. When laboratory facilities are not available, the following ways of gaging dosage are useful:

1. From the clinical response.

2. From the size of the burn as calculated by Berkow's formula.[5] Author's rule:[6] Give 50 cc. of plasma for every 1 per cent of body surface burned; after twelve hours additional plasma may be necessary.

If laboratory facilities are available, the following methods of estimating plasma dosage may be used:

1. Formula of Black.[7]
2. Formula of Elkinton, Wolff and Lee.[8]
3. Formulas of author.[9]

5. Berkow, S. G.: A Method of Estimating the Extensiveness of Lesions (Burns and Scalds) Based on Surface Area Proportions, Arch. Surg. **8**: 138-148 (Jan.) 1924.
6. Harkins, H. N.: The General Treatment of the Patient with a Severe Burn, in Burns, Shock, Wound Healing and Vascular Injuries, Military Surgical Manuals, National Research Council, Philadelphia, W. B. Saunders Company, 1943, vol. 5, chapter 1, pp. 3-26.
7. Black, D. A. K.: Treatment of Burn Shock with Plasma and Serum, Brit. M. J. **2**: 693-697 (Nov. 23) 1940.
8. Elkinton, J. R.; Wolff, W. A., and Lee, W. E.: Plasma Transfusion in the Treatment of the Fluid Shift in Severe Burns, Ann. Surg. **112**: 150-157 (July) 1940.
9. Harkins, H. N.: The Treatment of Burns, with Particular Emphasis on the Management of Burn Shock, brochure distributed in connection with Scientific Exhibit at the meeting of American Medical Association, Cleveland, June 2-6, 1941; the General Treatment of the Patient with a Severe Burn.[5]

(*a*) One hundred cc. of plasma for every point hematocrit exceeds normal of 45.

(*b*) Fifty cc. of plasma for every point hemoglobin exceeds normal of 100.

(*c*) Fifty cc. of plasma for every 100,000 red count exceeds normal of 5 million.

4. Monogram of Jenkins, Schafer and Owens.[10]

In children the amount of plasma to be given should be proportionately less according to body weight. Furthermore, the calculations do not indicate the amount of plasma that will be needed to manage the entire course of the burn. Just as in a diabetic patient when the blood sugar indicates that 30 units of insulin is necessary, this does not mean that 30 units will suffice for a whole month, so in a serious burn case frequent determinations are essential. Hematocrit readings should be taken as often as every three hours during the first day after a severe burn and the amount of plasma indicated each time given. The danger of burn shock is not over until the thirty-sixth to the forty-eighth hour.

Other measures of possible value in burn shock include:

Sodium Lactate: Recent articles have drawn attention to the possible beneficial effects of sodium lactate in the treatment of burn shock. Rosenthal,[11] experimenting on mice, produced a standard burn by immersing the animals up to the neck in water at 70 C. for a measured period of seconds. A large series of mice was thus burned, and treated groups were compared with untreated controls as to mortality. Sodium chloride by mouth or intraperitoneally caused a significant reduction in mortality. Intravenous administration was less effective. Isotonic solution of sodium chloride by mouth was superior to hypertonic solutions. Sodium acetate, succinate, bicarbonate and lactate were as effective as sodium chloride. Mouse serum injected intravenously was slightly less active than equivalent volumes of 0.9 per cent sodium chloride orally. These results are of great interest especially as they do not agree with the present concept of the superiority of intravenous plasma or serum in the therapy of burn shock. The possibility arises that the results obtained on mice are not entirely applicable to man.

A more recent author, Fox,[12] however, has made the application to man and in a series of 23 severe third degree burns described the clinical use of isotonic solution of sodium lactate administered orally (instead of plasma injected intravenously) in combating burn shock. A careful record of fluid intake was made and an exact schedule used so that approximately 7 to 10 liters (from 10 to 15 per cent of body weight) of the isotonic sodium lactate was administered within the first twenty-four hours. Any vomiting such as occurs frequently in severe burns was treated by administration of more fluid. This work of Fox needs careful consideration, it is so revolutionary in its implications. The results seem good, and the obvious objection that only minor burns were treated is refuted by the charts which indicate extensive third degree burns. On the other hand, adequate studies of plasma volume in these cases were not reported, and the possibility that it may have been dangerously low is to be considered. At the present time sodium lactate would seem to offer promise as an adjuvant to plasma therapy rather than as a substitute for it. A further discussion of Fox's work is included in Dr. Cope's article which appears later in this symposium.

Gelatin: Parkins, Koop, Riegel, Vars and Lockwood[13] studied gelatin solutions as a plasma substitute in experimental burns. These authors found that in reproducible burns in dogs the mortality after gelatin therapy was over three times that which followed plasma therapy.

Adrenal Cortex Extract: Rhoads, Wolff and Lee[14] reported promising results from the use of this substance. They estimated that after a severe burn the capillaries do not regain their normal permeability until the fortieth hour, but after treatment with adrenal cortex extract such normality was attained by the eighteenth hour. A more recent report based on additional cases by Rhoads, Wolff, Saltonstall and Lee[15] reverses this opinion, stating that "the results in this larger series have not fulfilled the promise of the earlier cases. They do not provide a satisfactory basis for the use of adrenal cortical extract in the routine treatment of shock following burns." These clinical observations are corroborated by the experimental studies of Rosenthal,[11] who tested standardized burns in mice and found no protective action whatever by either desoxycorticosterone or adrenal cortex extract injected subcutaneously as compared with controls with saline solution.

Oxygen: This substance has considerable value in burns, especially in those instances in which coincidental pulmonary edema exists. This fact was especially observed in the Cocoanut Grove disaster as shown by Beecher.[16]

Morphine: The recent revised (1943) edition of the pamphlet distributed by the Medical Division of the Office of Civilian Defense[17] states, concerning the use of morphine, that "burned patients usually require large doses of morphine. Following the initial dose of one-half grain, an additional one-fourth grain may be given (if required) as often as every three hours, except in cases of lung damage." In this connection a word of caution is advisable, especially since it is the observation of some of us that burned patients often have relatively little pain after the first half hour. They may moan but when questioned do not always complain of pain. The word of caution comes from observations of Beecher[18] in the North African theater of

10. Jenkins, H. P.; Schafer, P. W., and Owens, F. M., Jr.: Guide to Replacement Therapy for Loss of Blood or Plasma, Arch. Surg. **47**: 1-3 (July) 1943.

11. Rosenthal, S. M.: Experimental Chemotherapy of Burns and Shock: III. Effects of Systemic Therapy on Mortality, Pub. Health Rep. **58**: 513-522 (March 26) 1943.

12. Fox, C. L., Jr.: Oral Sodium Lactate in the Treatment of Burn Shock, J. A. M. A. **124**: 207-212 (Jan. 22) 1944.

13. Parkins, W. M.; Koop, C. E.; Riegel, C.; Vars, H. M., and Lockwood, J. S.: Gelatin as a Plasma Substitute, with Particular Reference to Experimental Hemorrhage and Burn Shock, Ann. Surg. **118**: 193-214 (Aug.) 1943.

14. Rhoads, J. E.; Wolff, W. A., and Lee, W. E.: The Use of Adrenal Cortical Extract in the Treatment of Traumatic Shock of Burns, Ann. Surg. **113**: 955-968 (June) 1941.

15. Rhoads, J. E.; Wolff, W. A.; Saltonstall, H., and Lee, W. E.: Further Experiences with Adrenal Cortical Extract in the Treatment of Burn Shock, Ann. Surg. **118**: 982-987 (Dec.) 1943.

16. Beecher, H. K.: Resuscitation and Sedation of Patients with Burns Which Include the Airway: Some Problems of Immediate Therapy, Ann. Surg. **117**: 825-833 (June) 1943.

17. Treatment of Burns and Prevention of Wound Infections, United States Office of Civilian Defense, Emergency Management Office, April 1943.

18. Beecher, H. K.: Delayed Morphine Poisoning in Battle Casualties, J. A. M. A. **124**: 1193-1194 (April 22) 1944.

operations. Men who were given large doses of morphine when in severe shock showed signs of morphine poisoning (pinpoint pupils and deep respiratory depression) after receiving shock therapy. Beecher stated that "it was not likely that morphine administered subcutaneously under these circumstances would be absorbed. Apparently it was not, for in many cases no pain relief occurred following its use, and the first dose of ½ grain (30 mg.) of morphine would be followed by a second or even a third injection over a period of hours, all of these causing little if any effect. . . . In men in shock, restoration of blood volume and blood pressure, followed eventually by warming, with renewal of the peripheral circulation, led to the seriously rapid absorption of all the morphine injected, and poisoning developed."

Other elements to consider in the early general care of the burned patient are the control of burn toxemia and of burn sepsis. Toxemia still remains somewhat of a mystery. While tannic acid may cause some toxemia, its abandonment has not entirely eliminated burn toxemia. The adequate administration of dextrose and protein and the control of anuria—still an important problem—remain the chief modes of managing toxemia. The management of sepsis is mainly a question of prophylaxis in the original care of the burn wound, but oral administration of sulfonamides and more recently of penicillin, parenteral or local, have much to offer. Tennison [19] has suggested the local use of succinylsulfathiazole, the advantage being its relative nonabsorbability.

Late General Care.—Infection still remains a problem at this stage, but essentially its control depends on maintenance of the patient's nutrition. Anemia, avitaminosis and hypoproteinemia are troublesome and demand continued correction. Taylor, Levenson, Davidson, Browder and Lund [20] have drawn especial attention to this most important aspect of the subject. The use of amino acid solutions is of value in the control of hypoproteinemia.

LOCAL CARE

Early Local Care.—At the present time tannic acid is declining rapidly in popularity as the pressure dressing method of therapy is becoming more widely used. Reasons for the abandonment of tannic acid include the facts that (1) the brittle eschar frequently becomes infected in third degree burns, (2) the difficulty in removing the eschar delays skin grafting, (3) destruction of remaining skin islands delays healing and increases scarring (Cannon and Cope,[21] Hirshfeld, Pilling and Maun [22]) and (4) the absorption of tannic acid leads to focal necrosis of the centers of the liver lobules. This last complication deserves separate mention, as follows:

Liver Necrosis.—The possibility that tannic acid might cause focal central necrosis of the liver was first postulated by Wilson [23] in Great Britain in 1938. This action was shown to be true experimentally by Wells, Humphrey and Coll [24] and later by Forbes and Evans,[25] by Baker and Handler [26] and by Hartman and Romence.[27] Erb, Morgan and Farmer [28] studied the postmortem findings in 61 cases of burns. Of these, 41 were tanned, with 25 (61 per cent) instances of liver necrosis, and 20 were not tanned, with 0 (0 per cent) instances of liver necrosis. Rosenthal,[29] working with mice, found that "significant increases in early mortality were produced by . . . tannic acid solutions and ointment . . . when applied to a scalded area." These observations seem to point rather conclusively to the potential danger of liver necrosis from tannic acid therapy. A closer study of the reported cases and experiments indicates that the tanning methods in which the active agent is kept in contact with the burned surface in a moist state for a long period of time (tannic acid jelly or baths) are especially dangerous. The increased chances for absorption seem to be the influencing factor. Rapid tanning, as by tannic acid-silver nitrate, where the agents dry within a matter of minutes or seconds, is less dangerous in this regard.

Abandonment of tannic acid has not entirely eliminated burn toxemia, and the question still remains whether selected burn cases may not under certain conditions be treated with advantage by this type of therapy. A more complete discussion of this important subject is made by Lee and Rhoads in their article in this symposium.

First Aid Care.—This subject is best summarized by quoting Koch: [30]

> How can we prevent infection? By covering the burned area immediately with clean towels and not exposing it even momentarily until personnel with masked faces and cleansed hands are ready to carry on. The practicability and helpful results of such protection were demonstrated by Churchill and his associates at the Massachusetts General Hospital after the Cocoanut Grove fire. Burned surfaces were covered with sterile towels in the emergency room and kept covered until the patients could be cared for by masked, gowned and gloved personnel.

Early Local Definitive Care.—The use of pressure dressings for wounds and skin grafts is not new, but the application of this method to burns was first made by Koch [3] in 1941. A full description of this important type of management is given in Dr. Koch's chapter in this symposium. In the treatment of the victims of the Cocoanut Grove disaster Cope [31] did not debride or cleanse his burns, whereas Koch's original method advised such cleansing. A discussion of this cogent aspect of the subject is given in Dr. Koch's chapter.

19. Tennison, C. W.: Use of Nonabsorbable Sulfasuxidine in Extensive Burns, Surgery **15:** 332 (Feb.) 1944.
20. Taylor, F. H. L.; Levenson, S. M.; Davidson, C. S.; Browder, N. C., and Lund, C. C.: Problems of Protein Nutrition in Burned Patients, Ann. Surg. **118:** 215-224 (Aug.) 1943.
21. Cannon, B., and Cope, O.: Rate of Epithelial Regeneration, Ann. Surg. **117:** 85-92 (Jan.) 1943.
22. Hirshfeld, J. W.; Pilling, M. A., and Maun, M. E.: A Comparison of the Effects of Tanning Agents and of Vaseline Gauze on Fresh Wounds of Man, Surg., Gynec. & Obst. **76:** 556-561 (May) 1943.
23. Wilson, W. C.: Personal communication to the author, October 1938.
24. Wells, D. B.; Humphrey, H. D., and Coll, J. J.: The Relation of Tannic Acid to the Liver Necrosis Occurring in Burns, New England J. Med. **226:** 629-635 (April 16) 1942.
25. Forbes, J. C., and Evans, E. I.: Tannic Acid and Liver Necrosis, Surg., Gynec. & Obst. **76:** 612-613 (May) 1943.
26. Baker, R. D., and Handler, P.: Animal Experiments with Tannic Acid Suggested by the Tannic Acid Treatment of Burns, Ann. Surg. **118:** 417-426 (Sept.) 1943.
27. Hartman, F. W., and Romence, H. L.: Liver Necrosis in Burns, Ann. Surg. **118:** 402-416 (Sept.) 1943.
28. Erb, I. H.; Morgan, E. M., and Farmer, A. W.: The Pathology of Burns: The Pathological Picture as Revealed at Autopsy in a Series of Sixty-one Fatal Cases Treated at the Hospital for Sick Children, Toronto, Canada, Ann. Surg. **117:** 234-255 (Feb.) 1943.
29. Rosenthal, S. M.: Experimental Chemotherapy of Burns and Shock: I. Methods; II. Effects of Local Therapy upon Mortality from Shock, Pub. Health Rep. **57:** 1923-1935 (Dec. 18) 1942.
30. Koch, S. L., in discussion on paper by Whipple, A. O.: Basic Principles in the Treatment of Thermal Burns, Ann. Surg. **118:** 187-192 (Aug.) 1943.
31. Cope, O.: The Treatment of Surface Burns, Ann. Surg. **117:** 885-893 (June) 1943.

Another treatment that seems to offer some promise is the encasing treatment of burns of the extremities in close fitting plaster of paris casts as advocated by Levenson and Lund.[32] As with the pressure dressing method, the first change of the bandage or cast is not made for ten to fourteen days. The use of plaster casts is based on the experimental work of Glenn, Gilbert and Drinker.[33]

It cannot be emphasized too strongly that local and general care must work conjointly to a common end. Whereas general care is essentially directed toward saving the patient's life and local care toward hastening healing and the prevention of scars and contractures, each should consider the other. In fact, the best local remedies (pressure dressings and the like) go far toward minimizing plasma loss and saving the patient's life, while adequate transfusions of plasma and blood hasten the healing of the local wound.

Late Local Care.—This includes the skin grafting of granulating surfaces and the correction of scars, deformities and contractures. It should be noted that the former (skin grafting of open wounds) must be done as promptly as possible, preferably within the first six weeks after the burn, and usually by the same surgeon and in the same hospital in which the original burn care was given. On the other hand, the latter (correction of scars, deformities and contractures) is best postponed for three to six months and can safely be done by other surgeons, preferably those especially trained in plastic work and possibly thousands of miles from the original scene of the accident.

The recent development of coagulum contact skin grafts by Sano [34] would seem to have little application to the covering of the extensive granulating surfaces that follow severe burns.

CONCLUSIONS

1. In the early control of burn shock, adequate doses of plasma are of especial importance.

2. In the late control of burn anemia and malnutrition, blood transfusions, protein and amino acids are of value.

3. The local first aid treatment of burns should include the application of clean towels until such time as definitive management by aseptic means is available.

4. The application of a sterile compression bandage which is not changed until the tenth to the fourteenth day after the burn is at the present writing the best definitive local management of thermal burns.

5. In all third degree burns the resulting granulating surfaces should be skin grafted as soon as possible. No burn is cured until any existing granulating surfaces are healed.

6. The late plastic correction of scars, deformities and contractures is of importance but in most cases can safely be delayed for three to six months.

32. Levenson, S. M., and Lund, C. C.: The Treatment of Burns of the Extremities with Close Fitting Plaster of Paris Casts, J. A. M. A. **123**: 272-277 (Oct. 2) 1943.

33. Glenn, W. W. L.; Gilbert, H. H., and Drinker, C. K.: The Treatment of Burns by the Closed Plaster Method, with Certain Physiological Considerations Implicit in the Success of This Technique, J. Clin. Investigation **22**: 609-625 (July) 1943.

34. Sano, M. E.: A Coagulum Contact Method of Skin Grafting as Applied to Human Grafts, Surg., Gynec. & Obst. **77**: 510-513 (Nov.) 1943.

THE CHEMICAL ASPECTS OF BURN TREATMENT

OLIVER COPE, M.D.
BOSTON

The rationale of the chemical treatment of burns is based on what is known of their pathologic physiology. In the modern therapy of the burned patient the chemical treatment is important, and a thorough understanding of the pathologic physiology is a requisite to the physician who treats the burned patient with intelligence. In this article are presented current concepts of the nature and consequences of a burn wound and the treatment recommended.

NATURE OF A BURN WOUND

The application of sufficient heat to a body surface to produce a burn sets in motion a sequence of events. The changes which appear in a patient with a burn, mild or severe, within the first hours are manifested as pain, alterations in blood flow, increase in capillary permeability, edema and increased flow in the lymphatic trunks.

Necrosis.—In a severe burn the picture is complicated by cell necrosis and its consequences. The superficial layers of the surface are, of course, the first affected. If the necrosis penetrates the epidermis, this layer may be lifted up by the pressure of the excessive fluid beneath and form the roofs of blebs. As temperature or duration of exposure is increased, the cells of the epidermis coagulate and no bleb can form. In the most intense burn, the cell necrosis and coagulation have penetrated the skin into the underlying tissues; the whole skin is turned into an eschar. No matter how deep the cell necrosis, beneath it there is always an edematous zone of first degree or incomplete burn where the cells are viable.

Pain.—The pain of a burn arises from the sensory nerve endings of the skin and the nerves of the vascular bed and of the deeper tissues. The immediate and severe pain is due to the effect of the heat on the skin, and if the nerves have not been destroyed the pain persists. Intense pain also arises in the damaged vascular bed if the intracapillary pressure is raised. Pain due to distention of the tissues is late and moderate.

Increased Blood Flow.—The alterations in blood flow are due principally to local damage of the vessels. Vascular reflexes, initiated by pain, also play a part. Immediately following the application of heat there is a transitory blanching of the affected surface followed by a flush which slowly extends out beyond the area of damage as a flare. The blanching and flush are due to direct trauma, but the flare is of neurogenic origin.

The flush or capillary dilatation is accompanied by an increased blood flow, the impulse in the artery leading to the burned area being exaggerated. In experimental burns of the feet of dogs, the arterial flow may be doubled and the arteriovenous oxygen difference just proximal to the burned area is decreased to almost zero.[1]

From the Surgical Research Laboratories of the Harvard Medical School and the Surgical Services at the Massachusetts General Hospital.

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Part of the work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.

1. Unpublished data from this laboratory.

Although vascular dilatation is the rule, the pain sometimes causes peripheral vasoconstriction with apparent decrease in blood flow to the burned area:

A patient was seen at this hospital thirty minutes after enduring a steam burn of the low thigh and knee. Blebs had already formed and ruptured. Instead of a flushed, weeping surface, the whole leg was blanched and the exposed surface dry. The foot and leg below the burn were cold and clammy. The patient was suffering intense pain. After the administration of 16 mg. of morphine sulfate the pain gradually subsided. As the patient felt more comfortable, a flush slowly spread over the burned surface and weeping started.[2]

Increased Capillary Permeability.—Capillary permeability in the burned area is increased, even with the mildest trauma, and a protein-rich plasma filtrate pours out into the extracellular spaces. The edema produced involves not only the skin itself but also the subcutaneous tissue spaces, and the volume of the vascular fluid lost into the tissues is greater than would be expected from the visible swelling of the skin. The combined vascular response and edema has been called the triple response by Thomas Lewis.[3]

The heat also increases lymphatic permeability,[4] and, accompanying the formation of edema and rise in interstitial fluid pressure, there is an increased return flow of the capillary filtrate into the blood stream through the lymphatic vessels.[5]

The volume of edema fluid is proportionate to the surface area rather than to the depth of the burn. The other characteristics of edema fluid are a high concentration of plasma protein and a lack of a substance to coagulate this protein.

The clue to the chemical nature of edema fluid is found in the analysis of bleb fluid. In the human being it has an average protein content of 4.0 Gm. per hundred cubic centimeters, the concentration fluctuating slightly with the severity of the burn and decreasing with the healing of the wound.[1] (The figures reported from other laboratories vary slightly.[6]) The proportionate concentration of albumin to globulin is greater than in blood plasma. The concentration of fibrinogen and prothrombin approximates that in blood serum. The diffusible portions of the electrolytes, carbohydrate and nonprotein nitrogenous substances have the same concentration as that in the blood.

The relatively greater proportion of albumin in edema fluid means that the albumin molecule escapes from the damaged capillaries more readily than the globulins. Since the albumin is the molecule in the plasma principally responsible for the osmotic pressure of the blood, its greater loss means a proportionately high loss of osmotic power of the blood.

In spite of the fact that all of the elements of plasma necessary for coagulation have seeped into the edema fluid, coagulation of this fluid rarely takes place in an uninfected burn wound. (The heat coagulates the cellular protein in situ, but the subsequent edema does not coagulate.) A spider web clot may form in a bleb, usually not before forty-eight hours. The fluid aspirated from such a bleb clots in a test tube, proving incomplete coagulation previously. This is also proved by the observations that a clean burn with ruptured blisters continues to weep for several days and that the fluid of an unruptured bleb is slowly resorbed as late as the third to eighth day.

The failure of burn edema fluid to coagulate is in contrast to the rapid coagulation of the protein in interstitial fluid following mechanical trauma sufficient to cause cell injury. Mechanical trauma to cells releases thrombokinase, which activates the enzyme thrombin, which in turn precipitates fibrin from the fibrinogen present in the interstitial fluid.

The failure of a burn wound to clot is presumably due to the absence of thrombokinase. Contact with substances which clot blood also causes bleb fluid to clot. The edema fluid of experimental burns has been shown to coagulate immediately on the injection of a tissue extract known to have a high content of thrombokinase.[7] It is presumed that if thrombokinase itself is present in the normal cell it is thermolabile or, if cells contain only its precursor, the precursor is not activated by heat.

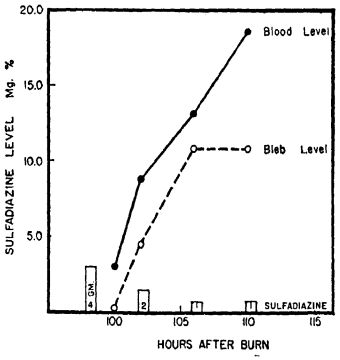

Fig. 1.—Concentration of sulfadiazine in blood and bleb fluid after withholding the drug until the fourth day. The patient had a second degree burn with multiple blebs. Four Gm. of sulfadiazine was given by mouth ninety-eight hours after the burn. The rise in concentration in the bleb fluid is almost as prompt as in the blood stream even at this late time after the burn. Chemotherapy, internally administered, even though delayed, should be effective.

Clinicians have observed the retarded coagulation of the burn wound but have failed to grasp the implications. Water soluble substances placed on the surface of a burn wound with ruptured blebs will be dissolved and absorbed into the general circulation. That sulfonamides applied to the surface are absorbable and may reach toxic levels in the blood is to be anticipated.[8]

The converse is true; substances which permeate the capillary wall, if injected into the general circulation, will be found in the fluid of the burn wound in the same concentration as in the blood plasma. It has been shown that sulfonamides administered intravenously or by mouth are found in the bleb fluid in the same concentration as in the blood plasma.[9]

In figure 1 the rise in sulfonamide concentration in blood plasma and bleb fluid is illustrated in a patient with a second degree burn. There were multiple unruptured blebs. Sulfon-

2. Reflex vasoconstriction on stimulation of the central end of a peripheral nerve, such as the sciatic, is known to experimental physiologists. Such a reflex following a burn decreases the loss of plasma from the capillaries into the burned tissues, thus serving a purpose. Were the effect of pain on the organism limited to this reflex, relief of pain would be contraindicated.

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5. Field, M. E.; Drinker, C. K., and White, J. C.: Lymph Pressures in Sterile Inflammation, J. Exper. Med. **56**: 363-370 (Sept.) 1932. McMasters.[4]

6. Harkins, H. N.: The Treatment of Burns, Springfield, Ill., Charles C Thomas, Publisher, 1942.

7. Glenn, W. W. L.; Peterson, D. K., and Drinker, C. K.: The Flow of Lymph from Burned Tissue, with Particular Reference to the Effects of Fibrin Formation on Lymph Drainage and Composition, Surgery **12**: 685-693 (Nov.) 1942.

8. Hooker, D. H., and Lam, C. R.: Absorption of Sulfanilamide from Burned Surfaces, Surgery **9**: 534-537 (April) 1941.

9. Cope, O.: The Treatment of the Surface Burns, Ann. Surg. **117**: 885-893 (June) 1943.

amides were withheld until ninety-eight hours after the burn, when 4 Gm. of sulfadiazine was given by mouth. The concentration of sulfadiazine measured at two hourly intervals in both blood plasma and bleb fluid showed only a slightly delayed rise in the bleb fluid as compared with blood even at this late time after the burn.

The nature of a burn wound may be altered by infection, a consequence of cell necrosis. When the corneum is ruptured, organisms penetrate freely into the recesses of the wound and infection results. As long as the corneum is intact, even though killed by heat and lifted off the dermis, it offers a formidable barrier to bacteria. This is proved by the observation that the fluid of unruptured blebs is free of virulent organisms.[9]

GENERAL CONSEQUENCES OF LOCAL CHANGES

As a result of the local changes in the wound, the life of the burned patient is endangered. The principal hazards are shock and infection. To these may be added the problem of disposing of the abnormal breakdown products of protein and the surfeit of water, salts and protein resulting from therapy.

Shock.—Shock develops rapidly in an extensively burned patient. Although pain may cause generalized vasomotor collapse, this is transitory and of relatively little clinical importance. The burn shock to be feared is that produced by a decreasing circulating plasma volume. The decrease is a result of the continued loss of plasma through the damaged capillaries into the burn wound.[10] The venous blood returning from the wound has a disproportionately high content of red cells, and eventual generalized hemoconcentration follows. Since the plasma remaining in the blood stream loses more albumin than globulin it becomes increasingly difficult for the circulating blood to withdraw fluid from elsewhere in the body, and increasing concentration of the plasma itself ensues. Not only is the effective plasma volume reduced but the remaining blood is so viscid that its flow is impeded, cardiac output is curtailed, blood pressure falls and an inadequate circulation to normal organs and tissues, as well as to the burn, results.

The most serious consequence of the circulatory failure is the disturbance of organ function. An inadequate supply of oxygen forces some tissues to subsist under anaerobic conditions. Since brain is unable to survive in the absence of oxygen, the body makes an effort to redistribute blood to the higher nervous centers in order to maintain life. This means that other organs, such as the intestinal tract, kidney, liver, and muscle, may be forced to live under almost completely anaerobic conditions.

Muscle is able to survive anaerobically, but the products of its metabolism differ from those in the presence of oxygen. Dextrose instead of being broken down completely to carbon dioxide and water is metabolized only as far as lactic and pyruvic acids.[11] Since the failing circulation is unable to remove the waste products of metabolism, these fixed acids accumulate in the muscle, and an alteration in electrolyte balance or acidosis results.

The liver suffers under conditions of a failing circulation, for it is dependent for part of its oxygen on blood which has already supplied another organ. The liver derives its oxygen from the portal vein blood as well as from that of the hepatic artery. With a drop in cardiac output, the blood flow to the intestinal tract is one of the first reduced by the body's compensating mechanism. The intestinal tract removes all of the oxygen it can from the blood passing through it, with the result that by the time the blood reaches the liver it contains but little oxygen. In the absence of oxygen the liver cannot perform certain of its functions, deamination and glycogen production.[12] Amino acids, a normal breakdown product of protein metabolism, accumulate in the blood. The glycogen store is rapidly depleted and resynthesis prevented.

The kidney is unable to perform its function of urine formation without an adequate blood flow. Waste products accumulate, and its compensating electrolyte selectivity is lost. Normally, if fixed acids appear in excess in the blood, the kidney excretes them by excluding the volatile carbonic acid and using ammonia.

Burn shock is accompanied by dehydration and electrolyte imbalance. Dehydration of the unburned tissues of the body continues slowly so long as hemoconcentration is present. The blood withdraws fluid and electrolytes from the interstitial spaces but, as this source is drained, intracellular fluid in turn is tapped. With the passage of water from cells to extracellular spaces, electrolytes also must pass in order to maintain isotonicity on both sides of the cell membrane. Sodium and chloride are the predominant ions of the extracellular fluid, while potassium and phosphate are their counterparts within the cells. With dehydration, therefore, the levels of phosphate and potassium rise in interstitial fluid and blood. As dehydration continues, cell metabolism is disturbed and, with the progression of burn shock, cells die. It is not proved whether it is the accumulation of phosphate and potassium in abnormal concentrations in the interstitial fluid that poisons the cells or whether it is the lack of adequate fluid and electrolyte within.

The pattern of the electrolyte imbalance encountered in untreated shock, as the consequence of prolonged inadequate circulation, is determined by the relative contribution of each of its three causes. The three causes are dehydration, anaerobic metabolism and absent kidney function. The pattern may be further altered by therapy and infection.

Infection.—The growth of infective organisms in a burn wound not only increases destruction of viable tissue and delays cellular regeneration but also fosters a generalized toxemia and malnutrition.

The degrees of infection and shock in the burned patient are closely interwoven. Infectious toxemia abets the shock due to loss of an effective blood volume, and it is probable that the worse the shock the more rapid the growth of organisms. Many of the virulent organisms, such as certain strains of streptococci, proliferate more rapidly under partially anaerobic conditions. Since in shock there is a shortage of oxygen in the burn wound, such organisms flourish and bacterial toxemia threatens in a few hours.

Infection adds to the nutritional load of the burned patient. There is some loss of nitrogen even with a moderate burn. In patients with deep, widespread, infected burns there is wasting accompanied by large

10. Blalock, A.: Experimental Shock: VII. The Importance of the Local Loss of Fluid in the Production of the Low Blood Pressure After Burns, Arch. Surg. **22**: 610-616 (April) 1931. Harkins, H. N.: Experimental Burns: I. The Rate of Fluid Shift and Its Relation to the Onset of Shock in Severe Burns, ibid. **31**: 71-85 (July) 1935.

11. Lipmann, F.: Pasteur Effect, in A Symposium on Respiratory Enzymes, Madison, University of Wisconsin Press, 1942.

12. Engel, F. L.; Harrison, H. C., and Long, C. N. H.: Biochemical Studies on Shock: III. The Role of the Liver and the Hepatic Circulation in the Metabolic Changes During Hemorrhagic Shock in the Rat and the Cat, J. Exper. Med. **79**: 9-22 (Jan.) 1944.

losses of nitrogen. Protein is discharged from the wound and excreted as breakdown products through the kidney. If the infection is not controlled, cachexia develops. Anemia may also result from infection, and rapidly if the toxemia is severe.

Late Resorption of Edema Fluid.—The burn wound increases the capacity of the extracellular fluid reservoir, and this reservoir must be kept filled by the addition of plasma protein, electrolyte and water in order to maintain bodily function. At the height of wound edema, therefore, there will be within the extracellular spaces a superabundance of vascular protein and fluid. When the lymphatic resorption of fluid from the wound exceeds the leakage into the wound and edema resorption has begun, the body is faced with the problem of eliminating the extra amount of plasma protein and fluid in order to prevent an excess plasma volume. In a burn of small area the excess protein and fluid offers no problem, since the protein can be either stored or metabolized and the fluid excreted by the kidney. In an extensive burn an amount of plasma and fluid will have been given which may prove unmanageable during the period of resorption.

The edema of the burn wound usually reaches its maximum within the first thirty-six to forty-eight hours and then begins to subside. By the end of five to seven days, if there is no active infection, the edema has almost entirely disappeared. It is after the third day, therefore, that the patient may develop an increased plasma volume and pulmonary edema. With an excessive plasma volume the hematocrit falls. With the destruction of red cells by infectious toxemia there is also a fall in hematocrit. From the point of view of therapy the two must be differentiated.

Elimination of Abnormal Protein.—Heat denatures protein, and the elimination of such protein may prove a burden to the kidney. Abnormal protein is created in the cells necrotized by the heat and in the surrounding blood and fluid. Some of the tissue proteins are eliminated as slough from the burn wound. The blood proteins are resorbed into the circulation and have to be either metabolized or excreted.

Hemolysis is an accompaniment of severe burns and is believed to be the result of the exposure of the red cells to heat. In experimental animals, hemolysis is encountered following a boiling water burn of the leg only if the exposure exceeds twenty seconds.[1] Shen and Ham[13] have noted increased fragility of red cells following burns.

The kidney may find it difficult to eliminate hemoglobin. Massive hemoglobinuria was encountered in a number of the patients of the Cocoanut Grove fire. In some no residual disturbance of kidney function resulted.[14] In others the hemoglobinuria was associated with decreased elimination of other substances.[13] The maintenance of adequate blood flow to the kidney by the prevention of shock is probably the deciding factor in overcoming the load of hemoglobinuria. In the cases without residual kidney damage the sodium bicarbonate therapy accompanying the sulfonamide therapy may have played a preventive role.

Albuminuria is unpredictable; it does not necessarily occur in even extensively burned patients.[14] The precipitable protein is eliminated by the kidney, presumably because it is an abnormal protein. It may be abnormal as a result of infection as much as of the burn.

13. Shen, S. C., and Ham, T. H.: Studies on the Destruction of Red Blood Cells: III. Mechanism and Complications of Hemoglobinuria in Patients with Thermal Burns; Spherocytosis and Increased Osmotic Fragility of Red Blood Cells, New England J. Med. **229**: 701-713 (Nov. 4) 1943.

14. Cope, O., and Rhinelander, F. W.: The Problem of Burn Shock Complicated by Pulmonary Damage, Ann. Surg. **117**: 915-928 (June) 1943.

CHEMICAL TREATMENT

The chemical treatment of the burned patient is directed toward the control of shock and infection and the production of the optimal condition of nutrition for wound healing.

Shock.—The ideal chemical treatment of burn shock would be the prevention of shock by an agent which would remedy the abnormal capillary permeability. Such treatment would prevent the excessive loss of plasma from the circulation. No chemical agent is known which will do this. It has been thought that adrenal cortex extract and desoxycorticosterone acetate were such agents, but on analysis these have been found to be ineffectual.[15] Posterior pituitary has been suggested, since it causes arterial constriction and reduction in blood flow. Theoretically, therefore, it should diminish the plasma loss but it has side-effects, the most undesirable of which is constriction of the coronary arteries, and cannot be recommended.

It is possible, although not by a chemical agent, to reduce the plasma loss by the application of cold or pressure. Cold produces its effect by causing reflex arteriolar constriction and reduction in blood flow. It has limited therapeutic use, since, if it is applied for any length of time or at too low a temperature, frostbite will result. If too extensive an area of the body is chilled the body temperature will fall. Yet the application of cold nonetheless for a burn of a single extremity should be resorted to when other measures for the relief of shock (and pain) are not available.

A plaster bandage or elastic pressure dressing applied to the head or to an extremity diminishes the plasma filtration from the capillaries by raising the interstitial fluid pressure. If the burn is deep, however, the escaped plasma may be forced through the tissues and appear as edema proximal to the dressing. The dressings must be applied with graded pressure, the greatest pressure being at the most distal portion. A constricting band proximally will result in occlusion of blood flow as edema develops, and gangrene will result.

Replacement of Lost Plasma Volume.—It is beyond our control to affect the concentration of water, electrolyte and plasma protein within the wound, and the chemical treatment of shock resolves itself into maintaining normal concentrations within the blood stream and unburned interstitial spaces and the administration of the fluid and foods which the body requires for kidney function and normal metabolism. Since the leakage of plasma starts immediately after the burn and since efforts should be directed toward the prevention of shock, the replenishment of plasma volume is to be started as soon as possible and to be continued during the period of increasing edema.

Theoretically the best solution to inject into the blood stream to replace the plasma is a solution chemically identical to that being lost. Such a solution can be made by diluting human plasma with an equal volume of isotonic solution of sodium chloride and adding sufficient albumin concentrate to raise the protein content to 4.0 Gm. per hundred cubic centimeters.

There are reasons which make it advisable to inject into the blood stream the most concentrated solution of plasma protein possible. A solution of high colloid osmotic pressure should retard the leakage through the

15. Cope, O., and Moore, F. D.: A Study of Capillary Permeability in Experimental Burns and Burn Shock Using Radioactive Dyes in Blood and Lymph, J. Clin. Investigation **23**: 241-257 (March) 1944. Rhoads, J. E.; Wolff, W. A.; Saltonstall, H., and Lee, W. E.: Further Experiences with Adrenal Cortical Extract in the Treatment of Burn Shock, Ann. Surg. **118**: 982-987 (Dec.) 1943. Cope and Rhinelander.[14]

capillaries. This is particularly important if there is pulmonary damage.[14] When a high concentration of protein is used, a smaller volume has to be injected intravenously. The remainder of the required fluid can be given by mouth, and this will prove more satisfactory to the patient. Whole plasma, therefore, should be injected rather than dilute plasma. There is one objection to giving full strength plasma; it flows slowly through a needle and if the patient is in shock it is imperative that the plasma protein replacement be expedited and pumping with a syringe may be required.

In the estimation of the volume of plasma to be replaced, calculate in terms of undiluted plasma. Since the plasma required is proportionate to the area of the burn, the surface area formula developed by Berkow and applied to burn plasma dosage by Harkins[16] is logical. For each 10 per cent of the body surface burned, 500 cc., or 2 units, of plasma should be given over the first twenty-four hours. In the second twenty-four hours approximately half of this amount will be required. When the patient is seen within the first half hour after the burn there will be little hemoconcentration to combat, and two thirds of the plasma should be injected in the first twelve hours and the remainder in the second twelve hours.

When the patient is not seen until shock is imminent and hemoconcentration is advanced, the plasma must be injected rapidly until hemoconcentration is virtually relieved. The remaining volume of the twenty-four hour requirement may have to be supplemented. If a hematocrit reading can be immediately obtained it is more efficient in this patient to calculate the initial requirement by using the formula of Harkins.[17] For each per cent that the hematocrit is above the normal of 45, 50 cc. of plasma should be administered. Subsequent plasma should be injected on the basis of the surface area formula in order to anticipate the continued loss.

Plasma is more economically used, according to Rhoads,[16] if some degree of hemoconcentration is allowed. In the patient with a burn of small area an excess of plasma will do no harm unless there is a concomitant pulmonary injury.[14] In the extensively burned patient, sparing use is advisable in order to avoid overburdening the body with plasma protein during the period of edema resorption.

Repeated hematocrit readings should be made in order to judge the success of the plasma therapy. Although the size of the surface area burned is the safest guide, the portion of the body affected and the depth of the burn must be taken into consideration.[14]

All figures quoted are for an adult of average size and should be adjusted for children or for small or large adults.

Blood bank and reconstituted plasma are equally good in the treatment of shock from the point of view of protein replacement. Reconstituted plasma unfortunately contains merthiolate as a preservative and, if more than 8 units is needed, blood bank plasma should be used in order to avoid mercurial damage to the kidneys.

Besides giving the calculated amount of undiluted plasma, saline solution to complete the replenishment of the plasma volume, water for kidney function and food for metabolism must be given. The foods required are described in the last section of this paper. The volume of saline solution required is equal to that of the plasma injected intravenously. The saline solution must be given subcutaneously if it cannot be taken by mouth, otherwise dehydration and salt depletion will take place. If the patient is given nothing but isotonic electrolyte solution, the kidney is forced to concentrate the urine beyond normal. Water is needed, and 1,500 cc. should be given in the first twenty-four hours and 1,000 cc. in the second twenty-four hours. If the patient is thirsty on this quantity of water, it is judged that an insufficient volume of saline solution has been given. During the period of resorption of edema fluid, water should be given moderately and cautiously.[18]

All of the requirements, other than plasma protein, should be given by mouth when the patient is not nauseated or burned in the pharynx. The entire intestinal tract can act as a reservoir storing water, electrolyte and foods until the extracellular fluid or kidney needs them. When dehydration exists, water and the appropriate electrolytes are absorbed; the reservoir is able to hold nonprotein nitrogen, phosphate and potassium which have piled up in excessive concentrations in interstitial fluid and plasma. If the patient is normally hydrated a moderate excess of intake does no harm, since the reservoir increases its capacity. This capacity is not unlimited, however, and a large excess of fluid is to be avoided, particularly during the period of edema resorption.

If plasma either in the frozen blood bank or in the dehydrated form is not available for the treatment of burn shock, there are substitutes in other forms of blood, colloid or electrolyte solutions. The two substitutes for plasma derived from blood are whole blood itself and albumin concentrate, and these rank as second choice. They have different advantages; they are best used in combination and with saline solution. Whole blood supplies complete plasma. It has the disadvantage of adding cells and is therefore less effective in reducing hemoconcentration. When a large volume of plasma has to be replaced, a corresponding excess of red cells will be added to the circulation. This will mean an increase in blood volume. It is not known how many of these extra red cells the spleen is able to remove. The number of whole blood transfusions, therefore, that can be given in the early hours after a burn and during the period of maximum plasma loss should probably be limited to 5 pints for the adult of average size.

The administration of albumin concentrate,[19] like that of plasma, is not accompanied by the formation of an excessive cell volume. Replenishment of the plasma ensues, but there is a distortion of the protein balance of the plasma since the single protein, albumin, is replacing both albumin and globulin.

The combination of whole blood and albumin concentrate in equal volumes of plasma equivalent is better than either alone. Fifty cc. of the 25 per cent solution of albumin diluted to 250 cc. with isotonic solution of sodium chloride is the osmotic or plasma equivalent of 500 cc. of whole blood. With the blood and albumin solution, an equal volume of saline solution should be given by mouth.

16. National Research Council Conference on Burns, Jan. 7, 1942.
17. Harkins, H. N.; Lam, C. R., and Romence, H.: Plasma Therapy in Severe Burns, Surg., Gynec. & Obst. **75**: 410-420 (Oct.) 1942.

18. D. W. Richards Jr. (personal communication) has obtained evidence of increased plasma volume and venous pressure in addition to signs of pulmonary congestion between the third and fifth days after the burn in patients who have received large quantities of parenteral fluid therapy beyond the third day. Two patients died; in the treatment of 2 other patients parenteral fluids were discontinued, diuresis occurred and the patients recovered.
19. Heyl, J. T.; Gibson, J. G., 2d, and Janeway, C. A.: Studies on the Plasma Proteins: V. The Effect of Concentrated Solutions of Human and Bovine Serum Albumin on Blood Volume After Acute Blood Loss in Man, J. Clin. Investigation **22**: 763-773 (Nov.) 1943. The human and bovine plasma albumin concentrates are not yet commercially available.

The noncolloid solutions, water, salt and dextrose, in large volumes have made possible the survival of many severely burned patients.[20] When no colloid solution is available, any one or a combination of noncolloid solutions should be given freely. A noncolloid solution is able to relieve dehydration by rebuilding the plasma volume and filling the extended interstitial space (fig. 2). With the improvement in the circulation the rate of edema formation, however, is accelerated because the proteins of the plasma are diluted. The acceleration is not as deleterious as would be expected, since it is compensated for by a decrease in the protein concentration of the edema fluid.[21]

Each noncolloid solution has its own peculiar effect beyond its dilution of plasma protein. Water fills the enlarged extracellular fluid reservoir but dilutes the electrolytes (fig. 2, diagram *C*). (Water as a substitute for plasma in the treatment of burn shock must be differentiated from that given in addition to plasma-saline therapy to maintain kidney function.) The dilution of calcium, magnesium, phosphate and potassium is readily compensated for because there is a store of these electrolytes within the cells. There is no storehouse for sodium and chloride, and in an extensively burned patient given water the levels will be dangerously low.

It is erroneous in the therapy of burn shock to urge the patient being treated to drink water freely with the idea that it will take the place of plasma and saline solution. Even if the patient is given plasma intravenously, a large intake of water without salt may result in electrolyte dilution. The following case illustrates such dilution:

A woman aged 29 entered the hospital with second and third degree flame burns of the entire face, head, neck, arms, shoulders and back and the sternal portion of the chest (34 per cent in total). She was given 750 cc. of blood bank plasma plus 750 cc. of isotonic solution of sodium chloride intravenously in the first twelve hours, in spite of which her hematocrit reading rose to 71 per cent. She was given a similar quantity of plasma and saline solution intravenously in the second twelve hours. Throughout this period she imbibed 4,500 cc. of water without salt. At the end of twenty-four hours her hematocrit reading had dropped only to 59 per cent but her plasma already showed dilution. On entry, one hour after the burn, the plasma protein was 7.4 Gm., total base 154 milliequivalents per liter and chloride 103 milliequivalents per liter, all within the normal range. Twenty-four hours later the values were respectively 6.2 Gm., 140 milliequivalents per liter and 87 milliequivalents per liter. The protein is still within the normal range but the electrolytes have been diluted to below normal. The required sodium and chloride were not given, so that the total base remained low at 140 for the next ten days. The sodium on the fourth day was 131 milliequivalents per liter and on the sixth was 114 milliequivalents per liter; the chloride slowly fell, so that on the eleventh day it was only 77 milliequivalents per liter. This continued dilution was presumably influenced by the fact that the burns became infected and some edema of the wounds persisted. Finally on the eleventh day a blood transfusion and isotonic solution of sodium chloride were given intravenously. There was a spontaneous remission of the infectious process and the electrolyte pattern returned to normal. This patient received inadequate therapy both of protein and of electrolyte, but particularly of the latter. With the development of dehydration and thirst, the patient filled with water the extracellular reservoir enlarged by the extensive wounds. Sodium and chloride should have been added to the fluids she drank.

The defect of salt dilution from treating with water is remedied by giving salt solution (fig. 2, diagram *D*).

Dextrose is not an effective colloid. It is, however, an important metabolyte and should be added to the saline solution or water. The eventual distribution of electrolytes and protein will depend on the solution in which it is given.

Isotonic sodium lactate is recommended by Fox[22] for the treatment of burn shock. Such therapy recog-

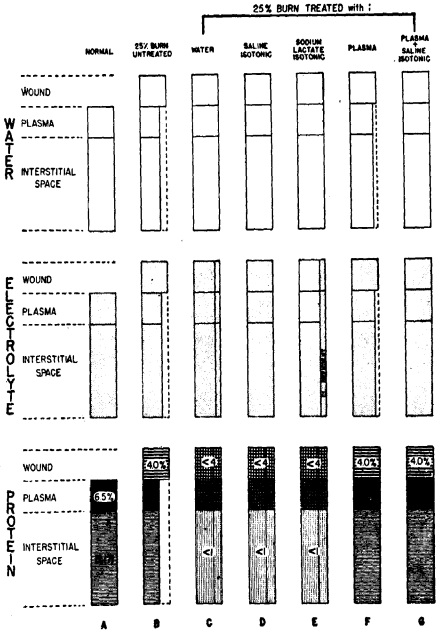

Fig. 2.—Hypothetic distribution of extracellular water, electrolyte and protein in the burned patient. The figures depict no single patient but are a composite of the data of many patients and experimental observations. The diagrams are based on the assumption that 25 per cent of the body surface is burned. The calculations of Elkinton, Wolff and Lee (Plasma Transfusion in the Treatment of the Fluid Shift in Severe Burns, Ann. Surg. **112**: 150 [July] 1940) suggest that similar changes are produced by a burn of less extent. The diagrams are drawn with the supposition that nothing is lost from the wound or kidneys, that nothing is withdrawn from the intracellular space and that the optimal amount of therapy is given in each instance. As the wound swells with edema, the body's capacity for water, electrolyte and plasma protein expands. Degrees of shading under electrolyte and protein indicate different concentrations. Diagram *A*, distribution of extracellular water, electrolyte and protein between blood plasma and interstitial space in the normal. The plasma volume approximates 25 per cent of the extracellular fluid. The average concentration of protein of the interstitial fluid is estimated as 1 per cent; it has not been measured in the human being. Diagram *B*, effect of the burn, untreated. As plasma leaks into the wound, both the plasma and interstitial fluid volume of the unburned tissues are diminished. The capacity of the interstitial space created in the wound in this hypothetic case is equal to the normal plasma volume. The edema fluid is drawn as having a protein concentration of 4.0 per cent. Diagram *C*, effect of treating the burned patient with water. The entire fluid reservoir, the vascular system, the unburned interstitial space and the wound; is filled. The concentrations of electrolyte and protein are reduced throughout. Diagram *D*, effect of treating with isotonic solution of sodium chloride. The entire reservoir is filled with water and there is an approximately normal concentration of electrolyte. The concentration of sodium is normal, that of the chloride above normal. Protein concentration is reduced. Diagram *E*, effect of isotonic sodium lactate solution. The reservoir is filled. The concentration of sodium is physiologic. The concentrations of chloride and protein are reduced. Diagram *F*, the effect of returning to the circulation in the form of plasma that quantity of protein which has been lost into the burn wound. There is a deficiency of both water and electrolyte in the reservoir. Only half of the deficiency of water and electrolyte has been met by the plasma. Diagram *G*, the result of combined plasma and saline therapy. The reservoir is filled with fluid. The concentrations of electrolyte and protein in the plasma and unburned tissue space are normal.

nizes the benefit to be derived from giving an adequate amount of water and sodium. It does not take into account the loss of plasma protein into the burn

20. Commercially available amino acid solutions which are useful in nutritional failure late in burns should not be used as an electrolyte substitute in burn shock, since in shock there is already an accumulation of unmetabolized amino acids. As far as the author is aware their effect has not been weighed in experimental burn shock.

21. Harkins (personal communication) and the author believe that acceleration of edema formation occurs but it is admittedly difficult to prove clinically. In the experimental animal adequate control is possible, and it has been demonstrated (unpublished data from this laboratory).

22. Fox, C. L., Jr.: Oral Sodium Lactate in the Treatment of Burn Shock, J. A. M. A. **124**: 207-212 (Jan. 22) 1944.

wound on which the circulation is dependent for its colloid osmotic and effective circulating pressure. Fox's patient W. R. had a systolic blood pressure of 70 mm. of mercury, a reading consistent with shock, for eight hours after the onset of sodium lactate therapy. The patient also had a plasma protein level of 4.2 Gm. at thirty-six hours after the burn, a level consistent with extreme plasma dilution.

The chloride deficiency which is concomitant with that of sodium is also not remedied by calcium lactate therapy. As could have been anticipated, the chloride level of Fox's patient descended to 77 milliequivalents per liter at thirty-six hours. It is true that chloride deficiency will be only two thirds that of sodium and that the administration of sodium as sodium chloride solution introduces an excess of chloride. Nevertheless it is probably wiser to give the excess of chloride than none at all. It is still to be proved that lactic acid is the physiologic equivalent of chloride in meeting the chloride deficiency. It is understood that all of the chloride given as therapy will have to be excreted by the kidney when the edema of the burn wound is resorbed, but so will the 50 per cent of the lactic acid which cannot be metabolized. The entire amount of sodium given will also have to be excreted, and there is no evidence that the kidney can excrete sodium lactate more easily than sodium chloride. Another acid, phosphate, ketone or carbonate, will have to be mobilized to balance the sodium relinquished by the lactate which is metabolized.

Sodium lactate solution should effectively combat the acidosis of late shock and promote the excretion of hemoglobin by the kidney. Half of the lactic acid is metabolized, freeing the sodium to neutralize the acidosis and also to alkalize the urine.

Solutions of three nonbiologic colloids, acacia, pectin and gelatin, have been introduced as plasma substitutes for the treatment of burn shock. Body cells retain acacia for many years, and damage from its inclusion is not excluded. Pectin [23] and gelatin [24] solutions satisfactorily restore the osmotic tension lost through plasma leakage. Neither of these two colloid solutions has had adequate clinical trial; both may prove easier to eliminate than plasma protein during the period of edema resorption, in which case their use would be preferable in the extensively burned patient.

Correction of Abnormal Metabolism Induced by Shock.—Prolonged low blood pressure shock with its inadequate supply of oxygen to tissues results in the collection of products of abnormal metabolism. There will be a rise in nonprotein nitrogen, including amino acid, and an electrolyte acidosis induced by an excess of fixed acids, ketone, lactic and pyruvic. The cessation of kidney function increases the concentration of these abnormal metabolites. Solutions containing the sodium ion are indicated for the immediate relief of the acidosis. This will result in a total excess of sodium ion within the body which must eventually be excreted. The amino acids will be metabolized when shock has been relieved and aerobic conditions exist in the liver. Above all, fluid is needed for kidney function. It is believed that the sodium ion given as sodium bicarbonate will promote kidney function in the presence of acidosis. Dextrose intravenously is also helpful in initiating a resumption of kidney function, but again the prime consideration is the relief of shock and the return of an adequate blood flow to the kidney. It is held that vitamin B, particularly thiamine, should be given in shock, since the B group of vitamins are rapidly dissipated and since they are a requisite for the normal carbohydrate metabolism of cells.

CONTROL OF INFECTION

It is to be assumed that all burn wounds are contaminated and therefore may become infected. Immediate covering of the wound and prophylactic chemotherapy are indicated. Since the organisms infecting a burn arrive as contaminants on the wound surface, it is imperative to protect the wound at once by applying an impervious dressing.[9] Chemotherapy, either by mouth or intravenously, is indicated for all burn patients except those with first degree or other burns of small extent in which the epidermis is intact. It may be withheld in a second degree burn in which it is anticipated that the blisters will not rupture.

No one or a combination of chemotherapeutic agents attack all the types of organisms known to infect burn wounds. Penicillin promises more than the sulfonamides, since it controls the staphylococcus as well as the organisms susceptible to sulfonamides. It is hoped that this drug will soon be available in greater quantities. In the meantime, sulfadiazine is the drug of choice. In severely burned patients with ruptured epidermis, the drug should be started early by the intravenous injection of 2.5 Gm. of the sodium salt. Subsequent doses should be given either intravenously or by mouth in quantities adequate to maintain a blood level above 6 mg. per hundred cubic centimeters and below 12 mg. per hundred cubic centimeters. It is probable that sodium bicarbonate or citrate should be concomitantly administered by mouth. Such chemotherapy should be continued until the wound is covered with epidermis firm enough to bar the entrance of organisms. If signs of a toxic reaction to sulfadiazine appear, the drug should be promptly omitted.

Many of the ointments currently recommended for the treatment of burns contain sulfonamides. The drug is absorbed from such preparations, and, if the ointment is applied over an extensive area, toxic levels may be reached.[8] It is easier to control the level of the drug by internal administration alone, and the drug permeates the wound in effective concentrations.

Chemotherapeutic agents other than sulfonamides and penicillin, such as the gentian violet and triple dyes, have been recommended for local use on burn wounds. These dyes at bacteriostatic concentrations are toxic to tissues and therefore delay wound healing.[25]

If prophylactic chemotherapy was not instituted and the burn is already infected when first seen, chemotherapy is indicated together with the general surgical procedures used for established infection.

Specific immune therapy may be indicated for tetanus and streptococcic infections. A small percentage of burned patients develop tetanus. All patients with burns in which the epidermis is broken should be given a prophylactic dose of tetanus antitoxin or a booster dose of tetanus toxoid if the patient has been previously immunized. If a streptococcic infection develops in a

23. Hartman, F. W.; Schelling, V.; Harkins, H. N., and Brush, B.: Pectin Solution as a Blood Substitute, Ann. Surg. **114**: 212-225 (Aug.) 1941.

24. Parkins, W. M.; Koop, C. E.; Riegel, C.; Vars, H. M., and Lockwood, J. S.: Gelatin as a Plasma Substitute, with Particular Reference to Experimental Hemorrhage and Burn Shock, Ann. Surg. **118**: 193-214 (Aug.) 1943.

25. Cannon, B., and Cope, O.: Rate of Epithelial Regeneration: A Clinical Method of Measurement, and the Effect of Various Agents Recommended in the Treatment of Burns, Ann. Surg. **117**: 85-92 (Jan.) 1943.

patient sensitive to sulfonamides and for whom penicillin is not available, immunotransfusion [26] should be considered.

A sound physiologic state of the body and careful attention to surgical detail may decide the fight against infection. The importance of combating shock and anoxia has been mentioned. Proper nutrition and fluid balance are necessary for the elaboration of protein immune bodies and the destruction of bacterial toxins. It is impressive how immobilization of the wound by a firm splint dressing and the avoidance of contamination with new organisms by dressing the wound only in the operating room decelerates the infectious process.

PROMOTION OF WOUND HEALING

No chemical agent is known which expedites wound healing above the physiologic optimum pertaining under the state of normal nutrition and the absence of infection. Substances such as Biodyne, vitamin ointments and light filters are foisted on us under the mistaken impression that they accelerate cellular regeneration. Critical analysis does not confirm the claims. It is possible that a principle will be discovered which will enable cells to multiply at a rate above that of normal tissue. In the meantime the substance to be applied locally to wounds is one which is known to be noninjurious to tissues.

General hygienic measures must be undertaken to bring the disturbed nutritional state of the burned patient back to normal. Whole blood transfusions for anemia, a high protein high vitamin palatable diet, together with adequate sleep, relief of anxiety and proper intestinal function are the measures.

Repeated whole blood transfusions are needed in the treatment of severely burned patients with infected wounds. Infectious toxemia is accompanied by a decrease in red cell volume and oxygen carrying capacity of the blood. The blood hematocrit gives the clue to the extent. The fall in hematocrit due to the decrease in number of red cells must be differentiated from that due to the hemodilution which occurs during the period of resorption of edema fluid. Whole blood transfusions are indicated in the former condition but may be hazardous in the latter, since the plasma volume may already be increased and transfusion may precipitate pulmonary edema. In both cases the oxygen carrying capacity of the blood must be built up to permit adequate kidney function. Rigid restriction of fluid intake, rather than transfusion, is indicated in the pseudo anemia of hemodilution.

There may not be an adequate supply of iron available within the body, and therapy is therefore indicated for the reconstruction of hemoglobin.

Infection results in rapid destruction of plasma protein as well as a decrease in circulating hemoglobin. A high intake of protein in the diet is therefore indicated from the beginning. The transfusions given for the anemia will augment the diet. The parenteral administration of amino acids is economical but probably not as efficient as transfusions.

SUMMARY

The chemical treatment of burns encompasses shock, its prevention and metabolic consequences, the control of infection, the promotion of wound healing and the maintenance of nutrition. The rationale for the therapy of each is dependent on a knowledge of the pathologic physiology of the burn wound.

26. Lyons, C.: Immunotransfusion and Antitoxin Therapy in Hemolytic Streptococcus Infections, J. A. M. A. **105**: 1972-1975 (Dec. 14) 1935.

The wound enlarges the body's reservoir for water, electrolyte and protein. All three should be given in the treatment of shock and in amounts sufficient to restore their normal concentrations. When water only is given, the electrolyte and protein in the plasma and interstitial space become dilute, but water alone is better than no treatment at all. Water and salt will fill the reservoir, but only when plasma protein is given in conjunction does the plasma volume possess an adequate colloid osmotic pressure.

Even more favorable results than those obtained with sulfadiazine in the control of infection are to be anticipated from the prophylactic use of penicillin.

No substance is known which expedites the healing of the wound above that obtained under conditions of normal nutrition and absence of infection. The optimal care of the burn surface consists in covering it promptly with a dressing impervious to bacteria and one which contains nothing injurious to cells.

THE GENERAL CARE OF THE BURNED PATIENT

CONRAD R. LAM, M.D.
DETROIT

It is easier to write an article on the general treatment of a patient with burns now than it was two or three years ago. Much controversial matter appears to have been settled, and certain previously recommended procedures can be omitted or will be mentioned only to be condemned. There has been a healthy change in the attitude of surgeons toward thermal injury; the trend has been toward simplification and uniformity, with the burn being regarded as a wound which will react favorably to many of the fundamental surgical methods which are commonly used in other kinds of trauma.[1] If this point of view in local treatment is extended to include general care, one may consider that the burned patient differs in only a few respects from patients with other surgical conditions, and this difference is one of degree only. I wish to stress especially that the care of burns has become vastly easier since the abandonment of tanning methods and the adoption of the large occlusive dressings described elsewhere in this symposium. For example, the stage of liver insufficiency and "toxemia" which so often ended with death on the third to the fifth day is unknown in nontanned patients.

General treatment will be considered under the following headings: (1) management of shock and certain shocklike symptoms, (2) nutrition and fluid balance, (3) general measures to combat infection and (4) nursing care.

SHOCK AND SHOCKLIKE SYMPTOMS

The term "burn shock" has been used extensively, and this may have led to an erroneous conception in the minds of some that there is a special kind of shock in burns which should respond to specific treatment in the form of the injection of this or that drug parenterally. The number of recommended substances and

From the Division of General Surgery of the Henry Ford Hospital.
This paper, in a symposium on "The Treatment of Burns," is published under the auspices of the Section on Surgery, General and Abdominal.
Many of the conclusions and the 2 case reports are based on work done in conjunction with Roy D. McClure and under a contract recommended by the Committee on Medical Research between the Office of Scientific Research and Development and the Henry Ford Hospital.

1. McClure, R. D.: The Modern Treatment of Burns, Surg., Gynec. & Obst. **66**: 1064 (June) 1938. Whipple, A. O.: Basic Principles in the Treatment of Thermal Burns, Ann. Surg. **118**: 187 (Aug.) 1943.

the enthusiasm of the investigators who work with them has been augmented by two conditions: (1) the apparent inclination of experimental animals, especially rats, to fare statistically better than controls when treated with any method which is being tested and (2) the stubborn tendency to recover in all cases in man, regardless of the type of treatment. The burned patient may show some symptoms commonly referred to as "shock," but it is unnecessary to look for some hypothetical substance, e. g. a histaminoid, to explain them. Probably the cause can be found in the simple outline given in the accompanying table, which was suggested by Norman E. Freeman and published by Lee.[2] I have changed the order of the items to conform to the sequence in which they should be considered for burns and have added the item of plasma loss.

This outline might be consulted with profit in the symptomatic treatment of the patient with acute burns. Pain is first on the list and justly so. It is the duty of the medical attendant to relieve this symptom adequately and rapidly. Morphine remains the drug of choice. There is some evidence that an excessively large dose may not alleviate pain more than a moderate one, while the former carries with it the danger of respiratory depression. Above all, it is essential that one does not try to treat restlessness or mania due to anoxia with morphine. Beecher[3] emphasized this point

Causes and Treatment of "Shock"

Cause	Treatment
Pain	Morphine
Fear	Reassurance
Cold	Warmth
Asphyxia	Oxygen
Exhaustion	Rest
Dehydration	Fluid
Plasma loss (as in burns)	Plasma
Hemorrhage	Transfusion

in his account of the management of the Boston Cocoanut Grove victims at the Massachusetts General Hospital. Injury to the respiratory tract was common and serious, and it was felt that excessive doses of morphine had been detrimental in several cases. The following instructive case was recently observed in the Henry Ford Hospital:

N. K., a boy aged 12 years, was sleeping in a tent during a camping expedition. At 4 a. m. the fire which was necessary on account of the extremely cold weather spread to his bedding, and he received deep second degree burns of the face and hands. He was admitted the next morning, and petrolatum dressings were applied to the burned areas. It was noted that he was hoarse, a condition which was tentatively attributed to a cold. During the ensuing night considerable dyspnea and stridor developed, and the next morning an oxygen tent was placed over him. There was no relief of the dyspnea, nor was any benefit derived from mask inhalations of an oxygen-helium mixture (25-75). At noon on the second day the patient had become irrational and was thrashing wildly about his bed. A house officer suggested morphine for the extreme restlessness. Fortunately this suggestion was not followed, but, instead, tracheotomy was carried out. This relieved the dyspnea completely; the boy became rational immediately and lay quietly in bed. Henceforth his recovery was uneventful.

It was obvious that serious burns of the respiratory tract did not extend below the glottis. This experience would suggest that tracheotomy should not be withheld from any patient with signs of dangerous oxygen lack which is due to burns of the air passages.

The intravenous route for morphine should not be forgotten, since, as Beecher has pointed out, there may be poor absorption from the subcutaneous tissues or muscles in severely injured patients with depressed peripheral circulation. For this route smaller doses are used (⅛ to ⅙ grain, or 0.008 to 0.011 Gm.). For sedatives in the treatment of fear and hysteria, repeated doses of barbiturates intravenously are useful, e. g. sodium pentobarbital (nembutal) in 90 mg. (1½ grain) doses.

A burned patient may be chilled, owing to exposure in inadequate clothing or having been drenched with cold water. Such a patient will show some signs of shock due to cold, and for him external heat in moderation is indicated. But beyond this heat plays no useful part in burn therapy. The patient should be kept in a comfortable environment, with the temperature of the room preferably not more than 75 F. The so-called "burn tent" with the battery of electric lights is rapidly passing from our hospitals. The cold extremities of the patient in true shock from burns or other trauma appear to be the result of a compensatory phenomenon to conserve blood volume for the vital centers. Efforts to produce vasodilatation and thereby warm up such extremities may do serious harm.[4] Experimentally, the survival rate of burned rats was notably affected by environmental temperature.[5]

The extraordinary amount of soft tissue injury in an extensive burn results in a condition which gives "burn shock" its only claim to individuality. I refer to the loss of plasma from the vascular tree which produces hemoconcentration as indicated by high hematocrit, hemoglobin and erythrocyte count values. Several complicated formulas have been suggested for estimating the plasma deficit. I have found the formula of Harkins[6] useful. This states that, for every point the hematocrit is above the normal of 45, 100 cc. of plasma should be given. This would apply to an average adult weighing 150 pounds (68 Kg.), and proportionally less would be given to children. Harkins also suggested a simple first aid rule, which is that 50 cc. of plasma should be given for each per cent of the body surface which is burned. This could be expressed as a pint of plasma for each 10 per cent of body surface. The latter rule was utilized with satisfaction by Cope and his associates[7] in the treatment of the Cocoanut Grove casualties at the Massachusetts General Hospital.

The following case shows how plasma was employed with a patient with moderately severe burns:

E. S., a boy aged 16 years, was burned on April 21, 1942 when his oil soaked trousers caught fire. He sustained second degree burns of an area estimated as 22 per cent of the body surface (fig. 1). A hematocrit reading of 55 was obtained (fig. 2). Since this was 10 points above normal, it was estimated by Harkins' formula that 10×100 cc., or 1 liter, was necessary to correct the hemoconcentration. Actually he received 900 cc., which brought the hematocrit to normal. It should be noted that a part of the hemoconcentration was due to simple dehydration, as indicated by the high plasma protein value (8 Gm. per hundred cubic centimeters).

2. Lee, W. E.: The General Surgeon's Approach to the Problems Presented by Fractures and Other Traumas, Surg., Gynec. & Obst. **74**: 514 (Feb.) 1942.

3. Beecher, H. K.: Resuscitation and Sedation of Patients with Burns Which Include the Airway, Ann. Surg. **117**: 825 (June) 1943.

4. Blalock, A., and Mason, M. F.: A Comparison of the Effects of Heat and Those of Cold in the Prevention and Treatment of Shock, Arch. Surg. **42**:1054 (June) 1941.

5. Elman, R.; Cox, W. A.; Lischer, C., and Mueller, A. J.: Mortality in Severe Experimental Burns as Affected by Environmental Temperature, Proc. Soc. Exper. Biol. & Med. **51**:350 (Dec.) 1942.

6. Harkins, H. N.; Lam, C. R., and Romence, H.: Plasma Therapy in Severe Burns, Surg., Gynec. & Obst. **75**: 410 (Oct.) 1942.

7. Cope, O., and Rhinelander, F. W.: The Problem of Burn Shock Complicated by Pulmonary Damage, Ann. Surg. **117**: 915 (June) 1943.

In summary it may be stated that the shock factor in the patient with acute burns is treated by relieving pain with the proper analgesic, alleviating the symptoms of fear, cold and exhaustion by appropriate measures, and restoring blood volume when necessary by plasma infusion. It will be noted that adrenal cortex extract,

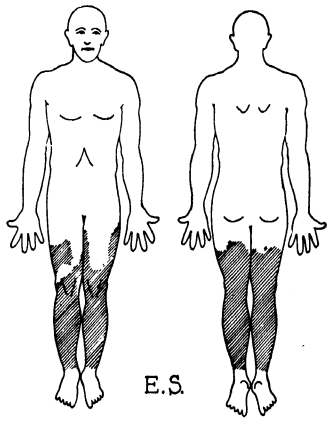

Fig. 1.—Burned areas of E. S. Approximately 22 per cent of body surface burned.

desoxycorticosterone acetate and similar preparations have not been recommended for reasons discussed at length elsewhere.[8]

NUTRITION AND FLUID BALANCE

With the patient with a moderate burn who is in the hospital about two weeks, there is no problem of nutrition. The order might read "Diet as tolerated." However, if there are large areas of third degree burn, the convalescence is inevitably complicated by more or less sepsis and multiple skin grafting operations under general anesthesia. There is loss of appetite at a time when a larger food intake is needed. There is loss of body proteins by necrosis and exudation, increased nitrogen excretion in the urine and increased metabolism due to fever. Therefore it becomes necessary to give maximum quantities of food by mouth, and frequently this must be supplemented by intravenous alimentation.

Special studies of a number of Cocoanut Grove victims were carried out at the Boston City Hospital.[9] Most of the patients responded to diets of 3,000 calories, of which 100 to 125 Gm. was protein. One seriously burned patient became edematous with a very low plasma protein level, and it was only by heroic feeding methods that the deficit was corrected. A maximum daily intake of 500 Gm. of protein was obtained by feeding a high caloric, high protein. high vitamin diet by stomach tube and giving intravenous amino acids, plasma and human albumin. The intubated food was supplemented by 30 to 40 Gm. of brewers' yeast, 20,000 units of vitamin A, 100 units of vitamin D, 200 mg. of ascorbic acid, 20 mg. or riboflavin and 200 mg. of nicotinamide daily.

For preventing and correcting dehydration, one may follow one of the usual clinical rules which have been applied to other conditions.[10] Sufficient fluids should be given by mouth to obtain a urinary output of 1,000 cc. daily as soon as possible. If vomiting occurs, intravenous solutions are given. It should be remembered that an excess of salt may be given with large amounts of isotonic solution of sodium chloride; 5 per cent dextrose is an alternative.[11]

GENERAL MEASURES TO COMBAT INFECTION

It is almost axiomatic that measures to promote the general well being of the patient, such as maintenance of adequate nutrition, correction of anemia by transfusion and the prevention of shock, will decrease the likelihood and seriousness of infection. There has been widespread hope that chemotherapy with the sulfonamides would reduce the incidence of infection. With this in mind the Cocoanut Grove patients at the Massachusetts General Hospital were given sulfadiazine.[12] It was found that the blister fluid of these patients contained bacteriostatic amounts of the drug. Infection was not a serious complication in these cases, but there was no control series. On the other hand, Meleney's [13] report of the cases studied by a number of National Research Council projects indicated that the incidence of infection in burns was not affected by sulfonamide therapy. Of 141 patients treated with general sulfadiazine, 31.2 per cent became seriously infected and 19.9 per cent were trivially infected, a total incidence of 51.1 per cent! Of the patients not treated with the drug, 10.4 per cent were seriously infected and 24.9 trivially infected, a total of only 35.3 per cent. It was concluded that the use of the sulfonamides did not lessen the incidence of local infection.

Naturally, there is hope at the present time that penicillin will be useful in preventing and treating the stage of sepsis in burns. No statistically significant figures are available at the time of the writing of this paper. I have treated 3 patients with penicillin, with gratifying results in 2. These patients had extensive third degree burns, and some of the later stages of skin grafting were entirely unsuccessful on account of infection.

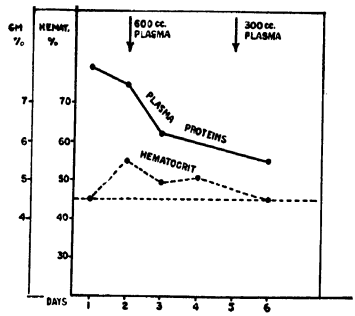

Fig. 2.—Effect of plasma therapy on the hemoconcentration resulting from burns (patient E. S.).

Subsequently there was a good take of the grafts when they were kept moist with small packs wet with penicillin solution (100 units per cubic centimeter) and

8. McClure, R. D., and Lamt C. R.: The Shock Factor in Burns and Its Treatment, Univ. Hosp. Bull., Ann Arbor **9**: 62 (July) 1943.
9. Taylor, F. H. L.; Levenson, S. M.; Davidson, C. S.; Browder, N. C., and Lund, C. C.: Problems of Protein Nutrition in Burned Patients, Ann. Surg. **118**: 215 (Aug.) 1943.
10. Maddock, W. G., and Coller, F. A.: Water Balance in Surgery, J. A. M. A. **108**: 1 (Jan. 2) 1937.
11. Lam, C. R.: The Chemical Pathology of Burns: A Collective Review, Surg., Gynec. & Obst. **72**: 390 (April) 1941.
12. Cope, O.: The Treatment of the Surface Burns, Ann. Surg. **117**: 885 (June) 1943.
13. Meleney, F. L.: The Study of the Prevention of Infection in Contaminated Accidental Wounds, Compound Fractures and Burns, Ann. Surg. **118**: 171 (Aug.) 1943.

the patients were given 100,000 units daily by the intramuscular route (12,500 units every three hours). The third patient died from staphylococcic septicemia after three weeks. He received what is now believed to be inadequate penicillin treatment, i. e. 40,000 units daily and no local packs.

NURSING CARE

Space does not permit an adequate coverage of this phase of burn treatment. The seriously burned patient needs the services of a special nurse who is patient, watchful and sympathetic and who preferably has had previous burn case experience. The exigencies of warfare, civil disaster and economic status will often preclude such care. Important items in nursing care are (1) providing comfort by the administration of medications, adjusting the room temperature and doing a hundred little things, such as applying cold cream to parched lips, (2) extending and reinforcing dressings, (3) providing and forcing fluids and food as outlined, (4) giving scrupulous attention to "pressure areas" to prevent decubitus ulcers and (5) serving constantly as a physical therapist, encouraging motion of all possible joints in order that when the skin is healed there shall be no disability from lame shoulders, knee contractures, foot drops and claw hands.

The adequate general care of the burned patient demands much from the hospital personnel, including doctors, nurses and orderlies. However, the reward of seeing such a patient recover and return to normal life is ample.

EDITORIAL NOTE.—This paper and the two papers by Drs. Harkins and Cope, which precede it, together with the four papers by Drs. Lee and Rhoads, Koch, Gurd and Gerrie and Davis, to appear next week, constitute a symposium on the treatment of burns.

STUDIES ON PROTHROMBIN

VI. THE EFFECT OF SYNTHETIC VITAMIN K ON THE PROTHROMBINOPENIA INDUCED BY SALICYLATE IN MAN

SHEPARD SHAPIRO, M.D.
NEW YORK

In their studies on the causative agent of the hemorrhagic sweet clover disease, dicumarol, 3-3′-methylene-bis-(4-hydroxycoumarin), Link and his students found that the prothrombinopenia inducing agent is active in vivo only and that a latent period exists before the prothrombinopenia becomes detectable. These facts led to the thesis that the delayed action occurs, at least in part, because the methylene bis-coumarin must first be metabolized in the body to a derivative which is, or from which is liberated, the effective agent. The quantitative degradation of dicumarol to 2 mols of salicylic acid prompted the agricultural chemists to study the action of salicylic acid on the prothrombin level (or activity) of the blood. They demonstrated in rats that salicylic acid can induce prothrombinopenia and that such prothrombinopenia can be prevented by vitamin K.[1]

This work was done in part at the Hospital of the Home of the Daughters of Israel, New York City.
The synthetic vitamin K (Menadione Bisulfite) used was supplied by Dr. Samuel M. Gordon, Endo Products, Inc., Richmond Hill, N. Y.
From the Third (New York University) Division, Goldwater Memorial Hospital, Welfare Island, and the Department of Medicine, New York University College of Medicine.

1. Link, K. P.; Overman, R. S.; Sullivan, W. R.; Huebner, C. F., and Scheel, L. D.: Studies on the Hemorrhagic Sweet Clover Disease: XI. Hypoprothrombinemia in the Rat Induced by Salicylic Acid, J. Biol. Chem. **147**: 463, 1943.

Following this, investigations conducted by Meyer and Howard [2] and by Shapiro, Redish and Campbell [3] established that the fundamental observations made on the rat apply to man also.

The possible clinical significance of the prothrombinopenia inducing action of salicylate as a cause of hemorrhage has recently been brought to light by English clinicians.[4] As was implied by Link,[1] they point out that vitamin K might be used prophylactically for patients receiving salicylates in large and continued doses. It has been possible to prevent the prothrombinopenia induced by 6 Gm. of acetylsalicylic acid given for one day by the intramuscular administration of 1 mg. of synthetic vitamin K.[3] My purpose in the present communication is to report the results of studies on the protective action of synthetic vitamin K against the prothrombinopenia produced by the more continued use of salicylate.

MATERIAL

Seventeen adults varying in ages between 22 and 76 were studied. In most of the instances the salicylate was used for therapeutic purposes. In addition to cases of chronic arthritis there were included in the group 3 cases of acute rheumatic endocarditis and 1 case of sickle cell disease.[5] In each subject the nutritional state was satisfactory and the food intake was adequate. The prothrombin time was normal during the control period in every case.

METHOD OF PROTHROMBIN ASSAY

The procedure used to estimate the prothrombin level (or activity) is based on the method of Quick [6] and includes estimation of the prothrombin time of whole and diluted (12.5 per cent) plasma. The rationale and clinical and experimental applications have been described in previous communications.[7] The greater sensitivity of the prothrombin time of 12.5 per cent plasma over that of whole plasma has been demonstrated also.

The thromboplastin used was prepared from fresh rabbit lung. All estimations were done in duplicate. The established normal standard for 12.5 per cent plasma was 39.5 seconds (standard deviation $\pm$ 2.5). Plasma was obtained by the addition of 4.5 cc. of venous blood to 0.5 cc. of $\frac{1}{10}$ molar sodium oxalate and centrifugation.

PROCEDURE

The salicylate and the synthetic vitamin K were given orally in three or four equally divided doses except where otherwise designated. Several estimations of the prothrombin time were executed during the

2. Meyer, O. O., and Howard, B.: Production of Hypoprothrombinemia and Hypocoagulability of the Blood with Salicylates, Proc. Soc. Exper. Biol. & Med. **53**: 234, 1943.
3. Shapiro, S.; Redish, M. H., and Campbell, H. A.: Studies on Prothrombin: IV. The Prothrombinopenic Effect of Salicylate in Man, Proc. Soc. Exper. Biol. & Med. **53**: 251, 1943.
4. Salicylates and Hemorrhage, Annotations, Lancet **2**: 419, 1943.
5. Murphy, R. C., Jr., and Shapiro, S.: Sickle Cell Anemia: I. Observations on the Behavior of the Erythrocyte in Sickle Cell Disease; Description of an Improved Technic for Studying the Red Blood Cells, Arch. Int. Med., to be published.
6. Quick, A. J.; Stanley-Brown, M., and Bancroft, F. W.: Study of the Coagulation Defect in Hemophilia and Jaundice, Am. J. M. Sc. **190**: 501, 1935.
7. Shapiro, S.; Sherwin, B.; Redish, M., and Campbell, H. A.: Prothrombin Estimation: A Procedure and Clinical Interpretations, Proc. Soc. Exper. Biol. & Med. **50**: 85, 1942. Shapiro, S.; Sherwin, B., and Gordimer, H.: Postoperative Thromboembolization, Ann. Surg. **116**: 175, 1942. Shapiro, S., and Sherwin, B.: Studies in Thrombo-Embolization: II. Observations on the Use of Dicumarol (3-3′-Methylene-bis-[4-Hydroxycoumarin]) in Embolization; Report of Five Cases, New York State J. Med. **43**: 45, 1943. Shapiro, S.; Redish, M. H., and Campbell, H. A.: Studies on Prothrombin: II. The Effects of a Single Small Dose of Dicumarol (3-3′-Methylene-bis-[4-Hydroxycoumarin]) in Liver Disease, Am. J. M. Sc. **205**: 808, 1943; Prothrombin Studies: III. Effect of Vitamin K on Hypoprothrombinemia Induced by Dicumarol in Man, Proc. Soc. Exper. Biol. & Med. **52**: 12, 1943. Flood, E. P.; Redish, M. H.; Bociek, S. J., and Shapiro, S.: Thrombophlebitis Migrans Disseminata, New York State J. Med. **43**: 1121, 1943. Shapiro, Redish and Campbell.[3]

control period before medication was commenced. Following this the prothrombin time was measured almost daily, at least five times each week. Acetylsalicylic acid was used exclusively. When the prothrombin time became prolonged the drug was discontinued until the prothrombin time returned to normal, when the medication was resumed and in addition to it menadione in the doses detailed later. Exceptions to this outline are indicated in the text.[8]

RESULTS

Sixteen subjects received 6 mg. of acetylsalicylic acid per day; 1 patient with acute rheumatic endocarditis, 5 Gm. daily. In 4 cases it was necessary to discontinue the medication because of intolerance to the drug. It is noteworthy that 2 of these disclosed unaltered prothrombin time after 30 Gm. of the salicylate had been ingested.

The remaining 13 subjects showed prolongation of the prothrombin time commencing the third to the fifth day after the initial dose of salicylate. Five of these received 2 mg. of menadione perorally daily. Two of the latter showed protection against the prothrombinopenia inducing action, the prothrombin time of the

Summary of Results

No. of Cases	Dose of Salicylate Daily	Extent of Prothrombinopenia After Salicylate	Dose of Menadione Daily	Number of Cases Protected by Vitamin K
5	6 Gm.	Moderate*	2 mg.	2
5	6 Gm.	Moderate	6 mg.	5
2	6 Gm.	Moderate	9 mg.	2
1†	5 Gm.	Moderate.	2, 6 and 9 mg.	Partial protection after 9 mg.

* Moderate: prolongation of the 12.5 per cent prothrombin time between approximately one and one half times and plasma twice normal.
† Complete protection during period when 400 mg. daily of ascorbic acid was added (intravenously).

diluted plasma remaining within normal limits. Two days after both drugs were discontinued the prothrombin time increased spontaneously and was promptly restored to normal by a parenteral injection of 6 mg. of menadione. In the remaining 3 cases the prothrombin time became prolonged approximately the same as when salicylate alone was ingested.

Five subjects were given 6 mg. of menadione daily and showed protection against prothrombinopenia as follows: Two exhibited increased prothrombin time after receiving salicylate for only two days and showed normal prothrombin time when synthetic vitamin K was added; 3 others exhibited prothrombinopenia following the ingestion of acetylsalicylic acid for one week and normal prothrombin time after the administration of 1 mg. of menadione for each gram of the salicylate taken. In 1 of these slight prolongation of the prothrombin time was detected for two days following withdrawal of both drugs, after which it spontaneously returned to normal.

Two patients were given 1.5 mg. of menadione to each gram of acetylsalicylic acid which had previously induced prothrombinopenia. Both exhibited complete protection, the prothrombin time remaining normal. One of these also showed slightly prolonged prothrombin time after both drugs were withdrawn, similar to the experience just noted.

One patient with acute rheumatic endocarditis was observed daily for eight weeks. Evidences of rheumatic activity including accelerated erythrocyte sedimentation rate were present during the entire period. The prothrombin time, which was normal initially, became prolonged on the second day of salicylate medication. Although the drug was withdrawn on the fifth day the prothrombin time remained increased even after the administration of 6.5 mg. of synthetic vitamin K for

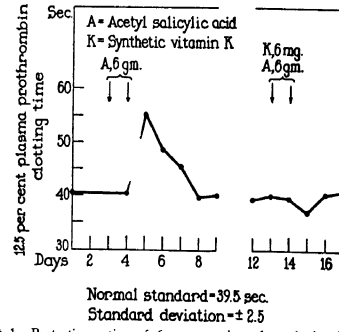

Chart 1.—Protective action of 6 mg. per day of synthetic vitamin K against the prothrombinopenia induced by 6 Gm. daily of acetylsalicylic acid.

three days. Subsequently the salicylate medication was resumed and 9 mg. of menadione was administered daily. During the first six days almost complete protection against the prothrombinopenia occurred, but following this the prothrombin time gradually increased despite the continuance of the antihemorrhagic substance. Commencing on the seventeenth day 400 mg. of ascorbic acid was given intravenously daily for four successive days (in addition to the salicylate and

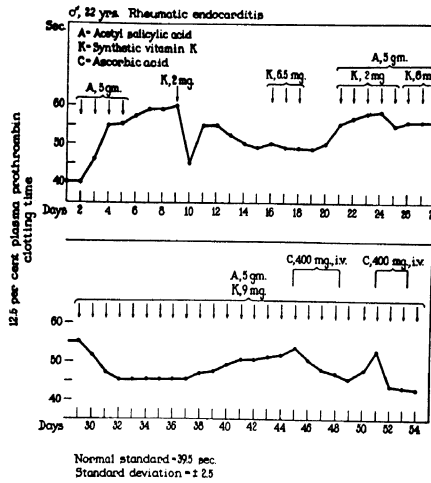

Chart 2.—Prothrombinopenia induced in a case of acute rheumatic endocarditis by 5 Gm. per day of acetylsalicylic acid and failure of doses of 2 to 9 mg. daily of synthetic vitamin K to neutralize this effect. The addition of 400 mg. per day of ascorbic acid administered intravenously restored the prothrombin time to normal.

menadione) and this was followed by a reduction of the prothrombin time to almost normal, which, however, became prolonged again immediately after the discontinuance of the ascorbic acid. Repetition of parenteral ascorbic acid was followed again by restoration of the prothrombin time to normal, which increased promptly

8. In 1 case (of sickle cell disease) the prothrombin time of both arterial and venous plasmas was studied (Shapiro, S.: Studies in Prothrombin: V. Arterial and Venous Plasma Prothrombin Time in Man, J. Lab. & Clin. Med. **28**: 1596, 1943). No significant difference between these was noted.

after the withdrawal of the vitamin C. This was repeated on three successive occasions, each time with the same sequence of responses.

COMMENT

The prothrombinopenia inducing effect of salicylate is a significant contribution to the possible understanding of the pharmacologic action of one of the foremost drugs used in medicine. Much work has been reported on the fate of salicylates within the body and some of their biologic effects.[9] Although the degradation product which is responsible for its influence on prothrombin is not yet decided, it nevertheless appears that the site of its activity is probably in the liver, for detoxification of salicylic acid is apparently accomplished by the formation of glucuronides. Hence the nutritional state of the individual, especially in respect to glycogen reserves,[10] should influence the response to salicylates. The importance of vitamin C in the mechanism, at least under certain conditions, is illustrated by the case that has been described in detail.

It appears that salicylate poisoning is a more serious problem in other countries, particularly Great Britain, than in the United States.[11] This might be accounted for by differences in diet, especially the freer consumption of fresh green vegetables in this country, which would augment the vitamin K intake.

The incidence of salicylate induced prothrombinopenia as a contributory cause of hemorrhagic phenomena will have to be determined by future observations when clinicians will have become more aware of the possibility. However, since salicylates alter the level (or activity) of prothrombin, prothrombin estimations should be made to determine the extent of this effect, especially when salicylates are used for continued periods of time. The studies reported indicate that prolongation of the prothrombin time is the most common effect; occasionally normal prothrombin time will be found compatible with extended salicylate therapy; in several instances reduction of the diluted plasma prothrombin time has been observed after salicylate administration.

There are no fixed levels of prothrombinopenia at which bleeding is known to be established. Indeed, the factors responsible for the maintenance of the integrity of the capillary wall still remain to be demonstrated. Consequently, except when a prothrombinopenic state is desired, prolonged prothrombin time after salicylates should be restored to normal by administration of vitamin K and, when necessary, vitamin C.

It has not been possible to establish in man a fixed dosage of synthetic vitamin K which will counteract the prothrombinopenia inducing effect of a given quantity of salicylate. Such possible relationship may be influenced by other factors such as the primary disease, fever, nutritional limitations and hepatic disorders. However, it appears that generally approximately 1 mg. of menadione is required to neutralize the prothrombinopenia inducing effect of 1 Gm. of acetylsalicylic acid.

For estimation of the prothrombin time we have found the most sensitive and most reliable method to be that which utilizes diluted (12.5 per cent) plasma. For proper execution this requires a thromboplastic agent of high activity, and for such an extract fresh rabbit lung has been found to be the best source.

SUMMARY

The level (or activity) of prothrombin should be determined by estimation of the prothrombin time when salicylate therapy is used.

Approximately 1 mg. of synthetic vitamin K will counteract the prothrombinopenia inducing action of 1 Gm. of acetylsalicylic acid. When factors such as fever, toxemia and limited nutritional intake complicate the situation, adjuvants such as ascorbic acid might be needed also. This is determined by serial estimations of the prothrombin time. This procedure has been found to be most sensitive and reliable when diluted (12.5 per cent) plasma is used.

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THE PRESENT STATUS OF THE TANNIC ACID METHOD IN THE TREATMENT OF BURNS

WALTER E. LEE, M.D.
AND
JONATHAN E. RHOADS, M.D.
PHILADELPHIA

Seldom in the history of surgical therapeusis has a new method entered an important field and swept all other methods into disuse as completely as did the tannic acid method in the treatment of burns in the decade following its introduction by Edward C. Davidson [1] of Detroit.

Now within a few years after the introduction of the sulfonamide drugs many of the old methods are back and the tannic acid method is disappearing from the field perhaps more rapidly than it entered it. It is worth while to review these changes, because we believe that their significance is probably basic and not determined solely by fashion.

Although many enthusiastic papers have been written about the tannic acid method and its variants, perhaps none sets forth its advantages better than the original presentation of Davidson. To understand Davidson's thinking on the subject of burns it is important to remember that in 1924 and 1925 the toxin theory of burn shock was nearing its zenith. The important experiments of Boyd and Robertson [2] had been reported in 1923, and the paper of Underhill and Kapsinow [3] contesting their results was not published until 1931.

While the New Haven theater fire was already a matter of history and Underhill's emphasis on the administration of fluids to burned patients was known to Davidson, the classic experiments of Blalock [4] and his associates on the plasma shift in burns, which did so much to establish the physical theory of burn shock, had not yet been reported.

Davidson's objective had been to find the means of precipitating denatured protein formed in the burned area and so to prevent or reduce the absorption of the toxic material which was thought to be responsible for the physiologic disturbances following burns. In systemic treatment he forced fluids both by mouth and by vein, reaching daily intakes of 10,000 cc. in some of his patients. His clinical results were good and impressed those who saw them. In addition to the precipitation of protein at the burned surface, he claimed for his method a reduction in the loss of fluid, electrolytes and plasma protein from the burn surface, the prevention of infection and decided relief of pain.

Critics of his method have based their attack until recently mainly on two points: first, that the tannic acid killed epithelium which might have survived, so

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1. Davidson, E. C.: Tannic Acid in the Treatment of Burns, Surg., Gynec. & Obst. **41**: 202, 1925.

2. Robertson, B., and Boyd, G. L.: The Toxemia of Severe Superficial Burns, J. Lab. & Clin. Med. **9**: 1, 1923.

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that in some instances a second degree burn might be converted to a third degree burn,[5] and, second, that tanning was not only ineffective in preventing infection but prevented the early recognition of infection and interfered with drainage.[6]

The most recent criticism of tannic acid is the charge made by Wells, Humphrey and Coll [7] that it is hepatotoxic when injected. Owing to the fact that the changes in the liver have been regarded as important manifestations of burn toxemia as defined by Wilson, MacGregor and Stewart,[8] this suggests that tannic acid might intensify the pathologic disturbances observed after burns rather than diminish or prevent them.

COMMENT

Considerable evidence on the arguments for and against tannic acid has accumulated and the points which appear important to us are summarized. The arguments for the use of tannic acid are discussed first.

Tannic Acid Precipitates Burn Toxins.—The consensus is now that for the liver, at least, tannic acid does more harm than good. Toxemic deaths were not infrequent among patients treated with tannic acid, so that its continued use over the years has not been sustained by general acceptance of the idea that it prevented toxemia. Evidence of impaired liver function was obtained in burns of only moderate severity treated with tannic acid at the Pennsylvania Hospital.[9]

The Use of Tannic Acid to Prevent Infection.—Tannic acid is fairly satisfactory in preventing infection in second degree burns, but we have never seen a third degree burn treated with tannic acid that did not eventually develop some infection. Some infection about third degree burns is usual with other methods also, and it is possible that the bacterial action may be useful in separating the eschar. The difficulty with the tannic acid method is that infection tends to spread under the eschar and may thus spread from third degree areas to second degree areas. A. H. McIndoe [10] believes that in this manner some second degree areas may become third degree areas. Infection is particularly apt to occur if the eschar becomes cracked, as in the more mobile areas of the body such as the hands. This could be avoided partially by splinting, but splinting has not always been practiced in conjunction with tanning.

In our opinion the advantage of tannic acid in combating infection was that it often postponed infection for at least a week, so that the patient passed the period of shock and through much of the period of toxemia before infection supervened. The tanning method shares with Koch's [11] pressure dressing method the advantage that the primary dressing is not changed during the first ten days unless serious infection develops.

The Use of Tannic Acid to Reduce Plasma Loss.—The untanned patient with second degree areas oozes copious amounts of fluid from the burned surface. This is not suppressed completely with pressure dressings but may be stopped by rapid tanning. Analysis has shown that bleb fluid has a composition resembling that of plasma,[12] so that the oozing from burned surfaces probably represents a serious plasma loss. It seems clear, however, that the major plasma loss is interstitial, and it is uncertain whether it is advantageous to prevent the loss of plasma from the surface if it has already left the circulation. Conceivably such material might contain toxic elements, so that it would be preferable for it to escape rather than for it to remain in the interstitial spaces to be reabsorbed when the edema goes down. The plasma requirements of patients treated with petrolatum gauze and pressure dressings have been no higher, as far as they have been reported, than are the requirements of patients treated with tannic acid. Even if it could be shown that tanning did save some plasma, this would no longer be a deciding argument for or against its use because of the greater availability of plasma at the present time.

Relief of Pain.—The tannic acid method remains one of the most effective methods of relieving the pain of burns. It is not, however, the only satisfactory method, as petrolatum gauze, pressure dressings and splinting also give good results.

Tannic Acid Is Toxic for Epithelial Cells.—The belief that tannic acid damages cells is supported by the recent experiments of Cannon and Cope,[13] in which various materials were applied to donor areas from which calibrated thickness grafts had been cut. The time required for healing was substantially longer with tannic acid than with boric ointment dressings.

Tannic Acid Causes Liver Damage.—Wells's work has been confirmed by Hartmann and Romence [14] and by Baker and Handler.[15] However, it seems clear that tannic acid must be much less toxic under clinical conditions than when injected experimentally. In the experiments of Baker and Handler it was applied to areas denuded of skin and proved to be hepatotoxic. However, even here the conditions are somewhat different from those existing in a burn. It must be remembered that the typical second degree burn is not reproduced in the dog or in most of the common experimental animals, so that experiments which duplicate clinical conditions are difficult to devise. The experiments of Underhill, Kapsinow and Fisk [16] should also be recalled in this connection in which it was shown that absorption of both phenolsulfonphthalein and strychnine was greatly retarded in a burned area.

That all interference with liver function following burns is not dependent on tannic acid was indicated to us by observations on 2 patients treated with 2 per

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cent gentian violet, as reported by Wolff, Elkinton and Rhoads.[9] In these cases the bromsulphalein retention rose to over 15 per cent thirty minutes after injection, the van den Bergh reaction was slightly increased and the plasma prothrombin was decreased. The literature contains evidence from human autopsies that degenerative changes may occur in the liver without the use of any of the tanning agents, and Wilson, MacGregor and Stewart[8] describe a case in which clinical jaundice occurred in which only oil was applied locally. The association of actual liver necrosis with the use of tannic acid, however, is striking in the autopsy series of Erb, Morgan and Farmer.[17]

The possibility remained that tannic acid would not be harmful on small burns but that with burns of more than one third of the body surface enough absorption would take place to cause fatal liver damage. The fact that patients with burns of over one third of the body surface frequently die even when no tanning agent is used and when the circulation is maintained with plasma would lead one to conclude that, although tannic acid may have been absorbed from large burns in sufficient amounts to cause some liver damage, this was seldom more than a contributory cause of death.

In summary, therefore, our present knowledge indicates that the tannic acid treatment relieves pain well, helps prevent infection in second degree burns and perhaps delays the development of infection in the deeper burns until danger from shock and toxemia have passed. It does not prevent toxemia, and if it is absorbed in sufficient quantity it is capable of producing liver damage. On the other hand there is as yet no substantial evidence that it has increased the mortality of burns in man. It decreased the loss of plasma-like fluid from the surface of second degree burns, but there is no conclusive evidence that it has reduced the loss of plasma from the circulation into the tissues.

Numerous statistics in the literature[18] indicate that its use was followed by a decrease in mortality, and we believe that it has saved many lives. Now, however, that invasive infection is so well controlled by chemotherapeutic agents it seems likely that the tannic acid method will be superseded more and more by other methods, many of which are similar to the very methods it displaced before the days of the sulfonamides.

The ultimate place of the tannic acid method will probably be based not on animal experiments nor on plastic results but on human mortality experience. Analyzing the statistics compiled by Harkins,[19] one finds that without tannic acid the combined mortality for 1,369 cases was 26.7 per cent, whereas with tannic acid in a series of 1,660 cases it was 10.5 per cent. It is premature to state whether a further reduction in mortality is being made by the nontanning methods now in use.

1833 Pine Street.

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SURGICAL CLEANLINESS, COMPRESSION AND REST

AS PRIMARY SURGICAL PRINCIPLES IN THE TREATMENT OF BURNS

SUMNER L. KOCH, M.D.

CHICAGO

In spite of the twin spurs of war and civil disaster to the study of burns we are still far from a complete understanding of the changes in the body tissues, the body fluids and electrolytes that result from a severe burn. In the past few years, however, many workers in our own and other countries have made helpful contributions to the solution of this difficult problem and have made the entire medical profession increasingly aware of its importance. McClure[1] and his able associates at the Henry Ford Hospital—Hartman, Harkins, Lam and Romence; Reid,[2] Siler, Altmeier and their associates at the Cincinnati General Hospital; Churchill,[3] Cope, Lyons and Cannon at the Massachusetts General Hospital; Lund,[4] Levenson and Taylor at the Boston City Hospital; Drinker[5] and his associates at Harvard University; Dragstedt[6] and his co-workers at the University of Chicago; Rhoads,[7] Wolff, Elkinton and Lee in Philadelphia; Hirshfeld[8] at the Detroit Receiving Hospital; Neal Owens[9] at Tulane; Elman[10] at the St. Louis City Hospital; Evans[11] and his associates at the Medical College of

From the Department of Surgery, Northwestern University Medical School, and the Children's Surgical Service, Cook County Hospital.

This paper, in a symposium on "The Treatment of Burns," is published under the auspices of the Section on Surgery, General and Abdominal.

With Dr. Siler's consent I have used almost the identical title of his excellent paper published in Surgery, Gynecology and Obstetrics in August 1942.

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Virginia; Wells [12] at Hartford; Gurd [13] and his co-workers at the Montreal General Hospital have all given time and intensive effort toward solving some of the unanswered questions in the minds of surgeons everywhere and in working out more effective methods of treating burned patients. Whipple,[14] in ably directing the activities of the Subcommittee on Burns of the National Research Council, has brought together men with helpful ideas and wide experience and has helped to make their ideas and experiences available for the armed forces and medical men everywhere.

In spite of the uncertainty concerning many of the physiochemical changes in body tissues and body fluids, three simple principles have come to be recognized as of fundamental importance in treatment: the surgical cleanliness that prevents infection, the compression of the injured area that prevents fluid loss, the rest that is so important if tissues are to heal in the minimum period of time. Each of these deserves brief consideration.

SURGICAL CLEANLINESS

The relative importance of the ounce of prevention and the pound of cure is nowhere more dramatically demonstrated than in the patient with an extensive burn. With the burned surface free from infection, covered with an occlusive compression dressing and left undisturbed, a patient can remain in comfort for ten, twelve or fourteen days and "emerge from the cocoon" at the end of that period with the burned surface completely healed wherever there has not been whole thickness destruction of skin. The same injury, if infection supervenes, can result in widespread destruction of the covering tissues that have survived the initial injury, long delay in healing and all the well known sequelae of an extensive, infected open wound.

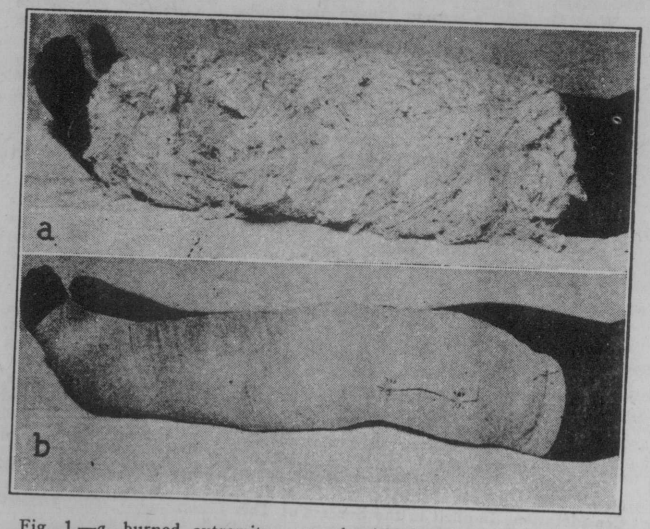

Fig. 1.—*a*, burned extremity covered with dressing surrounded with mechanics' waste; *b*, after application of elastic bandage.

We have long been aware of the rapidity with which virulent infection can develop in an unprotected open wound. Aldrich and Cruickshank,[15] particularly, have emphasized the vulnerability of burned surfaces to infection. We have been peculiarly slow in applying the simple remedy—to cover the burned surface at the very outset so as to avoid contamination; if it has been exposed to contamination, to cleanse it with care to avoid trauma and further contamination; and, finally, to "close" the open wound and keep it closed.

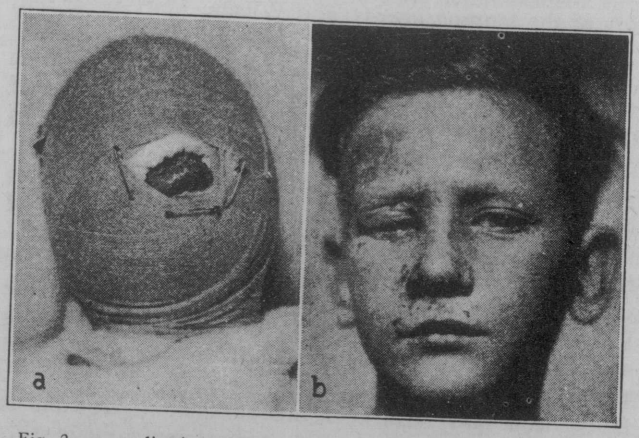

Fig. 2.—*a*, application of compression dressing over burned face; note swelling of upper lip where compression cannot be applied; *b*, appearance on removal of primary dressing seven days after admission.

To cover the burned surface at the earliest possible moment is simply to recognize the oft demonstrated fact that the most important sources of wound contamination are the uncovered mouths and noses of the patient and of every one about him. To cover the wound immediately with the cleanest protective at hand and eliminate those potential sources of infection is simple common sense.

If the open wound has been contaminated before the patient comes to us it is equally logical to attempt to convert it into a clean wound by simple soap and water cleansing, carried out with care to avoid trauma and to avoid further contamination. Concerning the necessity and wisdom of this procedure there has been considerable difference of opinion, owing in large part to the successful results obtained in spite of its omission at the Massachusetts General Hospital after the Cocoanut Grove disaster. There the burned surfaces of the patients were simply covered in the emergency room with sterile towels and shortly afterward with nonadherent gauze and occlusive pressure dressings. One may point out, however, that these cases were not typical of the cases of severe burns that come to the hospital day after day. They had had no first aid care; they had undergone a minimum of exposure to external contamination; they reached the hospital within a very brief period after the disaster occurred. Under these circumstances, and in view of the large number of cases demanding attention, no one, I think, would question the wisdom of the method of treatment carried out at that time.

What happens much more often, certainly with patients admitted to the Cook County Hospital, is that the patient has received extensive, often ill advised, first aid treatment—application of greasy ointment, butter, lard—whatever the corner drug store or home can furnish, and several hours after this application, carried out under voluble mouths and weeping eyes and noses, the patient is admitted to the receiving ward of the hospital.

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After such first aid treatment given three, six, twenty-four hours before the patient is admitted to the hospital, to attempt to convert the injured surface into a clean wound again seems to me simply common sense. Needless to say, one would insist on gentleness, the use only of soft cotton and plain white soap (never scrub brushes or tincture of green soap), irrigation with warm salt solution and the surgical cleanliness routinely employed in a well regulated operating room —masks, sterile gloves, thoughtful care to prevent additional contamination. Loose destroyed tissue which has not been washed away I should always lift away with sterile forceps and scissors. Blisters can be left untouched. Most of them will probably give way under a well applied compression dressing. That such cleansing can be of definite aid is indicated by cultures obtained before and afterward. One might say with some justification that swab cultures made after cleansing of a burned surface do not adequately represent the conditions present; but if the original culture shows a great variety of organisms present before cleansing is begun, and if many of these fail to appear subsequently during the process of wound healing, such findings seem to me significant.

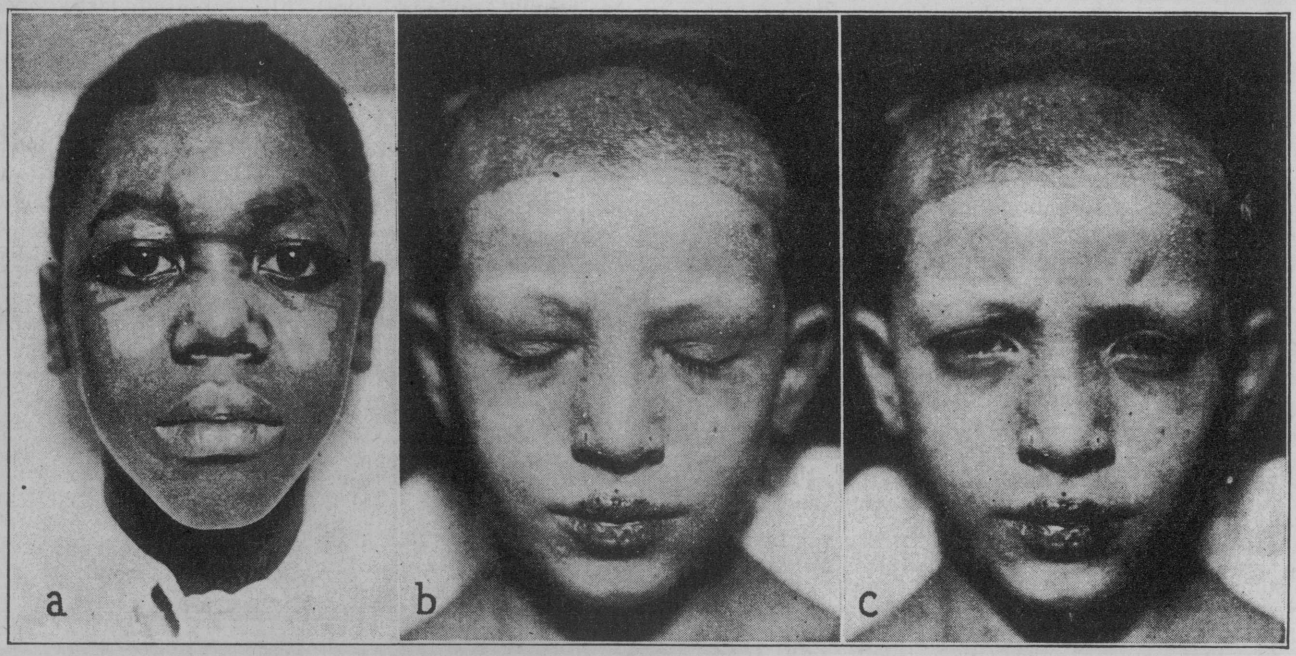

Fig. 3.—Appearance immediately after removal of compression dressing eight days and seven days after injury; note in patient at right absence of swelling in soft tissue of lids.

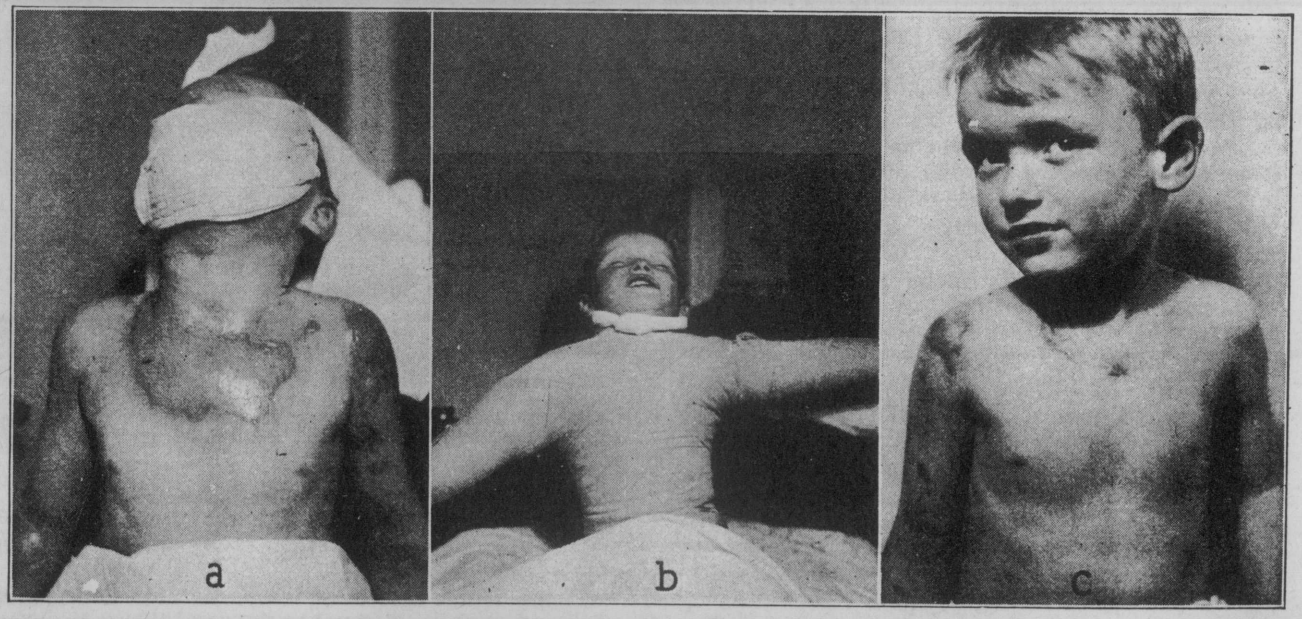

Fig. 4.—*a*, appearance of patient on admission one hour after injury; *b*, compression dressing; *c*, appearance on discharge, eight weeks after injury.

One would admit freely that there are borderline cases in which good judgment must be exercised as to what should be done and what omitted; and it is unnecessary to add that often treatment to anticipate or relieve shock must go hand in hand with local treatment.

THE DRESSING

To cover the burned surface with a nonadherent dressing which permits drainage through it and which can be removed with a minimum of difficulty when removal is indicated seems an entirely logical thing to do. Whether the fine meshed gauze applied directly to the wound surface should be impregnated with petrolatum, boric acid ointment, xeroform ointment, ointment containing zinc peroxide, sulfathiazole, sulfanilamide or sulfadiazine or simply saturated with salt solution is one of the details concerning which there is not sufficient evidence available to permit one to make dogmatic statements. My own-preference has been for simple petrolatum, though more recent experiences support the observations made by Meleney [15a] and others that the use of polyethylene glycols containing 20 per cent zinc peroxide is more helpful in controlling potential infection. The fact that a simple nonirritating dressing provides favorable conditions for wound healing is well demonstrated by Cope and Cannon's [16] study of the rate of healing under various methods of treatment of donor sites after removal of skin grafts, and by Hirshfeld, Pilling and Mann's [8] study of the comparative effect of tanning agents and petrolatum gauze on fresh wounds.

To cover the nonadherent gauze with sufficient gauze and mechanics' waste to make possible compression of the entire injured surface is the application of a simple method of arresting fluid loss. When confronted with bleeding, one's first impulse, whether layman or surgeon, is to apply direct pressure and stop the bleeding. Curiously, if we cannot actually see the bleeding we do strangely illogical things: apply ice bags, inject procaine hydrochloride (for the bleeding that follows a sprain), perhaps make incisions to relieve tension. Not often enough do we apply the simple principle of applying direct pressure when the site of bleeding is under the surface and when it involves a considerable area.

The fact is inescapable that the exudation of plasma that occurs from a burned surface and into the soft tissues underneath it is exactly comparable to the hemorrhage that occurs into the soft tissues following a crushing injury. It can be checked and limited in the same way—by the application of a compression dressing over the injured area.

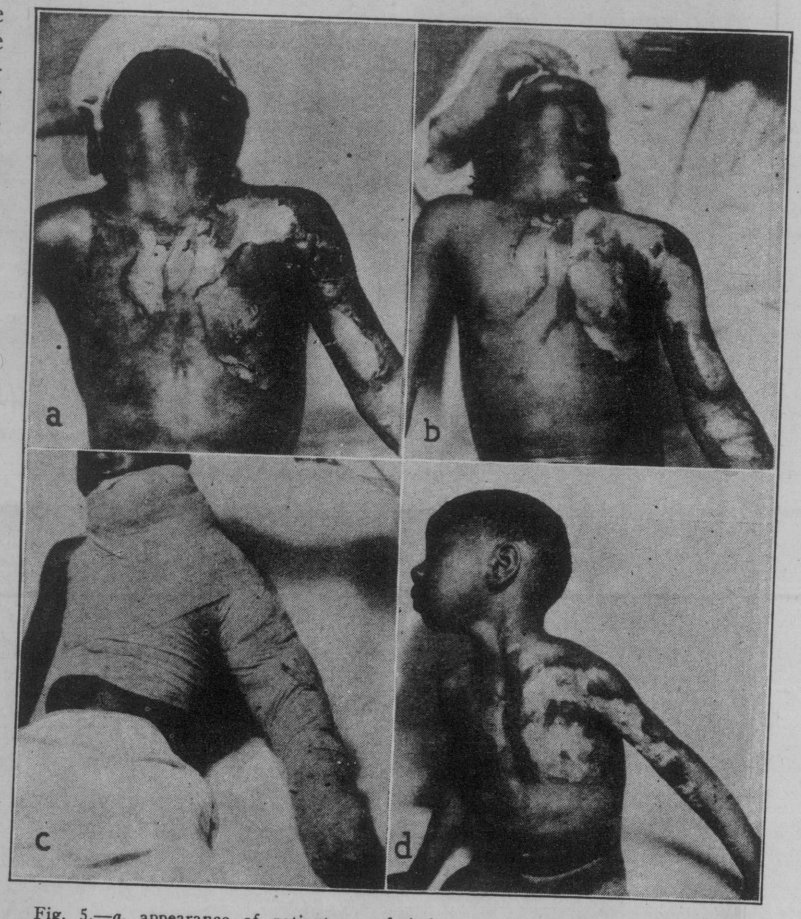

Fig. 5.—*a*, appearance of patient on admission two hours after injury; *b*, after cleansing; *c*, compression dressing; *d*, on discharge, nineteen days after injury.

It is scarcely necessary to point out that such a dressing should be a compression dressing and that it should not cause constriction or restriction of the circulation. If an extremity is involved, the dressing should begin at the tips of the digits, and the individual digits should be separated. Once properly applied, it should be supported with splints and left alone. If an extremity is involved, elevation of the part brings the aid of gravity to the return circulation and helps to prevent stasis and discomfort.

REST

Hugh Owen Thomas's dictum "Inflamed and injured tissues need rest" could well be emblazoned over the doorway of every surgical ward. Many of us can recall the days of our internship when it was considered proper and necessary to dress the burned areas of the injured patient each day from the time of his admission to the hospital. I look back with dismay to the injury I inflicted in years gone by on burned patients by repeatedly tearing down the tissue nature was trying to rebuild and by constantly exposing large open wounds to reinfection from the dust of the ward, from my own hands and from my uncovered mouth and nose. Yet I was trying to carry out the teaching and follow the practice of my attending surgeons.

Every surgeon and every burned patient is under everlasting debt to Davidson [17] because he taught us to "close the open wound"; and, since the coagulant crust that he devised could not be easily removed, we learned the wisdom of keeping the wound closed, of not subjecting it to repeated injury and of keeping it at rest.

That a tannic acid crust is not the most satisfactory means of attaining these objectives and that it has some serious disadvantages does not nullify Davidson's work, or the great impetus he gave to efforts to provide better care for burned patients.

Since 1925, when Davidson first advocated closing open wounds with a tannic acid crust, we have learned that the same closure can be obtained by a simple nonadherent dressing which does not produce coagulation of undamaged cells remaining over the burned area and which can and should be left alone, if no

15a. Meleney, F. L.: Study of Prevention of Infection in Contaminated Accidental Wounds, Compound Fractures and Burns, Ann. Surg. **118**: 171-186 (Aug.) 1943.

16. Cannon, B., and Cope, O.: Rate of Epithelial Regeneration, Ann. Surg. **117**: 85-92 (Jan.) 1943.

17. Davidson, E. C.: Tannic Acid in the Treatment of Burns, Surg., Gynec. & Obst. **41**: 202-221 (Aug.) 1925.

infection is present underneath it, until reformation of destroyed epithelium is as complete as possible. It has been demonstrated time and again that, if the whole thickness of the skin has not been destroyed, complete healing of the burned surface takes place in from ten to fifteen days if only infection can be prevented, if the injured surface is protected by the compression dressing and if the all important "rest for injured tissues" is maintained. The same conditions favor separation of the destroyed tissue if whole thickness destruction has taken place and help make possible early replacement by transplantation of skin from another part.

SUMMARY

The logical treatment of burns is based on three simple premises: 1. A burn, as Mason [18] pointed out so clearly, is essentially an open wound involving the superficial tissues and is subject to the same serious complications—infection and hemorrhage. 2. Both infection and hemorrhage can be prevented by well directed treatment at the very outset. The essential features of such treatment are surgical cleanliness, compression of the injured surface to prevent fluid loss, and rest. If infection and hemorrhage are not prevented, control of either or both can become exceedingly difficult. 3. If the injury has resulted in complete loss of covering tissues, replacement at the earliest possible moment is most certain to result in prompt healing, with maximum retention of function.

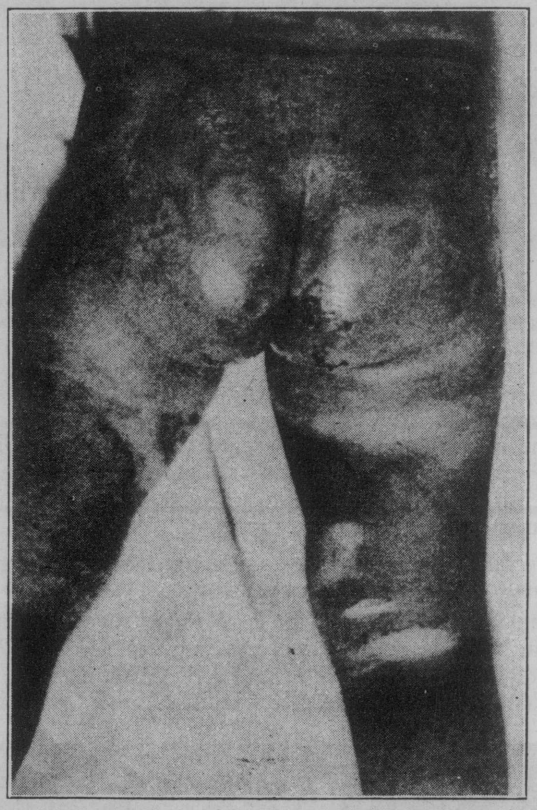

Fig. 6.—Appearance of burned surface twenty days after admission. With aid of indwelling catheter and good nursing care burns about the perineum can be kept covered with compression dressings throughout the period necessary for wound healing.

54 East Erie Street.

18. Mason, M. L.: Local Treatment of the Burned Area, Surg., Gynec. & Obst. **72**: 250-253 (Feb.) 1941.

THE EARLY PLASTIC CARE OF DEEP BURNS

FRASER B. GURD, M.D., C.M.
AND
JOHN W. GERRIE, M.D., C.M.
MONTREAL

Prior to 1925 in the treatment of burns, despite the fact that in all major clinics a high mortality was encountered, comparatively little attention had been paid to the victim of such a disaster. In that year the late E. C. Davidson commenced the publication of a series of articles which in recommending the tannic acid treatment of burns drew attention to the fact that loss of plasma was one of the major causes of death. Davidson's contributions were followed by an enormous increase in the interest shown in the treatment of the burned individual. During the ensuing years gradually more and more attention has been paid to the problem and, with the commencement of hostilities in September 1939, the problem of burn therapy has become one of the major interests of all surgical services in the armed forces.

One of the important features of this increased attention to these problems has been a recognition of the fact that skin is the most useful covering for a burned surface; that not only is plasma loss minimized and progressive hypoproteinemia halted by early skin coverage but that late deformity and disfigurement are thus minimized, and, more particularly perhaps, that the morale of the victim is improved. In our clinic at the Montreal General Hospital it has been one of our chief aims to plan the treatment of the burn wound so that early covering by means of skin grafting might be carried out.

A burn due to heat may be considered as a wound which is accompanied by special features, notably an immediate threat to life, owing to the particular form of shock which characterizes the lesion and the later threat due to toxemia, sepsis and nitrogen imbalance and, secondly, a widespread loss of surface tissue as compared with the depth of it.

Although tannic acid and other escharotics appear to have had a favorable effect in lowering immediate burn mortality, such methods delay the employment of skin coverage by grafting. This delay is due to several facts, namely (1) that the tanned tissue is frequently difficult to remove at an early date, (2) that suppuration beneath the tan has proved a bad feature in many cases and (3) that, as a result of the foregoing, exhaustion of the patient occurs so that, on the one hand, operative intervention must be delayed and, on the other hand, failure of grafts to take is probable. Gradually, and except under very special circumstances, the employment of tanning methods has been discarded in our Montreal General Hospital Clinic during the past three years. Effective shock and toxemia treatment with newer methods of controlling nitrogen imbalance and logical wound dressing care have resulted in a decrease in the mortality rate, and early and timed skin grafting has lowered morbidity and deformity.

Skin grafting is best started as soon after control of the shock and toxemia phases as the patient's con-

From the Montreal General Hospital, McGill University Faculty of Medicine.

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dition will permit. Theoretically this is before the burned areas pass into a phase of chronic infected granulations and the patient into a state of inanition and chronic protein loss. We are convinced that it is unwise to postpone grafting while waiting for problematic epithelium to grow in from the edges of the wound and for doubtful hair follicle and sweat gland epithelium to reach the surface and spread. The patient with an extensive burn recovers in proportion to the speed and extent of skin coverage. There is an optimum time, and we believe this to be early, when it is advisable to step in and surface the denuded area. In a recent publication from our clinic we [1] have stressed the importance of timing in burn treatment. By timing we have meant that a plan should be laid down so that each step in the treatment of the burned patient should be clearly indicated in a routine sequence. Part of this routine has been that a surgeon trained in plastic surgery is added to the shock and burn teams from the very outset. The plastic surgeon is made, therefore, a member of the team on which the time procedure is based from the first. He is available for advice and also to carry out whatever operative intervention for skin coverage is deemed advisable

ASSESSING THE BURN DAMAGE

At the outset the significant features of the burn history and the clinical appearance of the burned areas are carefully assessed and are of considerable value in

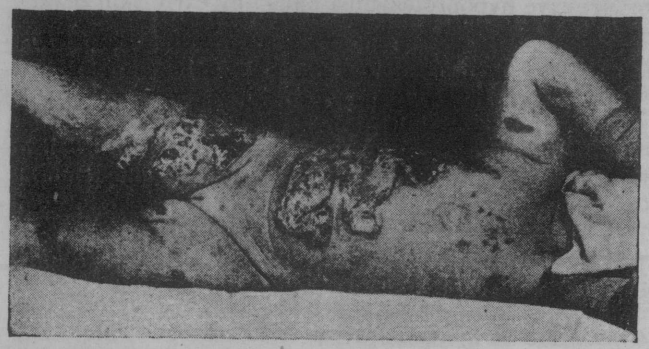

Fig. 1 (case 1).—Appearance on sixteenth day at first redressing, with the deep burns on the right side of the abdomen and thigh obvious, superficial burns healed.

"timing" the course of the future treatment. This assessment is the most reliable guide to burn depth and the probability of skin grafting to come. A scald from moderately hot liquid with an erythematous or lightly blistered skin will undoubtedly be a superficial burn, healed at the first redressing. A pressure steam burn or burn from excessively hot liquid with wide blisters and intervening searing will be deeper (figs. 1 and 2) and the plastic surgeon is "timed" in, is present at the first redressing and is prepared to go ahead with skin surfacing as indicated. Flash burns are of the same type. Flame burns as from ignited clothing are more uniformly deep (figs. 4 and 5), the skin is seared, and skin grafting will be required. Electrical burns are bad and there is often deep loss of tissue with coagulation which may further complicate the skin grafting which may follow. The plastic surgeon or one ready to carry on this work stands by at each redressing at ten to fourteen day intervals, prepared to carry out the necessary skin surfacing as early as feasible.

1. Gurd, F. B.; Ackman, D.; Gerrie, J. W., and Pritchard, J. E.: A Practical Concept for the Treatment of Major and Minor Burns, Ann. Surg. **116**: 641-657 (Nov.) 1942.

In assessing t e burn damage, the extent is calculated and recorded according to Berkow's tables. In estimating depth we have discarded all previous classifications, since they tend to confuse. The only useful classification, we believe, is that which indicates the eventual therapy required. Thus we have the super-

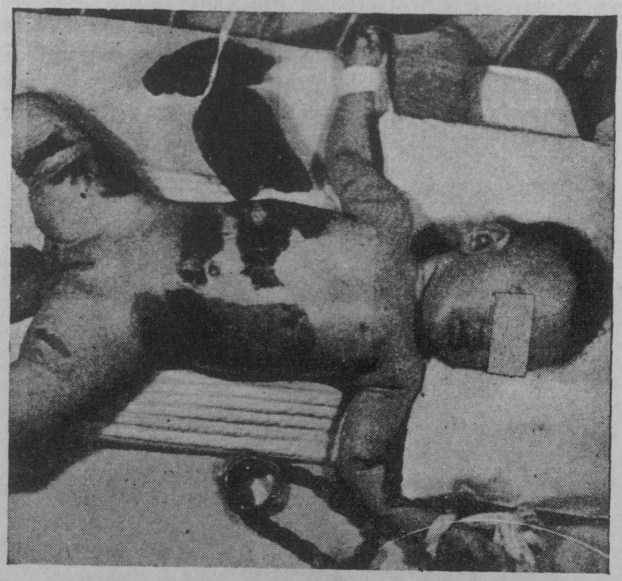

Fig. 2 (case 1).—Appearance at redressing ten days later with donor site on the left side of the abdomen healed and complete take of the grafts on the right.

ficial burn in which the epithelium will show quick regeneration or the deep burn which will require skin grafting.

The use of sodium fluorescein as described by Dingwall [2] may prove to be a help in assessing this matter of depth. How to incorporate the test in the "timed" treatment of burns is the problem.

THE OCCLUSIVE PRESSURE DRESSING OF THE BURN WOUND

The important principles of dressing in preparation of the burn wound for skin grafting are (1) rest, (2) infrequency, (3) occlusion and (4) pressure. To this is added a sulfonamide effect from a 5 per cent oil in

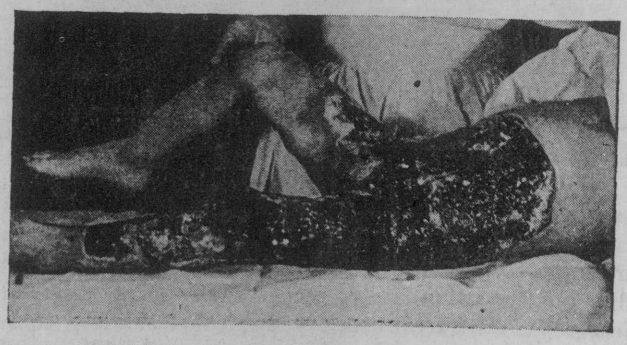

Fig. 3 (case 4).—An extensive body burn at the first redressing on the tenth day. This patient was completely grafted at the third redressing, on the thirtieth day, with isodermal grafts from the mother and autodermal grafts from the patient's back.

water emulsion of sulfathiazole prepared according to the Montreal General Hospital formula, as outlined by Ackman and Wilson.[3] Pressure keeps down edema,

2. Dingwall, J. A.: Clinical Test for Differentiating Second from Third Degree Burns, Ann. Surg. **118**: 427 (Sept.) 1943.

3. Ackman, D., and Wilson, G.: Surgical and Gynecological Experiences with an Emulsion of Sulfathiazole, Canad. M. A. J. **46**: 209-214 (March) 1942.

prevents fluid loss, promotes healing, prevents exuberant granulation and effectively replaces the eschar treatments. Infrequency of dressing and occlusion prevent cross infection and bathe the wound in an exudate which contains whatever immune bodies may be present. In none of our cases has a streptococcic infection complicated the picture. In the 5 per cent sulfathiazole oil in water emulsion we have an effective local agent, we believe superior to boric acid or petrolatum dressings. The sulfathiazole is active in the water phase of the emulsion, does not cake, has been found present in the granulations up to 120 mg per hundred cubic centimeters and has never given us a blood level of over 3.5 mg. per hundred cubic centimeters and rarely over 1.5. It is our opinion that the presence of sulfathiazole in the recipient graft bed up to these percentages in no way hinders but rather enhances graft take.

This dressing is placed following gentle soap and water washing and minimal débridement, with use of operating room technic, as soon as the patient's condition and the exhibition of shock therapy permit. The burned wound is shingled with 4 by 6 inch cuts of wide mesh lace (Nottingham lace of a bastard weave with 11 strands to the inch) well buttered with sulfathiazole emulsion (fig. 4). A liberal layer of emulsion is placed over the wide mesh lace and the dressing completed with surgical gauze, cotton waste to a depth of 4 to 6 inches and flannel bandage cut on the bias and reinforced with adhesive tape (fig. 5). The wide mesh lace tends to splint the wound surface and to provide a base for the dressing, which shows little tendency to slip. At no time have granulations become enmeshed in this or been a bothersome feature. On leg or arm burns, thin plasters may be added to prevent ankle, wrist or shoulder movement. This dressing (fig. 5) is infrequent. The patient is comfortable and, if the general condition permits, can be transported without fear of damage or contamination. This dressing is not removed, and there is no reason to remove it until the tenth to the fourteenth day. It is then taken down in the operating room and either (1) the burn wound is healed or early complete repair is evident or (2) it is grafted in whole or in part and redressed by the same technic for another ten to fourteen days or (3) grafting is deferred and the wound redressed

Fig. 4 (case 4).—The wide mesh sulfathiazole lace being applied.

by the same technic for another ten to fourteen days. In this way the whole burn treatment is "timed" into a series of one to five or six dressings at ten to fourteen day intervals with no intermediate dressings.

TIMING OF THE SKIN GRAFTING

In the treatment of wounds, whether operative or due to trauma, closure may be accomplished by either primary suture, delayed primary suture or secondary suture. For many years McKim and one of us (F. B. G.) have believed that in the care of wounds due to trauma the advantages of delayed primary and secondary suture have not been fully appreciated. In this communication we suggest that as far as the skin coverage of burns is concerned a similar terminology may be used and that there is a place in burn therapy for primary skin grafting, delayed primary skin grafting and secondary skin grafting.

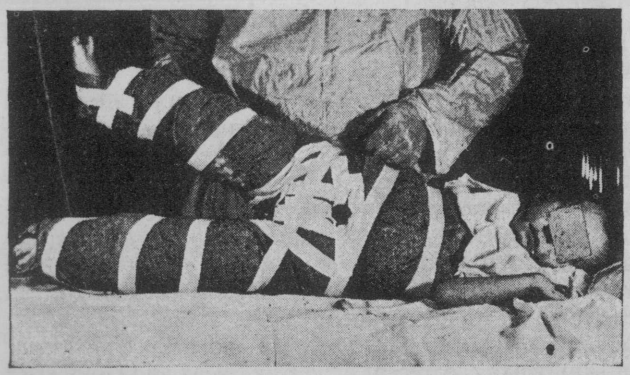

Fig. 5 (case 4).—The complete dressing with a surgical window over the umbilicus for wound bacterial cultures.

1. Primary skin grafting is analogous to primary wound suture. Under the name of immediate grafting it has been described by Forrest Young [4] and was suggested by Sir Harold Gillies on his recent trip to this country when he showed a four day old burn so treated. It is theoretically sound, as the burn is excised, the wound grafted and the area healed by first intention in a week or ten days, a minimum deep burn morbidity. However, it has a very limited applicability and is indicated only in obviously deep, circumscribed burns where there is no evidence, or likelihood, of shock. Few burns come into this category.

2. Delayed primary grafting of burns is analogous to delayed primary suture of wounds. The grafting has been delayed while the general condition of the patient and the local condition of the wound have been made ready. This is the usual and desirable type, and the aim of our clinic has been to graft most burns between the first and third dressings, the tenth to the forty-second day. The burn wound is dressed in an occlusive pressure sulfathiazole dressing and the decision to graft delayed until the tenth to the fourteenth day, when the shock and toxemia phases are passed and the first redressing is done. From the standpoint of the average patient's general condition, this is the optimum time for skin grafting, as it is before the period of inanition and chronic protein loss sets in. The local condition may not be ready but can often be prepared by judicious surgical excision of slough. The local condition is ready for grafting when slough and depth become apparent and before granulations become too obvious. It is at this stage that a test with sodium fluorescein might be helpful. The local area should be ready for a delayed primary graft at the first, second or third ten to fourteen day dressing.

3. Secondary skin grafting from six weeks on is undesirable but may be necessary in cases of badly infected wounds in emaciated patients in whom the burn wound was not initially controlled. It may be necessary in cases treated at home or in isolated areas where skin grafting was not available or its advantages were not realized.

4. Young, F.: Immediate Skin Grafting in the Treatment of Burns, Ann. Surg. **116**: 445-461 (Sept.) 1942.

ISODERMAL GRAFTING

Autodermal grafting, or the use of the patient's own skin, is preferable. Isodermal grafting, or the use of skin from other than the burned individual, is in the nature of a temporary dressing. It has a limited applicability, as in the case of a child recently seen with extensive burns whose own skin proved inadequate and whose general condition was bad.

Isografts dissolve and disappear in three to six weeks. The literature to date has not been very hopeful of securing permanent isodermal grafts of any sort. Padgett [5] tried isografting in 50 well controlled cases and concluded that isodermal grafting was not practicable. Bettman [6] advocates it as a life saving measure. Brown [7] reported 1 case of successful homografting, a small experimental graft from 1 identical twin to the other. However, the whole question of temporary isodermal grafts in the nature of a burn dressing is still wide open. Time [8] has set its stamp of approval on the method, giving publicity to the voluntary donation of convict's skin to burn sufferers and the establishment of skin banks. A recent article by Sachs and Goldberg [9] on foreskin isografts is reassuring and worthy of further work and substantiation. They stress the point of using skin from the newborn and premature infant in that its growth potentialities are greater. They believe that 65 to 75 per cent of their transplanted skin has survived and have been able to identify some grafts eight months later. This may open the possibility of refrigerated skin banks for burn emergencies.

INFLUENCE OF INFECTION ON GRAFTING

In none of the cases that have been under our control from the time of injury has infection delayed grafting. In a number of cases prior to admission to the hospital moderately severe infection with accompanying inflammatory reaction had already become established. In all of the latter group of cases the ulcers were ready for grafting after the application of but one dressing, that is at the end of from ten to fourteen days. The occlusive pressure sulfathiazole oil in water emulsion dressing in our experience has controlled or so moderated infection that grafting may be successfully carried out. At no time after the first redressing have we been able to culture streptococci, Bacillus pyogenes or Proteus vulgaris, the micro-organisms which seem to have the most deleterious effect on skin grafting. Although we have usually been able to culture Staphylococcus aureus, these have been present in but small numbers, and have not proved to be of importance. To summarize our experience in this regard, therefore, we feel justified in making the statement that controlled staphylococcus effects are not a contraindication to skin grafting when the technic recommended in this contribution is employed. The more serious infections have not in our experience occurred.

GRANULATIONS AND GRAFTING

Exuberant granulation tissue is an abortive attempt at wound healing that has been unsatisfied by epithelial covering at the appropriate time. It decreases the certainty of graft take and increases subgraft scar. Skin grafting of burned surfaces is therefore preferable before granulations become obvious. Exuberant granulations are the result of prolonged wound treatment and are no serious contraindication to skin grafting provided they are removed. It is preferable to remove exuberant granulations and graft rather than to give them prolonged treatment to render them less exuberant before grafting. The removal is done by a scraping motion with sharp scalpel rather than a slicing procedure. This provides a more even base with less bleeding. They are taken down to the firm yellow base from which they spring. The scalpel is preferable to a curet. Bleeding is, as a rule, easily controlled by the application of hot packs and the prompt application of the skin graft, which itself seems to have a pronounced hemostatic effect. Sutures or ties are seldom necessary to control hemorrhage.

THE CHOICE OF DONOR SITES

The choice of a donor area is occasionally influenced by the site and extent of the burn and the sex of the patient. With small burns in female patients it is well to take skin from the buttocks, loin or abdomen, or some place in keeping with the trend of increasing exposure in women's styles. In a limited arm or limb burn it is convenient to take skin from an adjacent area. In extensive burn cases one finds the skin where one may and hopes there will be enough. In such severe burns it is well to take extremely thin grafts and plan on cutting successive crops from the same donor sites.

Various body surfaces present definite anatomic features making them suitable, or otherwise, for skin donation. The Padgett dermatome makes all body surfaces available, and reasonably large and even sheets of skin may even be cut from the back or front of the chest cage.

Histologically, back, loin and buttock skin are most suitable and large sheets may be taken to considerable depth. These areas provide both a thick epidermis and dermis with excellent healing qualities. Abdominal and bicipital skins are thin, especially in young patients. Inner thigh skin is also thin, the outer thigh being of an intermediary type. The abdomen and front chest areas present dressing difficulties and tend to move with respiration even under our occlusive pressure dressings, with a resultant delay in healing.

The back, loin and outer thigh have been our donor sites of choice. They are dressed in identically the same way as the original burn with an occlusive pressure sulfathiazole dressing that is not removed for two weeks. The site is then usually healed or nearly so, is brought out into the air as quickly as possible and is massaged with petrolatum or olive oil. Prolonged dressing of donor sites tends toward maceration and further skin destruction or delay in healing.

THE TECHNIC OF FREE SKIN GRAFTING

The Padgett dermatome has taken some of the "ifs" out of skin grafting and has many advantages over the older knife and razor methods. It is not fool proof, and one must become familiar with the vagaries of knife, cement and drum. If used with care and understanding, even sheets of skin may be cut from almost any part of the body. It has the advantage of calibrated adjustment, and even thickness may be cut from 0.006 of an inch to full thickness of the skin. The Blair-Brown knife and suction box technic [10] has been an excellent method, especially in the hands of experts,

5. Padgett, E. C.: Care of the Severely Burned, Arch. Surg. **35**: 64-86 (July) 1937.
6. Bettman, A. G.: Homogenous Thiersch Grafting as a Life Saving Measure, Am. J. Surg. **39**: 156-162 (Jan.) 1938.
7. Brown, J. B.: Homografting of Skin, with Report of Success in Identical Twins, Surgery **1**: 558-563 (April) 1937.
8. Skinning Convicts, Time **42**: 46 (Nov. 15) 1943.
9. Sachs, A. E., and Goldberg, S. L.: Foreskin Isografts, Am. J. Surg. **60**: 255-259 (May) 1943.
10. Blair, V. P., and Brown, J. B.: Use and Uses of Large Split Skin Grafts of Intermediate Thickness, Surg., Gynec. & Obst. **49**: 82-97 (July) 1929.

but the consensus seems to be that the dermatome has even more to offer. The Marcks attachment to the Blair-Brown knife is possibly a further refinement of this method.

Thick grafts are less certain of take than are thin grafts, but the thick graft contains more of the elastic tissue of the dermis and is therefore of a better texture. In the early burn wound it is well to concentrate on take rather than on texture. The latter can be corrected later, if necessary. It is the opinion of our pathologist, Dr. J. E. Pritchard, that some of our thin grafts have acquired additional subgraft dermis and its contained elastic tissue in the course of time. Our preference has been for grafts between 0.008 and 0.016 inch in thickness, which is on the average 50 per cent, or less, of the total skin depth.

The dermatome is set up on a separate table and, if possible, the wound preparation and skin grafting are kept as separate procedures. The skin is sliced off to the desired amounts and depths. The thinner grafts are usually transferred to sheets of our wide mesh lace buttered with sulfathiazole emulsion. The skin adheres to the lace, so that it is more easily handled, since the surfaces which are at least partially covered by cement are prevented from sticking together. The lace and skin are sewn in; the lace helps to "fix" or "snub" the skin into position. If thicker skin is used, it is sewn in alone without the intervention of the lace, as it contains too much elastic tissue and curls away from, and will not adhere to, the lace.

The amount of skin transferable at one operation is limited only by the amount of time available and the patient's condition. In extensive burns it is wise to have two teams at work, one cutting the skin and the other sewing it in place. In this way maximum areas of skin may be transferred at one operation. Grafted and ungrafted areas are dressed together as was the original burn and left for ten to fourteen days before another intervention. If, at this next dressing, skin coverage is complete or nearly so, sutures are removed and the graft and donor sites exposed to the air and given petrolatum or olive oil massage.

SKIN GRAFTING THE BURNED FACE

Burns of the face present a different problem to those of other parts of the body. Free skin grafting, at best, is not a satisfactory method, since it is so likely to result in a "patchwork quilt" appearance. Skin transferred from other parts of the body usually tends to retain its own color and texture or, what is worse, to become pigmented in an unsightly fashion. It is clear that the surface of the eyelids must be replaced early, since serious injury to the eyeball may otherwise be suffered in consequence of the ectropion which is sure to develop. Similarly, repair of ectropion of the lips, particularly of the lower lip, should be an early procedure chiefly because the drooling which occurs under these conditions has such an unfavorable effect on the patient's morale. The grafting of skin to other parts of the face should however, we believe, be postponed for many weeks in most cases since, in addition to the unpleasant appearance of the grafted face, it is clear that on the one hand deep burns of the face are comparatively unusual, and on the other hand the skin of the face exhibits an unusual capacity for epithelial regeneration from deeply placed hair follicles and sweat glands.

It is a matter of common knowledge that the cartilages of the ears and nose are very liable to necrosis as the result of burns and that large portions are often lost. The repair of these and other gross deformities becomes the problem of late reconstruction and is not discussed in this contribution.

It should be an axiom that "face skin is best for face reconstruction." As a general principle we believe that a scarred face is preferable to the masklike effect which follows free grafting in the early post-traumatic period.

BURNED PATIENTS GRAFTED

A survey of the last 100 consecutive skin grafting operations that we have performed shows that there were 35 delayed primary grafts in 22 burn patients. In addition, 12 skin grafting operations were carried out on 8 patients for the late reconstruction of burn deformities. This is a total of 47 out of 100, or 47 per cent of our skin grafting operations for primary or secondary reconstruction of the burned patient. It is thus seen that approximately one half of our skin grafting procedures have been rendered necessary on account of burns.

REPORT OF CASES

A few instructive cases are briefed, with comment on the lessons learned:

CASE 1.—A 35 per cent scald in a 14 month old girl admitted in severe shock. Wide blisters with intervening "white leather" areas. Shock treatment and an occlusive pressure sulfathiazole emulsion dressing within four hours of the accident. Excellent recovery from shock. First redressing on the sixteenth day, when the superficial burns were found healed and the deep areas obvious (fig. 1). The sloughs were surgically removed and covered with skin of 0.008 inch thickness from the opposite side of the child's abdomen. Second redressing ten days (fig. 2) later with complete graft take, and child discharged three days later.

This case, in our opinion, exemplifies ideal "timed" treatment of a severely burned patient with a minimum period of morbidity. Early skin coverage, moreover, anticipated and so prevented the onset of exhaustion due to nitrogen imbalance.

CASE 2.—Circumscribed, obviously deep burn of the thigh 8 by 6 inches in a man when a motor car engine was pushed against the thigh in an accident. Treated as an outpatient and sent in for grafting on the thirty-fifth day. Placed in occlusive sulfathiazole dressing and grafted four days later with complete take. Discharged fourteen days after grafting with complete healing.

This would have been an excellent case for immediate primary grafting. The patient might then have been in the hospital only fourteen days from the time of burning until complete healing. There was a lapse of thirty-five days in "timing."

CASE 3.—A 55 per cent extensive deep burn of the abdomen, thighs and legs of a man from burning clothing. Remarkable recovery from shock and toxemia. In excellent physical condition from the fifteenth to the thirtieth day, with subsequent progressive decline from inanition and hypoproteinemia. Because of extensive slough, grafting was deferred until the fifty-fifth day. Further grafting on the sixty-ninth day, with death three days later from chronic protein loss.

We believe that this patient might have lived if operative removal of burn slough and the application of grafts had been instituted about the fifteenth day and carried on progressively while the patient was still in good condition. Such a case also would now receive the high protein instillation into the digestive tract, as recommended by Charles Lund and his associates at the Boston City Hospital.

CASE 4.—A 30 per cent deep flame burn of the abdomen, thigh and lower leg of a 23 month old boy. Occlusive sulfathiazole pressure dressing routine from the start, but grafting delayed until the third redressing (fig. 3) on the thirtieth day because of the extent of the burn and the depth of the slough. The thigh and lower leg were completely covered with 0.008 inch skin from the mother's buttock and the abdomen with 0.008 inch skin from the child's back, in one operation. Definite improvement in the child's condition. At the fourth dressing, on the fortieth day, the child's skin had taken completely on his abdomen and the mother's skin about 80 per cent on the thigh and leg. This will be redressed at ten day intervals and the thigh and leg regrafted with the child's own skin, if and when the mother's skin disappears.

An example of extensive skin coverage at one operation using autodermal and isodermal grafts. This is a recent case; we are consequently unable to report a final result.

CONCLUSIONS

1. Skin grafting of the burned area at the earliest possible moment is one of the chief aims in burn treatment. Such a procedure minimizes protein loss, prevents contractures and maintains morale.

2. An occlusive pressure dressing with a 5 per cent sulfathiazole oil in water emulsion, timed at infrequent intervals, has been helpful in achieving this result.

3. Delayed primary skin grafting between the tentl and fortieth days is the usual technic. Occasionally immediate primary skin grafting may be indicated.

4. Isodermal grafting has a limited but perhaps valuable applicability.

5. Under the occlusive pressure dressing routine infections are seldom bothersome and do not delay grafting.

1538 Sherbrooke Street West.

THE LATE PLASTIC CARE OF BURN SCARS AND DEFORMITIES

JOHN STAIGE DAVIS, M.D.
BALTIMORE

The enormous increase in the number of burns of all types, caused by all sorts of agents, which are the direct result of waging war on land, on the sea and in the air will necessarily increase the number of burn scars, many of which will eventually have to be treated by plastic repair.

The status of the modern methods of burn treatment in all its phases has been considered in the preceding papers of this symposium. In completing the symposium the late plastic care of burn scars and deformities is dealt with, and in preparing this contribution I have drawn freely from articles which I have previously published on the treatment of scars of various types.

The earliest paper of importance which appeared in the United States on burn scar contractures and the innumerable subsequent deformities was written by Thomas D. Mütter, professor of surgery in Jefferson Medical College, Philadelphia, and was published more than a hundred years ago, in July 1842, in the *American Journal of the Medical Sciences*. His remarks on burn scars and the methods of handling them by operative procedures are well worth while reading today by any one interested in the subject, and some of his procedures are good practice still.

This paper, in a symposium on "The Treatment of Burns," is published under the auspices of the Section on Surgery, General and Abdominal.

With the newer methods of treating burns, which are designed to eliminate infection and prevent the loss of serum and in which early skin grafting or flap shifting is done to induce healing, the occurrence of objectionable scars and of subsequent deformities due to scar contractures should be much less common than heretofore has been the case. However, in spite of these new methods burn scars still occur, and contractures may still form in those instances in which the full thickness of the skin and various depths of the underlying tissues have been destroyed.

The vast majority of burn contractures, and the deformities caused by them, must be treated by operative methods, and there is little that is new in the procedures which have been found most effective in dealing with them.

Burn scars and burn contractures are probably more consistently botched than any other group of cases, and this is largely because they are deemed simple and are attempted by nearly every surgeon in whose hands they may happen to fall or to whose service they may be admitted. The care of these contractures should be only in the hands of surgeons skilled in plastic repair, who have a thorough knowledge of how to deal with scar or scar infiltrated tissue and who understand the possibilities of its utilization.

It is a sound plastic principle that when scar tissue is present it should be completely removed before attempting repair by suture, by skin grafting, by flap shifting or by flap rotation. In many instances, however, it is obviously impossible on account of the size of the scar and the situation to excise it completely and carry out any of the procedures just mentioned. In these cases scar tissue can and should be utilized in accomplishing the final restoration.

The surgeon who is inexperienced in the handling of scars will frequently operate early on burn scars of all types, apparently not realizing that nature assisted by massage and physical therapy, and sometimes by carefully given x-ray or radium therapy, will in the course of months materially change the character and appearance of the scar. My own practice is to wait until at least six months have elapsed after healing is complete before operating; in other words, until the scar is matured. In this way useless operations are avoided and procedures which would have been required had the operation been done early are frequently found unnecessary. Furthermore, if it is advisable to utilize scar or scar infiltrated tissue in the final repair, the circulation is better, the scar is softer and suitable areas may be shifted as flaps with considerable advantage. It has been my observation that the employment of scar tissue in the final repair is not utilized nearly as often as it can and should be.

All types of scars may follow burns, and the character of the scar depends on the depth of destruction, on the extent of the burn and the location, and on the rapidity and character of the healing. Scars may be unstable, smooth (skin level), depressed, adherent contracted and keloidal in type. More than one type of scar may be present on the same patient following the healing of the original burn. Any one or all of these may cause deformity and loss of function. Pain may also be a symptom in any type of burn scar. The deformities caused by burn scars range from very slight skin defects and contractures to the most horrible distortions of all parts of the body.

The power of scar contractures is very great and may cause a variety of unusual deformities, for exam-

ple drawing the shoulders toward each other, or completely dislocating joints.

In burn contractures of the neck, chest and chin the shape of the mandible is often distorted, and the full development of the bone is checked by scar pressure. The alveolar process is frequently turned outward and the teeth forward and even downward in severe cases. Long bones may be bowed, and joint surfaces may be angled. In certain old extensive burn scars with adhesions, for instance between leg and thigh or thigh and pelvis or the arm and chest, it is advisable to do the relaxing operation in stages, as shortening of muscles under contracted scars and also shortening of vessels and nerves must be thought of. The friability of bones due to the atrophy of disuse is also of importance.

The location and extent of the scar modify prognosis and treatment. In the late plastic care of burn scars and deformities a number of methods may have to be employed, either singly or in combination, as there is no one procedure of choice which is applicable for the relief of all types of cases.

The most important procedures are excision with closure by sutures; excision with skin grafting; excision with lateral relaxation incisions in adjacent normal tissue, advancement to cover the original defect, with skin grafting of the relaxation defects; gradual partial excision with closure; the use of relaxation incisions in the scar itself with skin grafting; the use of Z-plastics; the use of skin grafts of various types, and the use of pedicled flaps. All of these maneuvers must be understood by the surgeon doing this work, and his judgment should be good enough to select the method, or combination of methods, which promises the best results in each individual case. These methods will be considered briefly, and then the various types of scars and the procedures which in my experience are most suitable for their correction in various regions will be taken up.

In my own operative work on burn scars I have found it advantageous to mark out with 5 per cent brilliant green in 95 per cent alcohol all the incisions to be made. This is most helpful in planning relaxation incisions and procedures such as Z-plastics, in tissue made up of scar or strongly infiltrated with scar. When scar is incised, it is quite remarkable in certain instances to note the retraction of the wound edges and, where scar flaps have been released, to note the extraordinary changes in line of pull into unexpected directions.

OPERATIVE METHODS OF TREATING BURN SCARS AND DEFORMITIES

Excision.—When it is possible, a burn scar which lends itself to such treatment should be excised completely and the wound closed in layers. If, after excision, closure is not possible, even with undercutting, then the defect may be skin grafted with the selected type of graft.

Another method which is very helpful, the principle of which was well known in the time of Celsus, is that, when the scar has been excised and the wound cannot be closed by suture, to make relaxation incisions in the normal tissue beyond the defect on one side or both down to the deep fascia if necessary and then undercut the tissue, making a double pedicled flap. This flap may then be immediately advanced to cover the defect left by excising the scar or, if circulation is doubtful, may be sutured back into its original bed to be used later as a delayed transfer double pedicled flap. The method may be modified by forming the double pedicled delayed flaps, before the scar is excised, and then, when the circulation of the flaps is assured, excise the scar and immediately shift in and suture the flaps. When the flap or flaps are shifted in finally and sutured over the original defect, the wounds in the normal tissue are grafted, to hasten healing.

Another method, which will sometimes give relaxation and avoids further incisions through skin is, after undercutting outward on each side as much as necessary, to make an incision from below, parallel to the length of the wound on each side, at the outer margin of the undercut area up through the subcutaneous fat without cutting through the skin, and then if relaxation is sufficient close the original wound with sutures.

Gradual Partial Excision with Closure.—The first paper on gradual partial excision was published in 1915 by H. Morestin. This most useful plastic principle is a progressive reduction in the size of a scar by successive excisions, with closure. The method depends for its success on the fact that the skin beyond the lesion has a tremendous stretching capacity, especially when the stretching is done slowly.

After determining the amount of tissue to be removed, the area is marked out. The pattern is usually made in the shape of an elongated ellipse whose long axis, in order to facilitate closure, is in the most advantageous direction; but any shaped area may be removed as long as the resulting defect may be closed by sutures. The marked out area should be cleanly excised with a sharp knife down to normal tissue, care being taken to avoid unnecessary injury to the wound edges. Some undercutting may be necessary. All hemorrhage should be checked and the wound closed with deep sutures of fine white waxed silk or cotton, to take off tension, and on-end mattress sutures of horsehair or nylon to approximate the wound edges.

After a suitable interval, several weeks to several months, depending on the situation and when the surrounding skin has stretched sufficiently, the process is repeated and again repeated at suitable intervals until the scar has been completely removed and a narrow line scar remains. The successive excisions should ordinarily be made inside the area of the scar until the final step is reached, when it may be necessary to encroach slightly on the surrounding tissue. Proceeding in this way, one finds that the final narrow scar will be little if any longer than the long axis of the original scar. The technic is simple, and there is little danger in the procedure. Before the primary and secondary operations, preliminary massage to soften and loosen tissues is advantageous. The amount which it is safe to excise at any one operation varies with the elasticity of the skin around the scar.

If carried out properly, gradual partial excision will result in a narrow scar, which may be either straight or curved or angled depending on the way in which the excisions have been made. Often it can be managed so that the final scar will lie in a natural fold. Occasionally in extensive scars it is impossible to remove it all by this method, but the size of the scar may in this way be reduced sufficiently to make a simple plastic procedure possible.

Relaxation Incisions.—As I have said before, it is a good plastic principle to excise all scar tissue completely before making a repair, but in many instances it is not practical to carry this out, and when this is the case relaxation incisions in the scar itself may be essential in order to bring about a satisfactory result.

The purpose of relaxation incisions through scar tissue is to relieve scar tension which may either interfere with growth or with function or with permanent healing or with all of these. Relaxation incisions have been most helpful to me in dealing with large contracted thick adherent scars, and also with unstable scars which are difficult to heal and which when healed frequently break down.

The contracted portion of the scar is put on the stretch, the most binding area is located and the proposed incision line is marked out transversely across that portion of the scar. If several areas are to be relaxed, they also are marked out. Then the scar is divided completely through its full depth down to normal tissue and if possible out to normal tissue surrounding the scar. Deep attachments are loosened. Relaxation incisions may be single or multiple. They may be straight, curved or angled. If the margins of the incisions are tight and the underlying tissue tends to bulge out into the wound, the incisions may be lengthened or the margins may be relaxed by radiating incisions until they are slack. On the shaft of an extremity the relaxation incisions are ordinarily made parallel with the length of the limb, but in other large scars the incisions are made across the scar pull in any needed direction.

In making relaxation incisions the tendency is always to underdo rather than to overdo, and it must be borne in mind that the relaxation must be as thorough as possible without interfering with the circulation of scar areas between the incisions.

After relaxation incisions have been made, the tissues in the defects may be ready for immediate grafting or it may be necessary to allow a few days to elapse in order to improve the circulation and stimulate granulations before grafting can be done. All types of grafts may be used to heal the defects made by the relaxation incisions. In order to conserve skin I frequently employ small deep grafts, but split (thick Ollier-Thiersch) grafts or whole thickness grafts may also be used, depending on conditions.

In very large scars one may have to proceed in stages, gaining as much as possible each time. The first incisions may be done, the defects healed by skin grafting, and later further relaxation in other portions of the scar may be carried out in the same way. After several months have elapsed, other relaxation incisions may be done if necessary, sometimes across those made previously or in the direction not indicated at the first operation. In the thick heavy scars after relaxation incisions have been made it will be found that in time the relaxed scar tissue, although it may originally have been hard and rigid, will soften and change its characteristics so that it will be usable in the final result.

Z-Plastics.—In the contracted scar where there is a prominent scar band, bridge or web, or a scar groove, with fairly lax tissue on each side, the Z-plastic is of great use and up to the present time has been used extensively by only a few surgeons. The name is given to the method because the outline of the incision is roughly that of a Z. The maneuver will be considered here in its simplest form.

With the scar bridle or groove under tension, the proposed incisions are marked out. The central line of the Z is laid down along the most prominent part of the bridle in the selected length. Then the arms of the Z, which are of the same length as the central line, are laid down at each end of the central line on the opposite sides and at an angle of about 60 degrees to it, and they should be parallel to each other.

When the incisions are made along these lines, two broad based triangular flaps are formed whose bases are opposite each other. These flaps are undercut and completely mobilized. They are then transposed so that their outer margins approximate and the tips of the flaps touch the outer corners of the bases of the opposite flaps. The edges are then sutured with interrupted or on-end mattress sutures of nylon or horsehair. The sutured wound is also Z-shaped, but the Z is turned through about 90 degrees, is elongated, and the central line of the original Z lies transversely across the scar pull. The actual amount of relaxation varies between 50 and 100 per cent of the length of the central line of the Z.

By Z-plastics, scar contractures may be relieved by the utilization of scar tissue. The success of the method depends on the presence of slack tissue on each side of the scar band or groove. By its use, in many instances, skin grafting or flap shifting may be avoided, and it is one of the most generally useful plastic procedures.

Skin Grafts.—A skin graft is a free transplant. The technic of obtaining the various types of skin grafts is so well known that I will not go into it here. Grafts may be grouped into thick grafts, which include small deep grafts, and whole thickness grafts (Wolfe-Krause grafts), half thickness or split grafts, which are really thick Ollier-Thiersch grafts, and thin grafts, which include true Reverdin grafts and true Ollier-Thiersch grafts, both of these grafts being composed of the epidermis with the tips of the papillae of the corium. In other words, the tiny Reverdin and the larger Ollier-Thiersch grafts are as thin as they can possibly be cut.

Another type of skin graft, which is sometimes used to fill out depressed scar areas, is the cutis or dermal graft. It contains all the components of the skin except the epidermis. The size of the graft required is marked out on the donor surface, and then every particle of the epidermis with the papillae of the corium is removed. The remaining portion of the corium is either completely dissected away from the fat or a small amount of fat may be left attached, and this tissue is then used to fill out the depressed area. It is usually placed in an undermined pocket and should be spread out and held with sutures in position in the bed prepared for it. The process may be repeated in a few months if the filling out process has been insufficient.

The type of graft used after excision or after the relaxation of burn scars depends on the area to be grafted or varies with the choice of the surgeon. In my work I seldom use either the Reverdin graft or the true Ollier-Thiersch graft in covering operative defects made in burn scars, as these grafts are too thin for stable repair.

In many clinics the so-called split graft or dermatome graft, which is a thick Ollier-Thiersch graft, is the only type of graft used for covering practically every raw surface, either granulating or freshly made. This is unquestionably a very useful type of graft and I frequently use it, but it should not be used on every occasion even if it can be easily cut with a Padgett-Hood dermatome. I find small deep grafts most useful especially in grafting relaxation incisions in regions covered by clothing, and I also frequently use whole

thickness grafts on such areas as the face, the hands, the feet and in other exposed positions.

Skin Flaps.—A skin flap is a mass of tissue which is attached at some portion of its periphery or base by a pedicle through which it receives its blood supply, and it can be shifted only as far as its pedicle will allow.

A skin flap is made up of the whole thickness of the skin with as much of the subcutaneous tissue as may be desired and is used for the repair of those areas which, in order to obtain a resistant elastic healing, require thicker tissue than simple skin grafting will supply.

There are three basic methods of using flaps, and innumerable modifications of each method:

1. The French method of advancing or sliding flaps from adjacent tissue after undercutting. In this there is little if any twisting of the pedicle.
2. The Indian method, in which the flap is obtained from neighboring tissue and is placed in its new bed by more or less twisting or rotating the pedicle.
3. The Italian method, in which the flap is obtained from a distant part, say the arm. The pedicle may or may not be twisted.

Flaps may have a single pedicle, or the pedicle may be double. The flap may be transferred to its new bed immediately after it is raised, or the transfer may be delayed, which means that in order to assure its circulation some time may be allowed to elapse between the formation of the flap and its transfer. The flap may be placed directly on the defect prepared for it, in one stage either at once or after a necessary delay. The transfer may be in more than one stage in those instances in which, on account of the distance of the defect from the tissue chosen to fill it, it becomes necessary to grow that tissue into another part and subsequently carry it on that part to the defect. Flaps may be carried in this way almost anywhere on the body. The area from which a flap is raised may be closed by sutures, or, if this is not possible, the defect may be skin grafted.

There are certain scars, which may be excised and which cannot be closed by suture, and where, for instance on the face, a skin graft would be unsatisfactory on account of color or the possibility of subsequent pigmentation. In these cases the repair may often be most satisfactorily made by the rotation of flaps of skin and the required amount of subcutaneous fat from adjacent tissue.

In the repair of any part which has been lined with skin or mucous membrane by means of a pedicled flap, it is necessary in order to prevent future scar contracture to line with epithelium the portion of the flap which is to replace this part. In order to accomplish this the flap may be folded on itself in one of several ways, or the portion to be used may be grafted with skin or mucous membrane, as the requirements may be.

The tubed flap is probably the most generally used of the delayed transfer double pedicled flaps. It was devised by Filatoff in 1916 and independently by Gillies in 1917. The method of its formation is well known.

TYPES OF SCARS

"Pain" is a symptom which may be found in every type of scar but which fortunately is fairly unusual in old scars. Burn scars may be painful, and the pain in certain instances is sufficiently severe to cause loss of function or to incapacitate the patient even though the scar may be quite small. Larger scars of all types may also be painful, but in these cases it is often possible to demonstrate localized points of hypersensitiveness, either single or multiple. The pain may be caused by inflammatory changes, by scar pressure on nerves or by the formation of neuromas on the small skin nerves. I have seen cases in which the pain in the scar increased in intensity as time went on, but more often the pain tends to become less severe with time and ultimately may cease as the scar matures and softens, so a period of observation is advisable before radical treatment is indicated. The treatment is simple and effective. If the painful scar is small, it should be completely excised and the wound accurately sutured. In the larger scars the painful point or points should be carefully plotted out, and then these areas should be completely excised down to normal tissue, preferably in the shape of an ellipse, and the wound closed with sutures. Sometimes a single painful point may cause the entire trouble in a large scar, and complete relief will follow excision.

Depressed Scars.—Depressed scars following burns are usually the result of third degree burns in which there has been considerable loss of tissue. The basis of all methods of dealing with depressed burn scars, when excision is possible, is excision with closure of the soft parts in layers to fill out the defect.

Some depressed burn scars are adherent to underlying tissues such as muscle or bone and, besides being objectionable in appearance, cause pain, deformity and interference with function. If possible these scars must be completely excised and the defect repaired by closing the soft parts in layers or by flap shifting. When the scar is long and narrow and depressed and adherent, as they are sometimes seen on a leg or arm following a steam radiator burn, the deeper portion of the scar may be used as a buttress over which skin and subcutaneous tissue is closed. This is the method of Poulard, and I find it most useful. The scar is outlined with an incision, which is carried down to normal tissue, and the margins are undercut. The epithelial surface of the scar inside the outlining incision is carefully dissected off, and then the tissues beyond the scar, which have been undercut sufficiently, are advanced over the scar buttress with deep sutures of waxed white or black fine silk or cotton, and the skin is closed with on-end mattress sutures of 4.0 or 5.0 single filament nylon, or with horsehair. This will eliminate the depression and result in a narrow unobjectionable normal level scar.

After the excision of a depressed scar, which can be closed by suture, it is sometimes necessary, in order to fill out the depression, to roll in flaps of adjacent subcutaneous fat whose pedicles are either below or are attached to the skin itself. These are advanced and sutured into proper filling position, and then the skin is closed.

If a depressed scar is large and cannot be repaired after excision by closure of the adjacent soft parts, it may be necessary to bring in a pedicled flap of skin with sufficient subcutaneous fat either from adjacent tissue or from a distant part to fill the defect. Another method is to make a delayed transfer double pedicled flap of skin and subcutaneous tissue and shift the tissue from one or both sides of the defect into the defect, thus filling it out. If the lateral defects cannot be sutured, a split or whole thickness graft may be used. If the depressed scar is large, soft and movable it may be improved by gradual partial excision, by rolling in pedicled flaps of adjacent subcutaneous fat, by undermining and inserting a fat and fascia graft or by inserting a dermal graft. In suitable cases autogenous carti-

lage or preserved icebox cartilage cut to pattern or diced, as suggested by L. A. Peer in 1943, may be used to fill out a depressed scar of reasonable size. In my experience the use of foreign bodies or paraffin for this purpose is contraindicated.

Extensive smooth (skin level) scars may follow burns on any part of the body and, when they are soft and movable, are frequently best left alone, especially if they are in inconspicuous situations. Should removal be desired, one or more of several methods may be employed: excision followed by skin grafting; the shifting in or the rotation of a flap of skin and subcutaneous tissue; gradual partial excision.

Large, thick contracted adherent scars are a common type and may present difficult plastic problems. They are often of long duration and may vary in thickness from that of the normal skin to 2 to 3 cm. or more. They may be found in any location on the body and may be completely healed, the surface, especially in the thickest portion, being covered with a scaly horny material, and these areas tend to crack open from time to time. In other instances, even after many years, there may be one or more chronic ulcerated areas on the most contracted portion of the scar which have never healed, and sometimes malignant degeneration may take place in these areas.

Contracted scars often seriously interfere with function and in the growing period check development or distort the involved portion of the bony framework. If they occur after the growing period has passed, then the lack of function caused by the scar contracture on such bones as the humerus or femur will sometimes cause atrophy from disuse.

These scars may be very extensive, and it would be poor surgical judgment in many instances to attempt complete excision because of the impossibility of obtaining sufficient skin to cover the defect made by the excision. In these cases the use of relaxation incisions is the method of choice as the relaxed scar between the incisions, when it changes its character, can be utilized in the final healing.

Unstable scars are those which usually follow extensive loss of the whole thickness of the skin and are most frequently found in cases in which the tissue losses involve the entire circumference of a limb or a large area over the vault of the cranium. These are in most instances too extensive to consider complete excision. The healing in the majority of cases has been by cicatrization, but sometimes the condition follows inadequate or unsuccessful grafting.

The scars are paper thin and are very unstable and may be entirely healed one day and, without apparent cause or following very slight trauma, on the day following may ulcerate in many places. The history is slow healing and then breakdown, and this may go on for years, during which time the patient may be completely incapacitated.

The first paper on the use of relaxation incisions followed by skin grafting in the treatment of unstable scars was published by J. S. Davis in 1917. It has been my experience that this is the only satisfactory method of dealing with this type of scar.

The method is as follows: The incisions should be marked out and should extend the entire length of the scar into the normal skin above and below it and should expose normal tissue even if necessary to go through the deep fascia. Many of these unstable scars are on legs and arms, and the incisions in these cases should not be made immediately over bone. For a leg or an arm or thigh, three incisions running parallel to the length of the limb are usually sufficient. The incisions may or may not go through the ulcerated areas. After the relaxation incisions have been made, the ulcers on the scar tissue between the incisions rapidly heal, and the scar itself changes its characteristics and becomes stable and strong. The wounds made by the incisions may be grafted immediately if the tissues are ready for grafting; otherwise after a few days when the granulations have formed, the grafting may be done. I use small deep grafts or thick Ollier-Thiersch (split) grafts for grafting the relaxation defects, depending on the conditions.

Keloidal Scars.—It is difficult to differentiate the thickened, hard, red, tender, itching painful type of keloidal scar, which not infrequently follows burns, from a true keloid, and doubtless many of them are true keloids. Be that as it may, this type of scar sometimes follows burns, particularly those caused by chemicals, and it is a particularly difficult type to treat. Up to this time no specific treatment has been devised. Sometimes carefully given x-ray or radium therapy will blanch out and soften these scars and make them more comfortable, but frequently this type of treatment is unsatisfactory. These scars are often found on the backs of the hands, on the ears, on the cheeks and on the neck, the chest and the trunk. They usually follow burns which have destroyed the full thickness of the skin and which have been slow in healing. Why they occur in some burns and not in others, or why they occur in some areas on the same patient and not on others, although the healing has apparently been the same, I do not know. If the keloidal scars are not causing loss of function, time and plenty of it should be allowed to elapse before attempting any operative procedure.

In keloidal scars of reasonable size, for years I have been giving preliminary irradiation and then by the process of gradual partial excision removed the scar, keeping the sutures always in the scar area and in time finishing up with a comparatively narrow scar. Irradiation is also given each time after the sutures have been removed. Whether the preliminary irradiation does any good or not is a matter of question, but I have continued to use the method with fair satisfaction. In large areas on the face and hands excision with skin grafting or flap shifting may be necessary, but the results cannot be counted on. Complete excision with closure is usually followed in the true burn keloid by a bigger and better keloid. Removal by the endotherm followed by irradiation is sometimes done, and successes have been reported. Capillary drainage is usually unsuccessful. In fact, there is no completely satisfactory treatment of this type of scar.

REGIONS

As burn scars and deformities are found on all parts of the body, it might be helpful to consider different regions and briefly to mention methods which might be useful in the treatment of some of the lesions in these regions. As these scars may vary in each case and in each location, the methods are offered simply as suggestions, as each case must be studied out individually as to the best method of repair. In all of these operative cases much help can be obtained by the proper use of suitable immobilizing dressings and appliances, which should secure the part in the desired position in comfort and at rest.

Head.—Scalp: Burn scars ot the scalp are at times very difficult to deal with. If the scar involves a large part. of the scalp, is thin, is unstable and is tightly stretched on the skull, sometimes "tunnel grafts" of whole thickness or split thickness skin are effective in starting lines of stable skin. At other times relaxation incisions, with grafting of the defects thus made, promise the best result; in other instances removal of the scar with skin grafting, either split or whole thickness, depending on the conditions. When the scar involves only a portion of the scalp and a considerable amount of hair bearing skin remains, it is often possible to shift pedicled flaps of this hairy skin into strategic areas of the scar and thus make it easier to cover the hairless scar with a better arrangement of the hair.

In the midst of extensive old burn scars of the scalp, masses of closely adherent necrotic bone may be found. In these cases the bone should be removed down to normal bone or, if it includes both tables, down to the dura, and then the repair should be made. A slower method is to bore numerous holes through the bone to allow the formation of granulations and then, when the granulations have formed, to graft.

Forehead: Contracted burn scars of the forehead, with or without scalp involvement, will often cause abnormal raising of the eybrows and even of the eyelids themselves. In order to overcome this, a relaxation incision completely through the scar is made at the selected level transversely across the forehead from one temporal region to the other, the tissues are loosened and into the defect thus made a single whole thickness graft or thick split graft is sutured. In suitable cases the scar may be completely excised and the defect filled by a whole thickness graft or a thick split graft. If a scar in the temporal regions is hairless and unsightly, a flap of hair-bearing skin may be brought forward successfully from the scalp back of the ear to relieve conspicuous baldness in front of the ear.

Eyelids: Frequently ectropion of one or both eyelids, on one or both eyes, due to burn scar contractures occurs. In my experience, thin hairless whole thickness grafts are to be preferred, either from the normal upper lid, from the inner side of the upper arm or from the prepuce. Thick split grafts may be used, however. Adhesions should be made between the lids at several points to put them on the stretch before the grafts are sutured into position. Sometimes a flap from the temporal region or forehead is to be preferred for the repair of the lower lid. Eyebrows and eyelashes are often destroyed by burns and cause pronounced disfigurement. After the contractions are relieved these can be replaced by transplanting free grafts or pedicled flaps of hair-bearing skin by special technics. The complete restoration of eyelids and also the formation of sockets for artificial eyes is necessary in certain severe burns, and this may be accomplished by the use of suitable flaps and grafts.

Ears: Burn scars of the ears vary greatly. The cartilaginous framework may be practically intact and covered with a thin adherent scar. In other instances there may be every grade of cartilage destruction. Sometimes the lobule of the ear is fused with the scar on the adjacent portion of the cheek. The cartilaginous framework may be completely embedded in scar and closely adherent to the skull. In some instances simple relaxation with Z-plastics or skin grafting may be all that is required, but in others the complete reconstruction of the external ear is required, with cartilage transplantation, skin grafting and flap shifting as may be necessary in each case. Keloidal scars are not uncommon on ears and, after considerable time has elapsed without improvement, should be excised and accurately grafted with split thickness grafts.

Cheeks: Contracted burn scars of the cheeks are frequently found, and often the scar will involve the eyelids and mouth as well. In repairing such deformities the contracted scars must be completely removed in order to relieve the contractures, and the repair made by means of large whole thickness grafts or of flaps from a distant part or by the rotation of skin from the neighborhood if such skin is not infiltrated with scar. Usually when these contracted scars are excised there is a defect of varying depth left and it is inadvisable to use a split graft, as it is not thick enough. Sometimes the whole cheek must be reconstructed, and in these cases a lining must be provided for the flap with which it is done.

Nose: Many deformities of the nose result from burns, and the repair presents a considerable problem, as frequently the skin of the forehead and adjacent cheeks is also involved in the scar. In many instances a forehead flap, either lined or unlined as may be necessary, presents the best means of making the repair, as the skin of the forehead matches the nose in color quite well. Sometimes a flap from the arm is prepared and transferred, or a flap from the abdominal wall is grown onto the hand or wrist and then brought up to the nose. When flaps are unnecessary I frequently use a free graft from the skin on the back of the ear, as this matches in color better than the skin from other portions of the body. Cartilage may also be necessary in repairing deformities of the nose due to burns, and this material may be autogenous rib cartilage, which is to be preferred, or preserved icebox cartilage may prove useful. The cartilage may be implanted into the flap some time before it is turned down on to the nose, or it may be put in after the flap has grown into its new position.

Mouth: The most common deformity of the mouth due to scar contracture is ectropion of the lower lip, and this is usually associated with burn contractures involving the chin and neck. The relief of this type of contracture calls for either whole thickness grafting or, in suitable cases, thick split grafts or flap shifting. Another deformity caused by scar contracture is the so-called "pucker string contracture" around the mouth. When the patient opens the mouth there may be one or two or even three lines of scar pull running from the nasolabial folds on each side downward and across the chin, and frequently this scar also involves the upper lip. The best procedure to relieve this condition is by Z-plastics at strategic points. Another deformity of the mouth due to burn scar contracture is microstomia. In some instances the opening between the lips has contracted down to pencil point size, and the problem of feeding the patient is a serious one. In these cases the opening is enlarged along the normal lip level to a little more than normal mouth size, the mucous membrane is sewed to the skin or remains of the vermilion border, and the angle of the mouth on each side is made with a flap of skin or mucous membrane.

In cases in which the burn has destroyed the angle of the mouth on one side and the adjacent portion of the cheek and there is a contracted scar, it is neces-

sary to reconstruct that portion of the cheek with a lined flap and incidentally to make that side of the mouth of normal size. Sometimes it is necessary to reconstruct both lips as well as the soft parts of the chin. In such cases lined flaps must be brought in to furnish the required tissue. Scar contractures involving the angles of the mouth, displacing them either too high or too low, may be corrected by Z-plastics.

Remarkable illustrations of the use of double pedicled flaps of scalp in the repair of burn defects of the face have been shown, but to my mind in most instances the procedure is poorly thought out.

Neck.—Burn contractures of the neck are quite common. They may be massive and obliterate the entire contour of the neck and chin, or they may be scar bands or bridles. Frequently the angles of the mouth, the chin and the lower lip are also involved in these scars. In the massive type the scar should be excised as thoroughly as possible, the head extended and the defect filled with a whole thickness graft, a thick split graft or with a flap, often from the back. Sometimes a broad double pedicled flap may be slid up from the lower part of the neck or the upper part of the chest. It is necessary sometimes in these cases to relieve the condition in stages. In the scar band type, Z-plastics offer the best solution, and by the use of this method skin grafting or flap shifting may be avoided.

Trunk.—Burn scars and contractures are quite common on the trunk and vary greatly in their type. The extensive contracted scar which involves the anterior part of the chest and abdominal wall frequently prevents the patient from standing erect. The shoulders may be drawn toward each other, the breasts may be drawn close together, and the patient is held in a round shouldered position. In these cases the problem is not the removal of all the scar area, which in most instances is impossible (although some of the most objectionable areas may be excised), but to produce relaxation so that the body may assume an erect position without hindrance. Relaxation incisions should be made where necessary and the defects grafted with small deep grafts or any other type of graft selected by the surgeon, a considerable portion of the scar being utilized in the final repair. If the scar is comparatively small it may be either completely excised and the wound closed or excised with grafting, or, in suitable instances, gradual partial excision is the method of choice. It is not unusual to have a scar drawing the breasts toward each other. In some of these and in other areas, Z-plastics are effective. We quite often see keloidal scars on the back, the chest and trunk, and these should be treated by methods already mentioned. Sometimes unstable scars may be found on either the front or the back of the trunk, and in these cases relaxation incisions with grafting is the method of choice.

Upper Extremity.—Axilla: Frequently associated with burn scars of the trunk one finds the axillary regions also involved in the scar contractures. The involvement of the axilla may vary between a shortening of the anterior or posterior axillary fold to complete obliteration of the axilla and sometimes the fusion of the arm to the chest wall. When these adhesions are of long duration, separation of the arm from the chest wall must be done carefully as the humerus, through atrophy of disuse, may become brittle and, if moved too rapidly or without great care, may be fractured by the manipulation.

In the reconstruction of the axilla, small deep grafts should not ordinarily be used, but either large split grafts or whole thickness grafts or, if suitable adjacent tissue is available, pedicled flaps should be used. Tubed flaps are also very useful in certain cases. In certain webs involving the axilla, Z-plastics are of great use.

Arms and Hands: Many burn contractures are found on the arms and hands. The joints may be involved and immobilized in abnormal positions. The contractures may be as constricting bands or grooves in the midst of reasonably good skin, or the whole extremity may be involved and function completely lost. Many of the contractures around joints cause flexion deformities, and on the hands hyperextension with subluxation is often seen. Around joints the scar must be removed and replaced by a flap or graft as seems best. All types of flaps may be used. On hands, one frequently finds the part literally "frozen," there being practically no voluntary motion of any kind possible. Sometimes dense keloid growths will form on the hands. All of these problems are difficult to overcome. Excision with replacement with flaps of various kinds or with whole thickness grafts or thick split grafts must be done. Special splints designed for the special cases are of great assistance and should be used when required. Fingers and adjacent portions of the hand may be completely destroyed by the original lesion and the stump covered by a thin, painful scar. In such cases excision and covering with a flap or graft is indicated and, if possible, the readjustment of the stump for practical use.

Unstable scars are not unusual on the arms and forearms. Here, as always where this type of scar presents, thorough relaxation incisions with skin grafting is the method of choice. In relaxing scar bands on the entire upper extremity, including the hand, Z-plastics are of great use.

On the palms after excision of scar, whole thickness grafts or thick split grafts should be used. When flaps are used, care should be taken that there is only sufficient subcutaneous fat for the purpose required and that the flap fits fairly accurately.

Sometimes the reconstruction of tendons is necessary in old burn scars on the hands. Skin and subcutaneous tissue must first be supplied and then, through this, tendon reconstruction may be made in suitable cases.

Lower Extremities.—Burn scars and numerous consequent deformities are often found on the lower extremities. These contractures may cause flexion of the thigh on the abdomen and the leg on the thigh and often cause complete loss of function. In some of these cases areas are found in the midst of dense scar which have never completely healed. Often the scar involvement will extend from the chin to the knees. I have found that relaxation incisions with grafting is by far the most useful and effective method of handling these scars. One should gain as much as one can at the first operation and later do further relaxation and more grafting. In this way the best ultimate results may be obtained. In dense old burn contractures in the popliteal space it is advisable to divide the scar transversely as deeply as is safe, then to apply traction by an orthopedic appliance and gradually stretch out the tissues, and then make the repair with a graft or flap. In this way the muscles, vessels and nerves which have contracted on account of position are gradually stretched without injury. Unstable scars on the lower extremity are also treated by long relaxa-

tion incisions with grafting. Contractures around the ankle and foot are treated by excision and grafting, and suitable splints are also helpful. Sometimes there is an extensive tight scar on the anterior surface of the knee which prevents flexion. This scar must be excised if reasonably small and replaced by a graft or flap, or relaxation incisions must be made and then grafting done. Thick grafts or flaps should be used in areas where there is weight bearing and continuous trauma, such as the sole of the foot.

SUMMARY

My purpose in this paper is to present a brief consideration of the late plastic care of burn scars and deformities.

All types of scars may follow the healing of burns. These scars contract and cause an infinite variety of deformities.

The vast majority of these deformities must be treated by operative methods.

The most important of these methods are excision with closure by sutures; excision with lateral relaxation incisions and closure followed by skin grafting of the relaxation defects; gradual partial excision with closure; relaxation incisions in the scar itself with skin grafting; the use of Z-plastics; the use of skin grafts of various types; the use of pedicled flaps.

In many instances it is impossible to excise the scar completely on account of its size and location. In these cases the utilization of scar itself or scar infiltrated tissue in making the final repair is essential.

In utilizing the scar itself in making the repair, three methods will be found useful, either alone or in combination. These are gradual partial excision with closure; relaxation incisions in the scar itself with skin grafting; Z-plastics.

Each of these maneuvers is of great value, and none of them have been employed as generally as they should be.

As a matter of fact, the use of scar tissue in making the final repair of an extensive burn scar with deformity has been greatly neglected, and this important source of material has in many instances been eliminated even when donor sites for grafts have been scarce.

In some clinics, where skin grafts are necessary in relieving scar deformities, the split graft, cut with the Padgett-Hood dermatome, has superseded all other types of grafts for all purposes, although frequently whole thickness grafts and grafts of other types would be more suitable.

All types of pedicled flaps may have to be used in relieving the deformities caused by burn scars, but, as in the use of grafts, there is no one type which should be used to the exclusion of all others.

Contracted scars may seriously interfere with function and during the growing period may check bone growth or distort the bony framework.

Each case must be studied out as an individual problem, as there are seldom two with exactly the same involvement.

None of the operative maneuvers found most useful in dealing with old burn scars and deformities are new. There is no single procedure which is the method of choice and which is applicable for the relief of all types of cases.

The application of adequate special splints following operation in many of these cases is very helpful.

It is to be hoped that, with the more general use of the latest methods of treating burns, which stress the prevention of infection and where early skin grafting is employed, deformities will become less frequent.

The operative care of burn scars and the deformities caused by them should be in the hands of surgeons who have a thorough knowledge of how to deal with scar or scar infiltrated tissues and who understand the possibilities of their utilization.

701 Cathedral Street.