Complications of Burns *

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WHEN A PERSON is subjected to thermal injury, the primary object of the attending physician is to combat the tremendous physiologic changes that occur in an effort to preserve the life of the individual. Aside from these widespread changes in body physiology occurring immediately because of the thermal injury, complications appear at varying periods of time following the initial trauma that may be a result of the thermal injury itself or an end product of the therapy administered.

At the Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas, from 1950 through 1956, over 1,000 patients were treated for thermal injury of varying degrees. In general, these injuries were rather extensive. The burns involved all parts of the body and were caused by a variety of agents, including flame, hot water, steam, electricity, chemicals and contact with heated objects, causing a wide distribution of depth of burn. In this large number of cases, the complications of the thermal injury and the therapy used are considered representative enough to warrant discussion as to etiology, course, and possible treatment. However, the records, particularly those for the earlier years, are not accurate enough to indicate a reliable incidence.

The purpose of this discussion is not to infer any impression of accuracy as to frequency of occurrence, but to indicate the types of complications that may occur. Since a long-term follow up has not been possible, the treatment and results noted are those of the early postburn period.

Burns of the Ears

When the ears are involved, as is usually the case in a burn of the face of any extent, there is always the possibility that the cartilage will be damaged. This involvement of the cartilage may be caused by a deep thermal burn or it may result as a secondary complication of a second-degree burn of varying depth. In the clinical experience at this Unit, either at the initial time of examination or for varying periods thereafter, it was not possible to determine which of the burned ears would be involved with this chondritis or, when involved, what the end result would be without surgical intervention. The clinical appearance is usually manifest by a swelling of the helix and and antihelix with local heat and tenderness. Characteristically a constant, rather severe pain is present in the ear which may not be effectively relieved even by opiates. Figure 1 shows the typical appearance of a full-blown chondritis prior to surgical intervention and the end result following surgical intervention.

The exact cause of this chondritis is not clear, although several opinions have been set forth. It is not known why the infection should occur in some burns and not in others when the extent and depth of the burn appear to be similar. Table 1 shows the cases treated each year from 1950, through 1956, and the distribution of the chondritis as it occurred.

Although the epithelial covering of the ear may be completely destroyed over the

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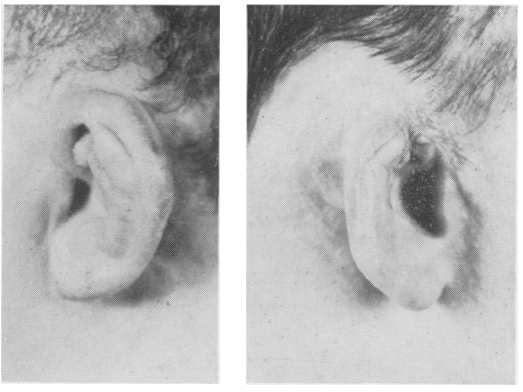


FIG. 1a. (*Left*) Swollen, tender, painful ear of a postburn chondritis. Note that the burn is otherwise healed.

FIG. 1b. (*Right*) Appearance after removal of cartilage and healing. With loss of cartilaginous support the soft tissue shrinks markedly.

helix and expose cartilage, this does not necessarily indicate that a chondritis will develop. If split-thickness skin can be placed over the area during early therapy, in some cases the clinical evidence of suppurative chondritis will not appear. Likewise, burns that initially appear to be superficial second-degree burns and later heal without evidence of a full-thickness destruction of the skin may subsequently develop a full-blown suppurative chondritis requiring surgical intervention. It is by no means an exclusive rule that the deeper the burn the greater the chance of a chondritis developing, and certainly chondritis is not frequent enough to warrant early prophylactic excision of the cartilage.

The only mode of treatment proved to be of value in this Unit was surgical removal of all of the nonviable and infected cartilage of the helix, antihelix, and other portions of the ear, if necessary, through an incision involving the entire margin of the helix (Fig. 2). Cartilage that is edematous, often soft, necrotic, and frequently granulating, is removed by sharp and blunt dissection and the two layers of the split ear are held apart by a single or double layer of dry, fine-mesh gauze (Fig. 2). Wet saline soaks are then applied to the entire ear to be changed at varying intervals.

The dry, fine-mesh gauze is changed daily until spontaneous healing occurs. It may be necessary to debride the cartilage on more than one occasion. This treatment results in relief of the often excruciating and almost constant pain characteristic of this infection, but it does not prevent a marked deformity of the ear resulting from the loss of the cartilaginous support. There does not appear to be any appreciable interference with auditory acuity as a result of this loss of substance. As a cosmetic procedure, reconstruction of the ears or the use of a plastic prosthesis proved satisfactory for those patients who desired it.

Another complication of burns of the face occurring quite frequently is ectropion of the lid. Table 2 shows the occurrence and the distribution of involvement in burned patients treated since 1952. Full-thickness loss of the epithelium covering the eyelids is quite likely to occur when the face is involved in a thermal injury of any appreciable depth because the skin of the eyelids is quite thin. When spontaneous healing occurs in a deep second, or a mixture of second and third degree, or there is graft coverage of a third-degree burn, it is probable that scar tissue contracture will cause an ectropion (Fig. 3). The upper lid is somewhat longer and more redundant than the lower lid; therefore it requires a burn of somewhat greater depth and extent to produce enough scarring to retract

TABLE 1. *Chondritis of Ears, 1950–1956*

Year	Bilateral	Right Only	Left Only	Total
1950–51	3	1	1	8
1952	13	5	0	31
1953	8	1	1	18
1954	3	1	3	10
1955	6	0	1	13
1956	6	6	1	19
	39			
Total	78	14	7	99

the upper lid to a point of functional impairment. Some cosmetic deformity usually results.

That portion of the face over the malar eminence and the nasolabial fold may undergo considerable contracture while healing either from a second- or third-degree burn, and this condition together with the original retraction of the lower lid may produce additional functional impairment and a more marked cosmetic deformity. The tarsal plate itself is rarely involved except in cases of localized deep burns, such as phosphorus burns, chemical burns, and hot contact burns.

In the initial or early postburn treatment of a patient, the ectropion itself is of no consequence as far as cosmetic appearance is concerned. In fact, the longer any surgical intervention can be delayed and allow scar tissue maturation to occur, the better will be the cosmetic result. This course of events should be followed if at all possible. The degree of ectropion which occurs, particularly when upper and lower lids are involved simultaneously, often results in drying of the conjunctiva and cornea due to the constant leaking of tears over the margin of the everted lid.

A corneal ulcer may develop rapidly and become secondarily infected, thereby causing permanent visual damage. When it appears that there is danger of an ectropion, it is mandatory that a tarsorrhaphy be done (Fig. 4).

If an ectropion has occurred, it should be corrected if any danger to vision exists. In correcting the ectropion surgically (Fig. 5) the primary aim is a good functional result, namely, the preservation of the moisture to the conjunctiva and cornea. A secondary consideration is the cosmetic result. During the surgical correction, it is not necessary to remove an ellipse of skin or large amount of tissue although small areas of deep scarring that would involve the margins of the graft may be excised without danger. Curved incisions are made two or three mm. from the lid margins after they have been sutured together with two or three interrupted 000000 black silk sutures. The incisions in the lids themselves are carried down by sharp dissection to the musculature of the lid. At this time it will be noted that, with the separation of the scar tissue by the sharp dissection, there is a wide gaping of the incision and complete relaxation of the lid. Split-thickness skin grafts are then applied to the defects and are held in place with a stent dressing of 000000 black silk sutures.

Dressings are changed initially on the third day and the lid margins are released on the sixth day. The eyes are irrigated for

TABLE 2. *Ectropion, 1952–1956*

Year	Bilateral	Right Only	Left Only	Total
1952	8	3	2	21
1953	3	0	0	6
1954	4	0	0	8
1955	0	1	3	4
1956	5	0	1	11
	20			
Total	40	4	6	50

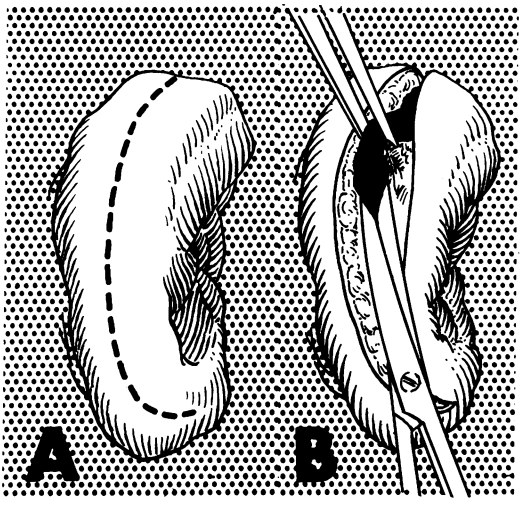


FIG. 2a. Removal of cartilage of ear. Line of incision is along entire curve of helix.

FIG. 2b. The non-viable, infected cartilage is removed.

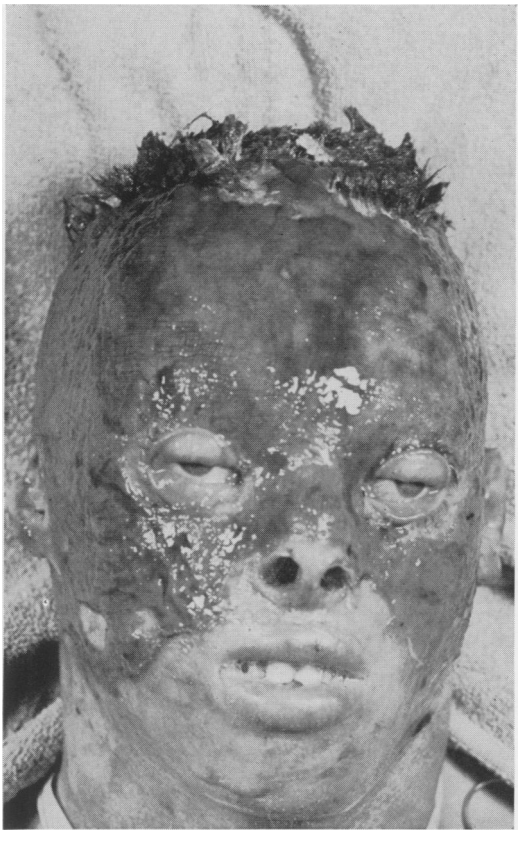


FIG. 3. Severe bilateral upper and lower lid ectropions from third-degree burns. Fortunately only superficial corneal ulceration had occurred. On arrival at the U. S. Army Surgical Research Unit, bilateral tarsorrhaphies were done and grafts placed on the lids.

as long a period as required postoperatively. Local antibiotic ointments are applied. Figure 6 shows the postoperative appearances of a bilateral correction. In this early operative correction of an ectropion, relatively large split-thickness grafts are applied. The incision extends from the inner to beyond the outer canthus of the eye. A large degree of overcorrection of the ectropion is carried out because of the continued and progressive scar tissue contracture that occurs during this post-burn period and for many months thereafter. If this condition is not adequately overcorrected, invariably a recurrence of the ectropion will develop (Fig. 7).

Hand Complications

Probably the most disheartening complications to both patient and physician are those developing as the result of thermal injury to the hands, particularly the extensor mechanism. In the large number of burns seen at this Unit, the flexor surface of the hand is unique. With the exception of the distal one-half of the flexor surface of the digit and margins of the thenar and hypothenar eminence, the palmar surface of the hand has rarely sustained full-thickness involvement unless the burn was produced by a chemical, by electricity, or by direct contact. This has been true even in burns covering 98 per cent of the body surface. However, the extensor portion of the hand does not escape such injury, and frequently it suffers full-thickness loss when the burns are of small extent or when initially they are thought to be superficial in depth. Infection frequently occurs in the

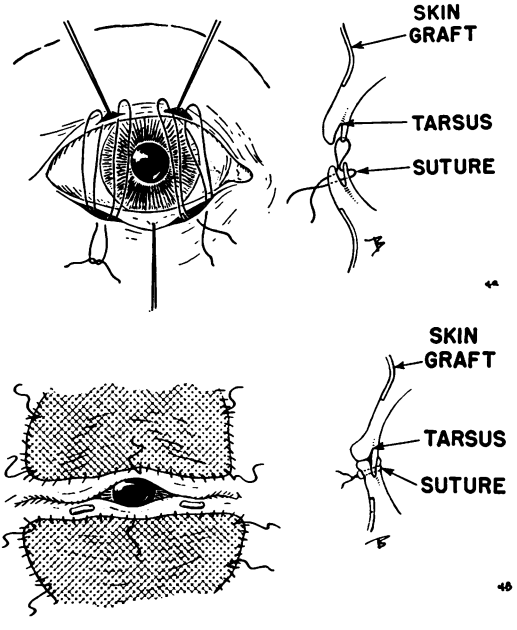
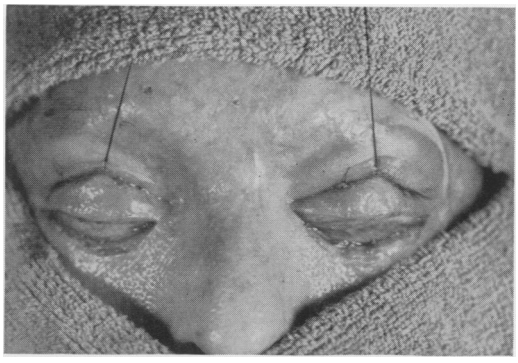


FIG. 4a. (*Upper*) Tarsorrhaphy. Fine sutures are used with the knots tied so as to avoid the cornea.

FIG. 4b. (Lower) Central aperature for vision with a medial and lateral for irrigations.



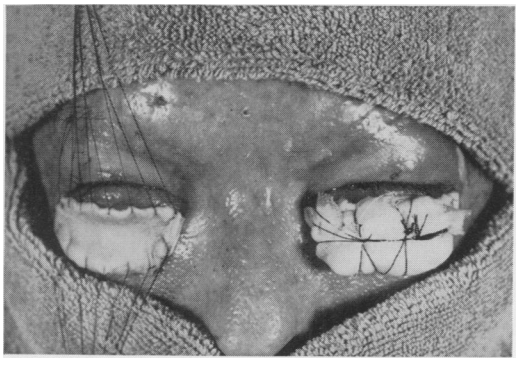


FIG. 5a. (*Upper*) Operative correction of bilateral lower lid ectropion. Incisions should be placed closer than shown here.

FIG. 5b. (*Lower*) The split graft works as well as a full thickness and takes much better.

area. Since the critical structures are quite close to the skin surface and have practically no subcutaneous tissue for protection, functional loss occurs frequently as the result of infection even though the structures may have escaped permanent damage from the thermal injury.

In general, the digital complications may be divided into three anatomic categories exclusive of the tendons on the dorsum of the hand. These categories are: loss of the middle extensor slips over the proximal interphalangeal joints, burns extending into the joint, and joint capsule fibrosis (Table 3). In the normal functioning of these joints, the capsule and collateral ligaments are in a state of full expansion when the joint is fully flexed. Therefore, it is obvious that any shortening of the fibers caused by thermal injury and subsequent healing will limit markedly the ability of the patient to flex these joints. Surgical intervention to correct this situation is to be avoided. If persisted in, active and prolonged physiotherapy will result in a considerable return of function. In conjunction with physiotherapy, further aid in restoring function is obtained by using dynamic splints with spring, or elastic, traction-resistance mechanisms.

Capsulotomies of the proximal interphalangeal joints are useless because they cause loss of the lateral stability of the joint. A capsulotomy of the metacarpophalangeal joint, however, does not appreciably affect the lateral stability of the joint but infrequently results in a good functional joint. Failure of surgical intervention to produce functional improvement is primarily a question of the motivation of the patient. The surgical division of the shortened fiber can merely produce a relaxation of the structures binding the joint. Thereafter it is a question of early, persistent, vigorous and conscientious effort on the part of the patient to maintain the motion of these joints. Unless the patient is extremely well motivated, the skill of the surgeon is of no avail and the operative procedure is doomed to failure from a recurrence of the scar tissue fibrosis.

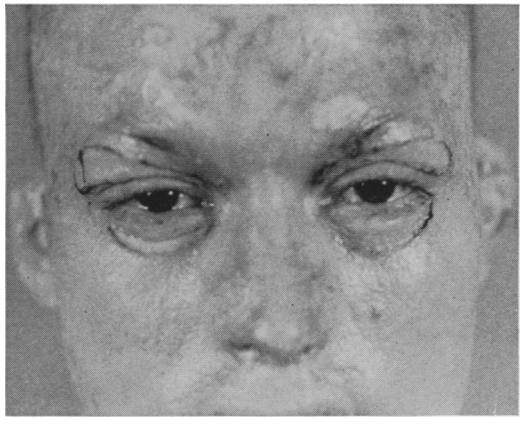


FIG. 6. Postoperative appearance of ectropion corrections. Excellent functional and cosmetic result.

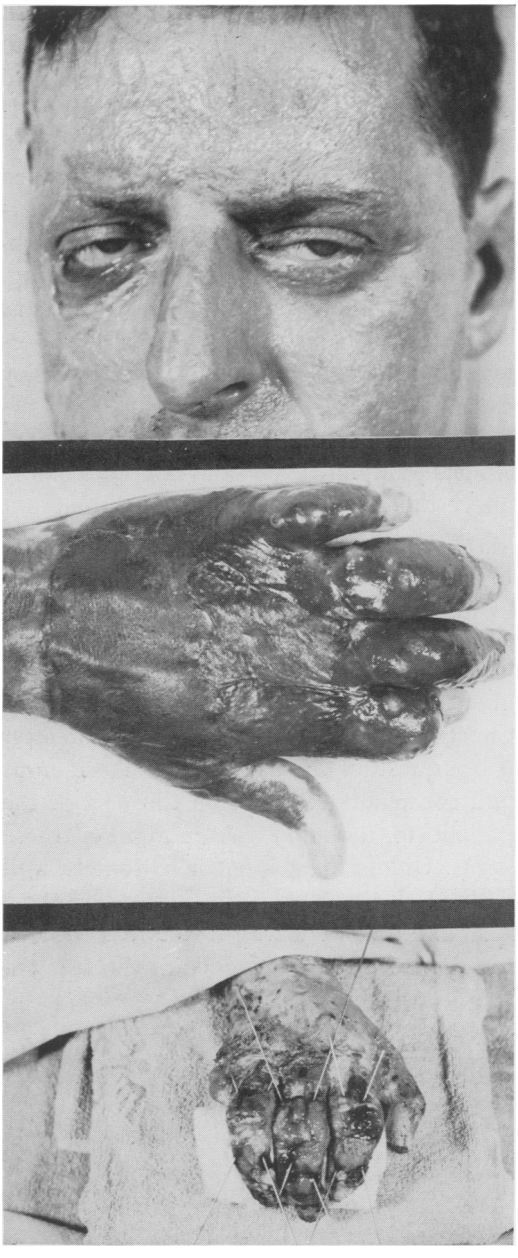


FIG. 7. (*Upper*) Recurrent ectropion. Continued scar retraction graft and adjacent area of deep second-degree burn in malar area caused this poor result of ectropion correction.

FIG. 8. (*Center*) This deformity was secondary to destruction of the extensor mechanism by infection. There is flexion of proximal interphalangeal joints and extension of metacarpophalangeal and distal interphalangeal joints with capsule fibrosis.

FIG. 9. (*Lower*) Pinning the joints. The wires are cut so that 2–3 mm. remains exposed. The position of 30–40 degrees flexion is best.

With the extremely thin skin over the dorsum of the fingers and its relatively exposed position in any thermal injury involving the hand, invariably the important structures beneath the skin are involved. The middle slip of the extensor tendon is the most important of these structures from a functional standpoint. As the extensor tendon of the digit passes over the metacarpophalangeal joint it divides into three portions. The medial and lateral divisions pass beside the proximal interphalangeal joint at approximately the level of the midpoint of the joint, and the middle slip passes over the midportion of the dorsum of the interphalangeal joint. All three join near the distal interphalangeal joint. The middle and medial and lateral slips are joined to each other by fibers of the "extensor hood" which are thinner than those of the extensor mechanism proper.

With destruction of the middle slip by direct thermal injury or by secondary infection, a "buttonhole" type deformity occurs. Flexion of the proximal interphalangeal joint is caused by the action of the flexor tendons and the medial and lateral slips of the extensor tendons that are now shifted below the midpoint of the interphalangeal joint hold this position.

A constant flexion deformity results and it may become progressively more severe unless it can be prevented. Reconstruction of the extensor mechanism is impossible

TABLE 3. *Complications of Digital Burns, 1950–1956*

Year	Loss of Middle Extensor Slips	Burn into I-P or M-P Joint	Joint Capsule Fibrosis
1950	4	1	3
1951	4	4	2
1952	2	1	1
1953	5	2	8
1954	0	3	3
1955	1	3	4
1956	5	5	5
Total	21	19	26

because there is no tendon sheath present in this area and no subcutaneous tissue to act as a buffer between the fascial planes and a thin, flat tendon not suitable for suturing. Figure 8 shows the result of the loss of the middle slips of the extensor tendons of the proximal interphalangeal joints and fibrosis of the joint capsule of the metacarpophalangeal joints.

Since reconstruction of the extensor mechanism is impossible, the only procedure left is an attempt to prevent further deformity and place the joint in the position of maximum function. This can be accomplished by a fusion of the interphalangeal joint at 30–40 degrees flexion; and, as the function of the metacarpophalangeal joint remains or returns, a functional grasp can be maintained.

Fusion is accomplished by inserting small calibre Kirschner wires into the proximal and middle phalanges of the involved digits and passing them through the proximal interphalangeal joints. This surgical procedure is demonstrated in Figure 9. After the pins have been placed through the digits, they are cut off near the surface and grafting is carried out. These pins are allowed to separate spontaneously. This situation usually occurs in approximately six weeks, at which time fusion has occurred.

It has not been found necessary to remove the cartilage of the joint. A pyarthrosis may develop and become of such severity that removal of the digital pins is indicated. Following the resolution of the pyarthrosis, the joints may be pinned a second time. Figure 10 shows the result of pinning of the proximal interphalangeal joints of severely burned hands. Thermal injury may be of such depth in extensive and deep burns of the hands that not only is the extensor mechanism destroyed but the joint capsule is also destroyed. Such destruction is frequently associated with periosteal burns of the dorsum of the middle and

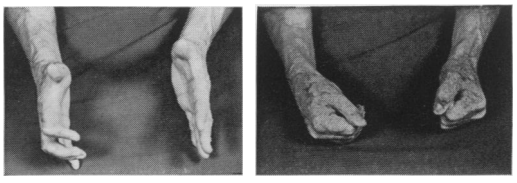


FIG. 10a. (*Left*) Function of hand after pinning proximal interphalangeal joint. The index, middle and ring fingers of right hand were pinned at 40 degrees flexion. Figure 10a. shows hands with moving joints fully extended.

FIG. 10b. (*Right*) Moving joints fully flexed. Note full range of metacarpophalangeal joints with fusion of proximal interphalangeal joints and good functioning hand.

distal phalanges and loss of the nails. It is obvious that no function reconstruction can be carried out when the extensor mechanism and the joint capsule are completely destroyed. Consequently, fusion of the joint is mandatory. This is carried out in the same manner as that described previously for the loss of middle extensor slips.

Upon exposure of the joint, particularly in the case of periosteal burns of the adjacent phalanges, skin coverage is quite difficult. Sometimes it is possible to drill the phalanges in such a way that the granulations grow up from the marrow cavity and split-thickness skin grafts can then be applied. Even after this procedure, early skin coverage over the exposed joint is prevented because pyarthrosis usually develops in varying degrees in the exposed joint. After complete destruction of the cartilaginous surfaces by the infection, usually granulation tissues will form, fusion will occur, and split-thickness skin coverage may then be carried out. Even in burns of great extent, the patients are frequently ambulatory a long time before these exposed joints finally heal. It is not unusual to see a patient two and three months after he was burned with a draining sinus from a previously exposed interphalangeal joint.

If this pyarthrosis causes an osteomyelitis of an adjacent phalanx, it is sometimes necessary to amputate the finger. However,

because of the obvious loss of function usually occurring in adjacent digits, it is of prime importance to maintain as much length of each individual digit as possible and to save as many digits as possible. Consequently, the smoldering pyarthrosis is usually given the opportunity to burn itself out rather than to be corrected by curettement. Shortening of the phalanges and subsequent shortening of the digits will cause increased amounts of deformity and may possibly result in amputation of the digit.

It is of extreme importance to keep burned hands elevated to assure adequate lymphatic drainage. Dependency cannot be allowed for any but short periods until the lymphatics have reappeared in the burned area. Unless this procedure is followed, a brawny, indurated edema of the hands occurs and, in spite of intact tendon and joint mechanisms, it produces a frozen hand. Early correction of axillary and elbow contractures may be necessary in order to assure good drainage.

The physician should be aware of the immediate complications that may occur in circumferential third-degree burns of the extremities. When such burns occur, the eschar is inelastic. It does not expand with the edema of the burned area and thus causes a rather rigid compartmental compression of the structures within the limb. This results in a very effective ischemia, particularly in the upper extremity and in the lower leg from the knee down. If untreated, avascular necrosis of muscle groups and entire extremities is frequent and may result in loss of borderline burned structures. The only effective measure is relief of the compression by splitting the eschar wide, particularly at the knee, malleoli, wrist, and elbow, thus allowing the tissues to expand freely. Frequently an immediate improvement in color and temperature is noted.

Burns Involving Bone

Burns are occasionally of such a depth or location that they involve the underlying bone. This presents a difficult problem from the standpoint of skin coverage and the prevention of osteomyelitis. Bone involvement is apt to occur particularly in a localized deep thermal injury such as a phosphorus, electrical, chemical or contact burn. It is seen most frequently in the ordinary thermal burn involving the soft tissue that overlies rather superficial bone such as the skull, the anterior tibia, the malleoli, and the dorsum of the digits and hand. At the initial examination of a deep third-degree burn, it is usually impossible to say whether or not the periosteal involvement is significant enough to result in necrosis of underlying bone. It is inadvisable to remove bone thought to be involved at an early date merely as a prophylactic measure in an attempt to prevent the development of osteomyelitis. Usually by the time spontaneous separation of the eschar occurs or, as in the case of localized third-degree burns, after the local area is excised and granulations are formed, viability of the periosteum can be determined. A surgeon should be able to determine whether or not the periosteum is viable. If it is not, additional operative procedure must be performed.

Table 4 shows those cases of third-degree burns of the skull involving the cranial bones and the treatment elected. These data indicate that when the periosteum is nonviable and efforts are made to stimulate granulations to cover the exposed bone, the possibility that osteomyelitis will develop is greatly minimized. The one patient who developed an osteomyelitis of a cranial bone was admitted to this Unit late in the burn course and no attempts had been made to cover the scalp defect. Examination on admission revealed an osteomyelitis with a dural abscess.

There are two ways to hasten the appear-

TABLE 4. *Burns Involving Cranial Bones*

These result from severe burns. If granulations can be produced, serious complications are avoided.

Year	Patient	Complication	Treatment	Result
1950	H. B.	Deep electrical burn of occiput with osteo of occipital bone.	Sequestrectomy and drainage abscess of dura. Tantalum cranioplasty for 3 weeks, then graft over granulating dura.	Excellent coverage. Still requires protection.
1954	R. W.	Periosteal burn left frontal bone.	Removal outer table and graft coverage in 14 days.	Very good.
	C. K.	Periosteal burn left parietal.	Removal outer table and graft coverage by 46 days postburn.	Very good.
1955	G. A. L.	Periosteal burn with exposure frontal bone.	Calvarium drilled and granulations grafted.	Good, Rapid coverage.
1956	G. S.	Deep burn involving periosteum of entire left side of skull and and frontal sinuses.	Debridement and outer table drilled. Outer table removed and allowed to granulate. Frontal sinuses unroofed.	Osteo of outer table. Grafted over granulations took well.

ance of granulation tissue in an area of deep periosteal burn of the skull. The simplest and least traumatic procedure, although frequently least effective, is to drill the outer table of the skull with a large dental drill to permit the granulating buds to grow out through the holes (Fig. 11). However, this procedure is frequently ineffective because the outer table degenerates and has to be removed by a chisel or osteotome. Grafting can then be carried out on a good granulating base. An alternative procedure is to remove the outer table as soon as it is felt that the periosteum is nonviable to enable granulations to be produced from the marrow. If the outer table is removed, it requires considerable care in order not to penetrate the inner table. Upon removal of the outer table, the entire area is dressed and granulations are allowed to form. As soon as granulations are present, grafting can be carried out without difficulty, and an excellent take usually results.

Deep burns involving the superficial portion of the long bones, joints, and the bones of the hands and digits are not uncommon, particularly among patients sustaining burns as a result of accidents involving jet aircraft.

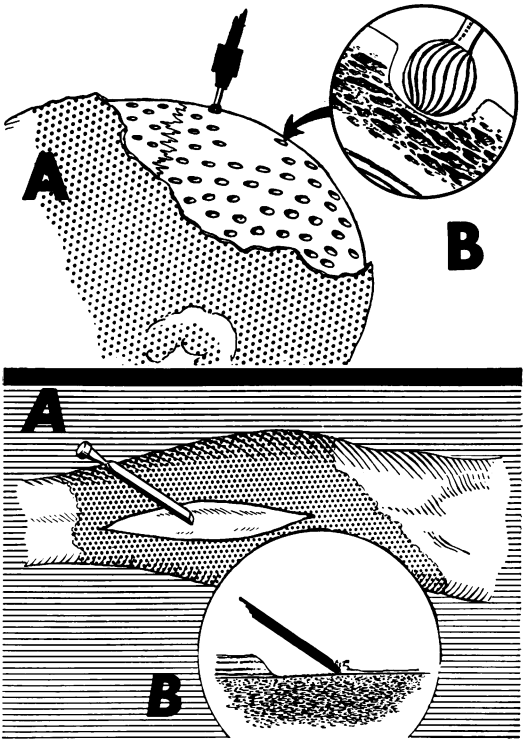


FIG. 11. (*Upper*) Drilling the outer table. Care must be taken not to penetrate the inner table. If the outer table degenerates, it is removed.

FIG. 12. (*Lower*) Cortical bone removed by a chisel. Drilling may also be used but is more difficult.

TABLE 5. *Deep Burns Involving Peripheral Bones*

Joint involvement often occurs with it. Coverage by skin requires granulations but once covered results are good.

Year	Patient	Complication	Treatment	Result
1950	M. L. C.	Septic knee joint and osteo of femur due to deep joint burn.	Immobilization by cast, aspiration of joint, antibiotics.	No shortening. Stiff joint.
	T. M.	Osteo of ankle due to deep burn. Infection penetrating joint when immobilized for sprain at time of burn.	Removal tibio-astragular cartilage for fusion. Amputation at middle 1/3.	Fusion failed. Stump closed secondarily.
1952	R. A. M.	Osteo medial malleolus.	Sequestrectomy.	Excellent. No limitation.
	J. P.	Osteo phalanges index, middle and ring fingers.	Amputation and graft.	Good.
1953	H. D. R.	Osteo of fibula due to deep burn.	Curettement and closure by granulation.	Excellent. No symptoms.
	M. B.	Periosteal burn, 8" left tibia.	Drilled, granulation grafted.	Excellent, rapid coverage.
	R. E.	Periosteal burn left tibia.	Drilled, allowed to granulate, grafted.	Very good.
1954	S. D. O.	Periosteal burn of tibia and both malleoli.	Removal cortical bone, granulations formed and grafted.	Very good.
1955	G. F. B.	Osteo 1st metacarpal due to deep electrical burn.	Four attempts at grafting over granulations.	All grafts failed. Filled in from margins. Stiff joint.
1956	R. Y.	Osteo radial-ulnar joint secondary to deep burn and fracture ulnar styloid.	Curettement, enzymes, antibiotics and immobilization.	Stiff wrist.

In the treatment of deep periosteal burns, it must again be emphasized that the cortical bone should not be removed in the early postburn period. A surgeon should wait until it is quite evident that no granulations will form from the involved periosteum and that the only source possible is the marrow cavity of the involved bone. As soon as this is evident, the surgeon may gain access to the marrow cavity by the same method used for producing the granulations in cases of periosteal burns of the skull.

In areas where thick cortical bone is present, such as the tibia, it is much easier to chisel away the cortical bone and expose the marrow cavity (Fig. 12), thus allowing granulations to form. Usually granulations form in a very short time and coverage can be attained with little delay. In the smaller bones, such as the metacarpals and the phalanges, it is more feasible to use a dental drill to penetrate the cortex and gain access to the marrow cavity. Table 5 tabulates some representative cases of deep periosteal burn and the methods of treatment used. Figures 13 and 14 show actual cases of deep periosteal burns of the tibia and skull that were treated by the methods outlined.

Neurologic Complications

The neurologic complications attendant upon thermal injury are generally due to localized areas of deep third-degree burn; often, however, they are dependent upon the improper use of therapeutic measures designed for treatment of the burn wound. Table 6 shows the neurologic complications that occurred from 1950 through 1956, the methods of treatment utilized, and the re-

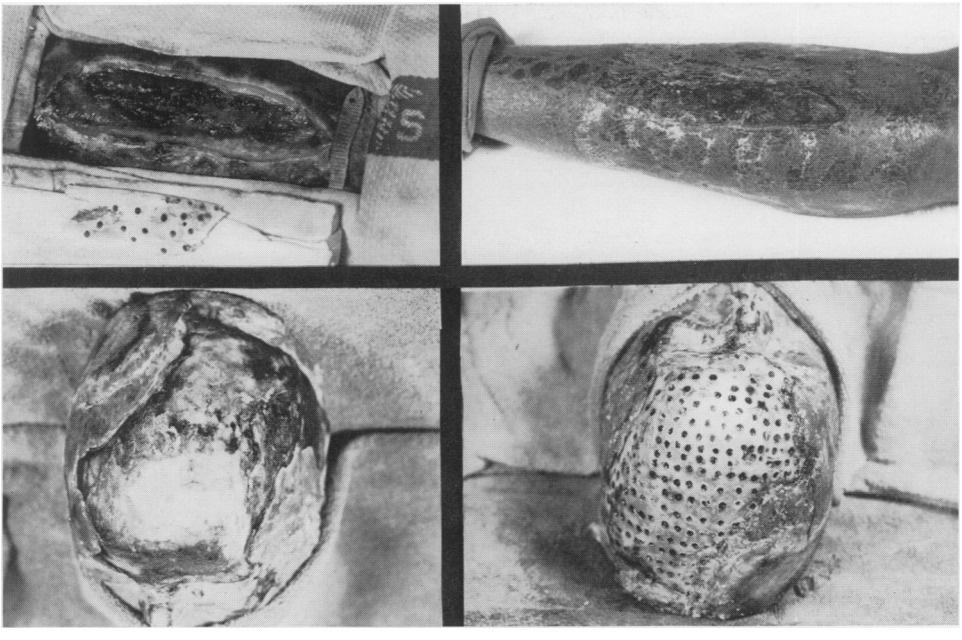


FIG. 13a. (*Upper left*) Deep burn involving tibia. After drilling some granulations appeared but cortical bone became necrotic and was removed.

FIG. 13b. (*Upper right*) Result following grafting.

FIG. 14a. (*Lower left*) Deep burn involving skull. Granulations have formed from the viable periosteum but the central area is dead.

FIG. 14b. (*Lower right*) Following drilling, granulations formed but outer table became necrotic and was removed. Grafts took very well over granulations of exposed marrow.

sults obtained. It will be noted that only one central neurologic complication occurred; this was secondary to a dural abscess resulting from an osteomyelitis of the occipital bone following a deep electrical burn in the area. Other complications involved peripheral nerves. The neurologic deficit was due either to a localized deep third-degree burn or to incorrect application of therapeutic measures, the most common being the application and prolonged use of tight compressive dressings over areas where the peripheral nerves were quite superficial.

Initially, it is of little therapeutic importance whether or not peripheral nerves are involved as an immediate result of thermal injury because widespread infection invariably accompanies all thermal burns. Therefore, a surgeon should not attempt any primary repair of the nerve tissues in a burned area. Early in the postburn period, it might be of prognostic value to determine whether or not a neurologic deficit exists, whether it is progressive, and the extent of involvement. The primary object in the treatment of these neurologic complications is skin coverage of the involved area; only under these circumstances will the infection in the area resolve. Unless infection is controlled, there is no opportunity for regeneration of the nerve; hence no therapeutic surgical procedures should be carried out.

During the period in which the burned area is being prepared for grafting, during the actual grafting procedure, and after any peripheral nerve surgery the most important adjunctive treatment is active physiotherapy and the functional support of the

TABLE 6. *Neurologic Complications, 1950–1956*

Year	Patient	Burn Agent	Complication	Treatment	Result
1950	H. B.	Electrical	Meningitis. Secondary to deep burn of occipital bone and abscess of dura.	Sequestrectomy, tantalum cranioplasty and graft. Drainage of dural abscess.	Cleared rapidly.
	Y	Gasoline	Radial palsy and wrist drop due to tight dressings.	Support and P. T. and skin coverage.	Slow, incomplete return of function.
1951	C. P.	Gasoline	Peroneal palsy due to compressive dressing.	Removal dressing. P. T. and support.	Complete recovery.
1952	C. E. L.	Jet fuel	Peroneal palsy due to deep burn about knee.	Support, P. T., and Graft coverage.	Marked weakness.
	T. L. C.	Gasoline	Peroneal palsy due to deep burn.	Support, P. T. and graft coverage.	Permanent weakness.
	P. B. E.	Jet fuel	Ulnar paralysis due to deep burn about elbow.	Graft coverage. Support to hand and digits.	Complete loss ulnar function.
	B. L. F.	Gasoline	Peroneal palsy due to dressing.	Removal dressing, P. T. and support.	Permanent weakness.
	W. J.	Electrical	Ulnar hypesthesia without motor loss due to deep burn of elbow.	Graft coverage, P. T. and support.	Partial incomplete return. Pain on use of hand.
1953	J. R.	Jet fuel	Median, ulnar and radial palsy below shoulder due to cast for immobilization after graft.	Support and P. T.	Marked weakness below elbow with median sensory loss.
	A. B.	Gasoline	Median and ulnar paralysis due to deep burn of wrist.	Graft coverage and support.	Claw hand with loss of all intrinsic hand muscles.
	J. A. B.	Gasoline	Ulnar palsy due to dressing.	Removal dressing, P. Y. and support.	Complete recovery.
1954	G. K.	Jet fuel	Bilateral peroneal paralysis due to dressings.	P. T., support, graft coverage.	Only toe extensors returned. Requires braces.
	F. G. M.	Gasoline	Peroneal palsy due to deep burn.	Support, P. T. and graft coverage.	No recovery. Requires brances.
	M. P. S.	Unknown	Deep burn severing ulnar nerve at elbow and great tissue loss.	Graft coverage and support.	Complete motor and sensory ulnar loss.
	R. S.	Gasoline	Peroneal palsy due to dressing.	Graft coverage, P. T. and support.	Slow return of full function.
1955	R. R. W.	Electrical	Severed nerves (and vessels) or arm.	Amputation.	Prosthesis.
1956	V. F.	Clothing	Bilateral peroneal palsy due to cast for immobilization after perineal grafting.	P. T. and support.	Rapid complete return of Function.
	R. H.	Gasoline	Radial palsy due to fracture of humerus and poor immobilization due to burn.	Skeletal traction, graft coverage and P. T.	Slow union of transverse fracture. Slow return of radial function.
	G. S.	Jet fuel	Brachial palsy, left complete due to stretch injury at time of burn.	Support and P. T.	Rapid complete recovery.

involved extremities to prevent contractures and atrophy from disuse. The neurologic deficit and functional deformity is often considerable even under optimum circumstances.

In the treatment of burns of the extremities, considerable care must be taken to assure protection of the peripheral nerves. Particular caution should be taken of the peroneal nerve as it crosses the fibular head, the ulnar nerve in the groove of the elbow, and the median, radial and ulnar nerves in

TABLE 7. *Eye Complications*

Local care consists of tarsorrhaphy and ectropion correction as indicated, frequent irrigations and antibiotic ophthalmic ointment.

Year	Patient	Burn Agent	Complication	Treatment	Final Result
1950	R. E. B.	Jet fuel	Severe bilateral keratitis due to infection secondary to severe ectropion, bil. on arrival 40 P. B.	Local. Perforation of anterior chamber and blindness. Enucleation left eye with conjunctival flap. Right blind.	Progressive infection with *Proteus* and *Pseudomonas* finally resistant to everything
	O. C. H.	Wh. phos.	Bil. corneal burn. Cataract O. D. due to rupture of lens.	Local.	"Light perception only."
1952	R. W. D.	Gasoline	Corneal burn and cataract. O. S.	Local.	Blindness O. S.
	R. J.	Wh. phos.	O. S.—corneal burn—leukoma.	Local.	"Vision markedly reduced." O. S.
	G. A. K.	Gasoline explosion	O. D.—Commotio retinae with cystic degeneration macula.	Local and rest.	O. D. 5/400. O. S. 20/20.
	M. F. K.	Gasoline	O. D. corneal erosion due to ectropion.	Ectropion corrected.	Healed rapidly 20/20.
	R. C. L.	Gasoline	O. S. corneal ulcer due to infection with pus in anterior chamber.	Tarsorrhaphy and local.	Cleared rapidly 20/70.
	E. D. N.	Gasoline	Bil. corneal ulcer due to infection	Local.	Healed 10 days 20/20.
	C. C. S.	Gasoline	Bil. corneal ulcer due to *Proteus*.	Local.	Healed 7 days 20/20.
	A. B.	Gasoline	Bil. corneal ulcer due to infection.	Local.	Clearing well at death 6 days later.
1953	A. J. B.	Jet fuel	Bilateral deep corneal burn.	Tarsorrhaphy and local.	O. S. "Light perception only." Cataract, anterior synechiae, and pupil obliterated O. D. No vision. Corneal transplant not possible.
	H. D.	Gasoline	Bil. corneal erosion due to ectropion.	Ectropion corrected.	Healed rapidly 20/20.
	M. F.	Gasoline	Bil. corneal ulcers due to *Pseudomonas*.	Local and conjunctival flap.	Improved.
1954	P. A. Y.	Wh. phos.	Bil. corneal burn.	Local.	"Slight blurring of vision."
1955	G. A. C.	Gasoline	Bil. corneal burn.	Tarsorrhaphy and local.	Healed rapidly 20/20.
	E. C.	Gasoline	Bil. corneal burn.	Local.	Healing rapidly at death 7 days later.
1956	G. S.	Jet fuel	Thermal destruction left eye. Thermal destruction cornea, O. D. with loss Descemet's membrane and prolapse of iris.	Conjunctival flap. Enucleation.	Blind, O. S. Light perception only. O. D. No graft possible

their location adjacent to the humerus in the midarm. A prolonged application of compressive dressings or plaster cases for immobilization in these areas, even if adequate padding has been provided, may lead to a peripheral nerve palsy. If damage is severe, the loss of function may be permanent (Fig. 5). Dressings and immobilizing casts should be changed frequently, and the patient is observed for signs of any neurologic defect. It is obvious that a motor defect could not be detected easily

when the extremities are completely immobilized by a plaster cast or by a large compressive dressing. It is of little benefit to a patient to have a burned extremity completely covered with skin and, at the same time, be functionally useless because of peripheral nerve damage.

Complications in Burns of the Eye

The eye and visual complications are seldom observed because very few occur as a direct result of thermal injury. In more than 1,000 burned patients treated, approximately 500 sustained burns involving the face and the regions of the eyes. In only a few instances did thermal injury occur to the structures of the globe or to the globe itself, and most of these were localized third-degree burns caused by phosphorus or severe burns caused by accidents involving jet aircraft. Table 7 shows the eye and visual complications encountered from 1950 through 1956, the treatment utilized, and the results obtained. In the years before prophylactic procedures were well established, the complication of infection by resistant microorganisms secondary to the periorbital burns and ectropions was equally important as those due to direct thermal injury.

Thermal destruction of ocular tissue cannot be prevented by the physician who does not see the patient until after it has occurred. However, the progression of this injury by prolonged contact with a burning agent, such as phosphorus, and the added destructive effect of infection and drying can be completely prevented if adequate prophylactic measures are carried out. Irrigation of the eyes with copper sulfate solution and the meticulous removal of the remaining fragments of phosphorus is mandatory in the treatment of phosphorus burns. This therapy must be carried out immediately after exposure to the burning agent. Delay in therapy for any significant period of time will allow the destruction of tissue and will lessen the chances of saving a patient's vision. Even if prophylactic measures are carried out quickly, there may be a marked diminution in visual acuity. If the burn is of such a penetrating nature that the lens or anterior chamber is involved, vision may be completely destroyed.

The prevention of corneal erosion secondary to drying of the eye or a purulent conjunctivitis spreading from the surrounding burned tissue is the responsibility of the attending surgeon. Surgical intervention is required in many cases. In the discussion of ectropion, a description was presented of burns of the lids that cause a drying of the ocular and palpebral conjunctiva due to inability of the lids to close and the progressive eversion of lid margins. If this drying is of such a degree that corneal erosion is impending or present or if a chronic conjunctivitis develops, it is necessary either to do an ectropion correction or a lid tarsorrhaphy (Fig. 4). If long-term apposition is required, simply suturing the lid margins together will not suffice because the sutures invariably pull through the margins in three to four days. A firm union can be established only by suturing the tarsal plates together. A central aperture is left for vision and the medial and lateral canthus is left open to facilitate irrigation and frequent applications of local antibiotic ointments.

Whenever a burn of the lids or the face adjacent to the lids occurs, particularly if any conjunctivitis or corneal erosion develops, it is mandatory that eye care be carried out. Therapy consists of frequent daily irrigations of the eyes with a saline solution and repeated application of ophthalmic antibiotic ointments after each irrigation. Sulfacetamide is the ointment of choice because it possesses wide-spectrum activity. However, if some specific microorganism is isolated having a known susceptibility to a single antibiotic ointment, this particular ointment should be used. It cannot

TABLE 8. *Genito-urinary Complications*

Urethral and bladder infection results from prolonged catheter drainage.

Patient	Complication	Remarks	Result
C. B.	Periurethral abscess.	Catheter in bladder 37 days.	I & D—continued catheter drainage—still present at death 23 days later.
J. S.	Periurethral abscess.	Catheter in bladder 30 days.	I & D and bladder drainage—cleared in 10 days.
F. S.	Periurethral abscess.	Catheter in bladder 36 days.	I & D and continued drainage, cleared slowly.
B. V.	Periurethral abscess.	Catheter in since 8 days postburn. Adm. 21 postburn and abscess found 25 PB. Treated by drainage and suprapublic cystotomy but drainage poor so perineal urethrotomy done.	Fair after dilatations but still leaks some from posterior urethra.
K. M. B.	Cystitis, severe and urethral stricture.	Deep charred burn of genitalia.	Cleared with removal of catheter—dilated to 16 F.
M. J. K.	Purulent cystitis.	Not noted clinically. Found at autopsy.	Died of septicemia.

be emphasized too strongly that great care must be taken in the prophylactic treatment of the eyes when the periorbital structures are involved, since secondary complications will invariably develop if this is not done and they will cause a disability far out of proportion to the magnitude of the burn. This is tragically demonstrated in Patient R. E. B. (Table 7).

Genito-urinary Complications

The complications involving the genito-urinary system, in most instances, are those connected with the therapy instituted rather than those resulting from the thermal injury itself. Although severe burns to the external genitalia may result in complications attributable to direct thermal injury (Table 8), the majority of these complications are caused by indwelling urethral catheters or prolonged immobilization, or a combination of the two. Table 8 shows that one of the most common complications of indwelling urethral catheters is a periurethral abscess that develops at varying postburn periods, although it usually follows prolonged use of catheter drainage with or without bladder irrigation. The abscess usually appears at the base of the penis near the junction with the scrotum. The only satisfactory treatment is incision and drainage, continued catheter drainage in an attempt to prevent a urethral fistula, and closure of the wound by secondary intention. It is obvious that the best way to avoid this complication is to remove the urethral catheter as soon as possible. This procedure is usually feasible from the seventh to the tenth postburn day, unless there is some strong contraindication. Even if a periurethral abscess does not develop, it is quite probable that the patient will develop a purulent cystitis in addition to catheter cystitis accompanying bladder drainage if the catheter is in use for a long term.

With prolonged catheter drainage and the almost absolute immobilization accompanying an extensive third-degree burn, a surgeon must always bear in mind the possibility of the development of a renal or bladder calculus (Table 9). Although the cause of stone formation is not definitely established, it is quite likely that important contributory factors are protracted catheter drainage, attendant infection, and the mo-

TABLE 9. *Genito-urinary Complications*

Renal and bladder calculi are not uncommon with prolonged immobilization.

Patient	Complications	Remarks	Result
V. R.	Bilateral staghorn calculi. Bilateral hydronephrosis due to above. Right hydroureter due to ureteral stone. Periurethral abscess.	Increasing in size. CVA pain, fever, rising NEN.	Left nephrectomy. Calculus removed from right pelvis and ureter. I & D and continued catheter drainage—cleared slowly.
J. H. W.	Bladder calculus. Urethral stricture.	7 months postburn.	Removed per urethra—small fragments remain. Dilated.
P. B.	Bladder calculus. Urethral stricture.	Found 105 days postburn. Old G. C. stricture. Required a filiform for initial postburn catheterization. Catheter left in 83 days after perineal urethrostomy done.	Still requires intermittent dilatation.
L. F. S.	Bladder calculus.	Catheter in bladder 74 days.	Stones removed per urethra—Cleared.

bilization of large quantities of calcium salts from the osteoporotic bones of the immobilized patients. In order to minimize the incidence of calculus formation, the duration of bladder drainage should be held to an absolute minimum; the urinary tract infections should be treated without delay, and the patient should be mobilized as rapidly as possible. Once the calculus has formed in a burned patient, its treatment is no different than in an unburned patient.

Homologous Serum Jaundice

One of the most serious complications of therapy in thermal injury is that of homologous serum jaundice resulting from the use of plasma and/or blood during the resuscitative period or during his recovery period for mantenance of the patient. Table 10 shows the total number of cases of homologous serum jaundice encountered in this Unit from 1950 through 1956. This cannot be taken as any indication of the incidence of homologous serum jaundice occurring in the treatment of thermal injury, since the vast majority of the patients in 1950 received their initial resuscitative therapy elsewhere and this was also true of patients in 1952. Blood and plasma are implicated as the etiologic carrier in approximately an equal number of cases (Table 10).

In this series only three patients died (B. R., P. H., J. S.), but death occurred soon after the onset of jaundice. The duration of life after onset of jaundice was two days in two patients and five days in the third. The treatment of the homologous serum jaundice in the burned patient is essentially the same as in the unburned patient. To subject a patient to any type of general anesthesia or any major surgical procedures during the period of this hepatic disease and its immediate recovery period is contraindicated. It is fortunate that the vast majority of the patients will probably have complete skin coverage at the time of onset of hepatic disease. However, the three patients in this group who died did not have complete skin coverage, and surgical procedures were carried out under general anesthesia in the period immediately preceding clinical detection of jaundice. It is quite possible that surgical intervention contributed significantly to the rapid death of these patients.

Gastro-intestinal Complications

If the complications of homologous serum jaundice, Curling's ulcer, and the para-

TABLE 10 *Homologous Serum Jaundice, 1950–1956*

One additional case who received only blood has been seen. Recovery was uneventful.

Year	Patient	Age	Initial Treatment	Onset	Result
1950	C. P.	23	Plasma	60	Cleared 4 weeks.
	O. B.	30	Blood and plasma	65	Cleared 4 weeks.
	R. W.	20	Blood	108	Cleared 6 weeks.
	A. R.	20	Plasma	90	Cleared rapidly.
	R. M.	24		85	Cleared rapidly.
	J. S.*	18	Blood and Plasma	86	Expired 88 P. B.
1952	B. B.	44	Blood and plasma	70	Cleared 3 weeks.
	T. C.	20		90	Cleared 4 weeks.
	G. C.	22	Plasma	90	Cleared 6 weeks.
	C. D.	22	Blood and plasma	98	Cleared rapidly.
	P. H.*	26	Blood and plasma	94	Died 76 P. B.
	G. H.	60	Blood	60	Cleared slowly.
	G. H.	27	Blood and plasma	71	Cleared 6 weeks.
	R. L.	24	Blood	100	Cleared 3 weeks.
	R. M.	21	Plasma	65	Cleared 3 weeks.
	E. N.	31	Blood and plasma	90	Cleared 4 weeks.
	C. R.	27	Plasma	120	Cleared 4 weeks.
	C. S.	36	Blood and plasma	111	Cleared 4 weeks.
	R. S.	25	Plasma	67	Cleared 4 weeks.
	J. W.	25	Plasma	65	Cleared 4 weeks.
1953	E. A.	21	Blood	89	Cleared rapidly.
	A. J. B.	28	Blood and plasma	86	Cleared in 3 weeks.
	M. B.	25	Blood	120	Cleared rapidly.
	E. B.	24	Blood	90	Cleared rapidly.
	W. A. E.	36	Blood	120	Cleared rapidly.
	H. H.	22	Blood and plasma	95	Cleared in 2 weeks.
	J. J.	22	Blood	90	Cleared rapidly.
	B. R.*	22	Blood and plasma	72	Expired 77 P. B.
	V. R.	21	Blood and plasma	77	Cleared in 3 weeks.
	R. S.	26	Blood	60	Cleared in 10 weeks.
	B. V.	21	Plasma	130	Cleared 5 weeks.
	J. H.	24	?	80	Cleared rapidly.
1954	J. M.	23	Blood	100	Cleared in 5 weeks.
1955	W. D. B.	23	Blood	63	Cleared in 4 weeks.
1956	J. M.	32	Blood and plasma	111	Cleared in 4 weeks.

* Death

lytic ileus associated with septicemia are excluded, the other gastro-intestinal complications associated with burns are relatively uncommon. Table 2 shows the complications encountered from 1950 through 1956, the type of treatment utilized and the results obtained. The most dramatic complication is acute gastric dilatation. This complication demands immediate therapy and fortunately responds to therapy quite rapidly and successfully. If not recognized, this condition may be the direct cause of death. Even if recognized and treated, the attending physician must be aware of the possibility of a recurrence at some future date. It is probable that acute gastric dilatation is more likely to occur in the burned patient than in the average hospital patient, since the gastro-intestinal tract shows other evidences of dysfunction during both the immediate postburn period and early convalescence.

In burns about the face, particularly where the mobility of the mouth is impaired, the nose is plugged so that mouth breathing is mandatory. As the patient is unable to take oral fluids properly, it is imperative that good oral hygiene be maintained. This can be carried out only by adequate and painstaking nursing care. If this care is not given, the tongue becomes dry and fissured, the lips crack and bleed easily, the gums become soft and hypertrophic, and a significant parotitis may develop. If the latter condition occurs, it is unlikely to resolve without the instituting of proper oral hygiene. Other treatment of inflammation of the parotid gland depends upon the attending physician. Lugol's solution, antibiotics, x-ray therapy, and other

forms of treatment have been used with varying degrees of success. The remaining cases tabulated in Table 6 are self-explanatory. Patient H. V. vividly demonstrates the potential danger of prolonged esophageal intubation no matter what the cause. The case of Patient R. R. illustrates the long-debated subject of the relationship between acute stress and Curling's ulcer. The problem of Curling's ulcer is not included in this presentation as it has been presented adequately in a publication from this Unit by Dr. Hummel.

Cardiac Arrest

Cardiac arrest occurs in the burned patient as well as in the unburned patient. It is possible that the general physiologic derangements occurring in the burned patient may predispose to its occurrence more readily. These derangements include the toxicity resulting from widespread infection, the marked malnutrition, and general body exhaustion accompanying a severe burn. Table 7 shows the episodes of cardiac arrest as they have occurred in this Unit from 1950 through 1956. In all but one of the five instances, cardiac arrest occurred during induction of anesthesia (H. F.). This patient was a 5-year-old youngster who sustained a very extensive burn and was in a severe stage of malnutrition. Inadvertently he was given one-eighth grain of morphine intravenously for sedation. It would appear that all of the circumstances under which these episodes of cardiac arrest occurred are obvious and preventable ones, and one should be alert to the possibility under these circumstances. When cardiac arrest occurs, prompt resuscitation must be instituted. If resuscitation is instituted immediately, the chances of recovery are quite good as demonstrated in the cases of M. L. and R. J.

Renal Insufficiency

The question of significant renal insufficiency occurring in the immediate postburn period is not clear. Obvious derangements in renal function occur as evidenced by changes in urinary output, albuminuria, and varying quantities of cellular elements appearing in the urinary sediment. It is not known whether these conditions are a result of the direct effect of thermal injury or of the extensive physiologic changes in a human body sustaining a burn of great severity, but they are more likely to be a combination of all these factors. It is unlikely, however, that the deaths that occur are secondary to the typical "tubular necrosis" associated with the usual concept of "acute renal insufficiency." The vast majority of severely burned patients recover rapidly from any evidence of renal impairment. This is not true, however, in the case of tubular necrosis.

The laboratory tests used routinely to determine renal function are of little value in the immediate and early postburn period. Microscopic examination of the urine may reveal the abnormalities present, but it does not give any indication as to their etiology. The tremendous changes in blood volume occurring as a result of the shifts of extracellular and intracellular fluid volume can cause significant and rapid changes in glomerular filtration rate disassociated with any true structural changes in the kidney. The volume of urinary output cannot be considered as an accurate guide since following oliguria there may be an increase in the volume of urine while the glomerular filtration rate remains below or does not exceed "normal limits." Unless determined with extreme frequency, insulin and PAH clearances are of doubtful accuracy since the factors upon which they depend are changing rapidly during the early postburn period.

Which patients then might be considered as having developed a renal failure on the basis of tubular necrosis? Certainly those patients expiring within the period of three to 14 days after thermal injury and having

a clinical picture of septicemia and a positive blood culture could not be considered as having died of renal failure. Death undoubtedly was due to septicemia. At the time of death, such patients invariably had a progressive oliguria and refractory hypotension, but the decrease in urinary output was only one factor in the over-all picture. Some patients, particularly those sustaining extensive burns of full thickness, do not excrete what would be considered adequate quantities of urine during their entire postburn period. Are such patients suffering from renal failure on the basis of tubular necrosis? They do not present the usual clinical picture seen in this syndrome. Except for the initial rise during the first 12 to 24 hours, a rapid rise in NPN is seldom observed and the potassium reaches dangerous levels only in rare instances. These same changes in NPN and potassium occur frequently in patients who have normal urinary output but burns of rather large extent. The pattern typical of urinary output in acute renal insufficiency is not seen; but an immediate oliguria persists instead of a rapidly progressive oliguria. The patients who have this persistent oliguria and succumb usually die within the first to the fourth day. Thus death occurs much more rapidly than in the case of a patient having tubular necrosis.

During this period a generalized depression of body function occurs as evidenced by a low white blood count, a minimal elevation of general body temperature in spite of the obvious presence of massive infection, a relative tachycardia, and a very poor response to stimuli or questioning. Blood chemistries during the period just prior to death reveal levels of NPN, potassium, and other blood substances that are quite compatible with life. The deaths of these individuals could not be attributed to renal insufficiency in the usual meaning of that term.

At postmortem examination, the kidneys of patients dying of thermal injury do not show any changes that are consistently associated with any particular course. An interstitial and/or focal nephritis with or without focal thrombi may be present, or there may be a generalized pyelonephritis. Cloudy swelling and hydropic degeneration of the tubular epithelium may occur in varying degrees. In those patients dying with protracted periods of hypotension, hydropic degeneration is often present, although not always, and frequently it is disassociated with any particular changes in blood pressure. The individuals dying of septicemia may show evidence of focal abscesses in the kidneys. Other than the changes reflecting the presence of widespread bacterial invasion, however, there are no morphologic features characteristic of this group of patients.

In the clinical experience of this Unit, patients oliguric in their immediate postburn period become so usually as a result of inadequate replacement fluid therapy in the resuscitative period immediately following the burn. Oliguria was observed in the immediate postburn period among many patients admitted from other installations but upon the institution of adequate resuscitative therapy (frequently requiring volumes far in excess of that dictated by any so-called "burn formula") their response was rapid and their volume of urinary output was adequate. Noteworthy is the frequency of this pattern of an initial oliguria due to inadequate resuscitative therapy and its rapid response to fluid replacement.

Other patients have been encountered who failed to respond to fluid therapy. Oliguria persisted. It is probable that this is a circulatory renal insufficiency secondary to renal ischemia. Recovery should occur in two to four days. How to differentiate these conditions is difficult. A good water load test is the best means.

TABLE 11. *Gastro-intestinal Complications (Other than Curling's Ulcer), 1950–1956*

In addition to those listed there have been 23 proven cases of Curling's ulcer, four of whom died of hemorrhage or peritonitis. The only two resected showed no healing of the gastro-intestinal anastomosis at autopsy.

Year	Patient	Complication	Treatment	Result
1950	R. R.	Recurrence of hematemesis and melena from old ulcer existing prior to burn.	4,000 cc. blood. Ulcer diet since admission.	Expired 10 days postburn of hemorrhage.
1952	C. W.	Acute gastric dilatation on four occasions.	Suction with temporary relief.	Died 4 days postburn of septicemia.
	M. G.	Acute gastric dilatation.	Suction with removal 1,000 cc. gastric contents.	Immediate relief of cyanosis and respiratory difficulty.
1953	M. F.	Acute gastric dilatation on on two occasions.	Suction with relief.	Died of cardiac arrest during anesthesia.
	T. H.	Acute liver atrophy.	Nonspecific.	Expired 11 days postburn of septicemia.
	V. R.	Bilateral parotitis.	Oral hygiene.	Cleared slowly.
1954	L. S.	Acute gastric dilatation with retention of tube feeding.	None found at autopsy.	Died—cause of death unknown.
1955	R. D.	Parotitis, left.	Lugol's, penicillin and x-ray (70R X3).	Cleared without sequelae in 4 days.
	H. V.	Esophageal stricture secondary to feeding tube in place 4 months. Symptoms at 5 months. X-ray evidence at 9 months.	Dilatations.	Eats all but hard foods and large bolus.
1956	F. H.	Hepatic parenchymal (central) degeneration. Probably due to hypoxia secondary to emphysema and general anesthesia.	None found at autopsy.	Died 8 days postburn.

Table 13 shows three types of renal insufficiency observed at the U. S. Army Surgical Research Unit from 1950 through 1956. Patient M. J. illustrates a combination of inadequate resuscitative therapy and improper fluid replacement therapy as evidenced by intravenous administration of distilled water. This patient's burn was not of such an extent that the burn would cause any great difficulty if treated adequately. Therefore, it is probable that the renal insufficiency was due to a combination of

TABLE 12. *Cardiac Arrest, 1950–1956*

The period of induction of anesthesia is a dangerous one.

Year	Patient	Remarks	Treatment	Result
1953	M. L.	First episode 29 days postburn during intubation for anesthesia. Second episode 36 days postburn under same circumstances. Died 2 months postburn of septicemia.	Thoracotomy, massage, I. V. calcium chloride. Same treatment.	Recovered with no evidence of neurologic damage. Recovered after 30 min. of fibrillation and massage. No residual.
	R. J.	Occurred during induction of anesthesia.	Thoracotomy and massage.	Recovered with no evidence of neurologic damage.
	M. F.	Occurred during induction of anesthesia.	Thoracotomy after 3–5 min. delay.	Recovery of beat but died quietly 8 hours later.
1956	H. F.	Secondary to respiratory arrest from morphine intoxication.	Thoracotomy and massage with no delay.	Recovery of heart beat but died 8 hours later.

TABLE 13. *Renal Insufficiency, 1950–1956*

This demonstrates three etiologic factors.

Patient	Onset	Cause	Remarks	Treatment	Result
M. J.	Day of burn.	Inadequate resuscitation and improper fluid.	Inadvertently received distilled water I. V. as portion of resuscitative therapy.	5% G/W, sodium lactate, insulin.	Expired 10 days postburn still oliguric.
J. R.	8 Days postburn.	Repeated episodes shock.	Extremely labile blood pressure with 3 distinct episodes of shock during surgery progressing rapidly to pulmonary edema with resuscitative therapy. Deep muscle burns of legs.	5% G/W, insulin, sodium lactate, resins.	Potassium level controlled easily. Diuresis began on 13 postburn day but expired 16th day due to uremic hemorrhage.
A. J. M.	Day of burn.	Inadequate resuscitation in first 12 hrs., hypotensive for 12 hrs.	Oliguric for 2 days postburn, then anuric.	Fluid resuscitation after 12 hrs. Quite vigorous, but no urine response.	Died 7 days postburn.

inadequate resuscitative therapy in the immediate postburn period and the hemolysis of red blood cells associated with the administration of distilled water. The clinical course of this patient was typical of renal failure in that the NPN and potassium rose progressively and she became somnolent and showed clinical signs of uremic bleeding. This patient was still oliguric on the tenth postburn day when she died.

The second patient (J. R.) had adequate urinary output immediately following thermal injury. Because of an extremely labile blood pressure that could not be combatted adequately, he suffered three distinct episodes of shock during surgery. His condition was complicated by the fact that resuscitative therapy, whether given rapidly or slowly, almost invariably resulted in pulmonary edema requiring phlebotomy. As a result, the patient developed clinical evidence of renal insufficiency eight days after injury with a typical progressive oliguria and increasing elevation of NPN and potassium. The potassium was easily controlled with intravenous glucose, and insulin and resins. The NPN continued to rise rather rapidly until diuresis was initiated on the 13th postburn day, and then it ceased to rise. However, the patient died on the 16th day from generalized uremic hemorrhage that could not be controlled. The renal insufficiency in this case was not considered the direct result of the thermal injury but the result of repeated episodes of hypotension.

The third patient (A. J. M.) is representative of one of several patients observed whose clinical course followed a rather typical pattern. These patients received inadequate resuscitative therapy immediately after their injury and were subjected to rather prolonged periods of hypotension extending in some instances from 12 to 24 hours. Because of hypotension rather than thermal injury, they were oliguric from the onset. These patients died at various postburn periods, usually from the fourth to the seventh day, and during this interval their output of urine remained inadequate. They may or may not have died showing clinical and bacteriologic evidence of septicemia.

The U. S. Army Surgical Research Unit has an artificial kidney with its ancillary personnel and laboratory equipment. It was originally attached to the Burn Ward on the basis of reports in the literature of renal insufficiency occurring after thermal injury;

TABLE 14. *Periarticular and Fascial Calcification, 1950–1956*

Maintaining 15–20 degrees flexion of the elbow during immobilization with dressings has diminished the limitation of motion but the calcification is still occurring.

Year	Patient	Joint Area Involved	Treatment	Result
1953	L. S. K.	Extensive soft tissue calcification along fascial planes inferiorly from lesser trochanter. Same as seen in paraplegics.	Physio-therapy. Ambulation after 75 days bed rest.	Walks with cane. Has pain in left hip on motion.
	B. V.	Extensive soft tissue calcification about both hips along fascial planes.	Physio-therapy. Ambulation after 210 days bed rest.	Pain on initial walking but rapidly cleared.
1954	L. W.	Fusion left hip secondary to extra-articular strut from neck of femur to upper lip of acetabulum. Not present 6 months postburn, but solid at 24 months postburn.	Excised surgically.	Good range of motion.
1956	C. C.	Periarticular calcification right elbow.	Physio-therapy. Excision tip of olecranon.	Limited to 30° range of motion.
	J. F. Q.	Periarticular calcification left elbow.	Physio-therapy.	Only 60° flexion.
	W. McL.	Periarticular calcification left elbow.	Physio-therapy.	Full range of motion.
	L. R.	Periarticular calcification left elbow.	Physio-therapy.	Limited to 30° flexion.
	R. P.	Periarticular calcification left elbow.	Physio-therapy.	Limited to 20° flexion.
	T. H.	Periarticular calcification right elbow.	Physio-therapy.	Limited to 60° flexion.

but on only one occasion has the artificial kidney been used in the treatment of a burned patient. This patient was not dialyzed because of his burns, however, but because of an acute glomerular nephritis associated with the thermal injury.

The staff of the U. S. Army Surgical Research Unit is of the opinion that acute renal insufficiency does not occur when adequate resuscitative therapy is instituted. This opinion is strengthened by repeated experiences with patients having borderline or inadequate resuscitative therapy who are oliguric when first examined but respond rapidly to adequate intravenous therapy as evidenced by an adequate output of urine. In rare instances, the amount of intravenous colloids, electrolytes, and 5 per cent glucose in water administered far exceeds the amount dictated by any type of fluid formula developed for the treatment of burns before urine is produced in any adequate quantities. The physician must proceed with great care in these instances, since pulmonary edema, particularly in the older age groups, is likely to occur. Again, let it be emphasized that adequate resuscitative therapy immediately after thermal injury prevents the occurrence of renal insufficiency. Without adequate fluid replacement, the prolonged hypotension resulting can cause renal insufficiency similar to that observed in hemorrhagic shock.

Joint Complications

A previously active person on being immobilized throughout his entire body for prolonged periods of time will naturally suffer from this inactivity, particularly about the joints. A patient suffering from extensive thermal injury does not escape the consequences of his forced immobilization. The joints affected are not necessarily those involved in the most severe burns, nor is there any obvious selectivity in the involvement. Table 14 shows the occurrence of periarticular and fascial calcification about major points from 1950 through

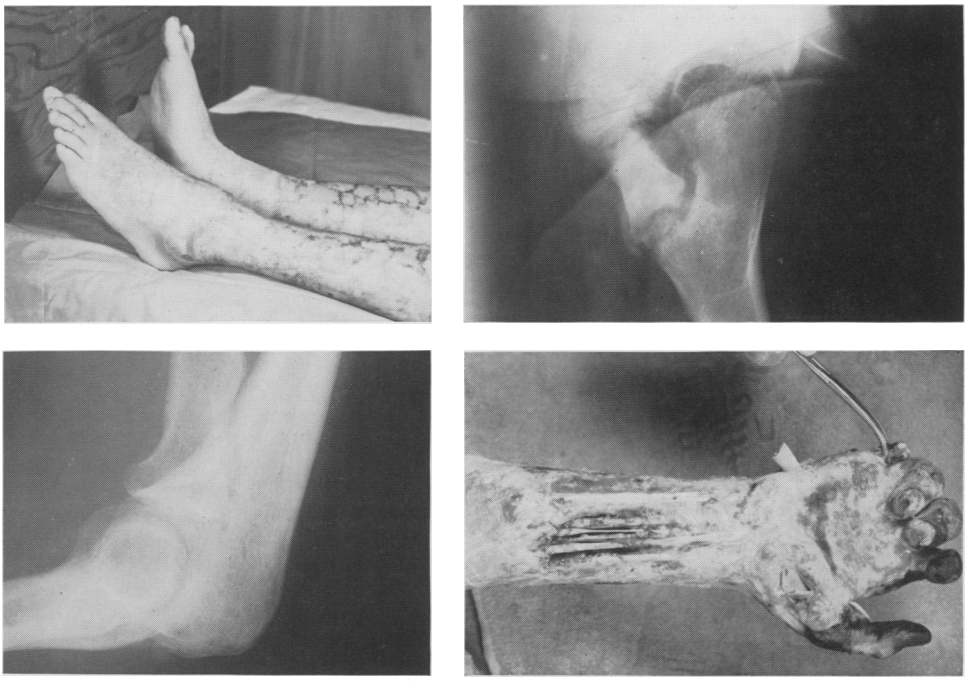


FIG. 15. (*Upper left*) Foot drop secondary to deep burn and prolonged tight dressings. After skin coverage brace was used. Function is slowly returning eight months postburn.

FIG. 16. (*Upper right*) Periarticular calcification. Note the extremely large, bony strut from trochanter to acetabulum. Surgical removal resulted in full range of motion.

FIG. 17. (*Lower left*) Periarticular calcification. The calcification can be noted on the extensor surface at the junction of ulna and humerus. There was only 20 degrees of motion of the joint, which is gradually improving with physiotherapy.

FIG. 18. (*Lower right*) Tendon destruction. This resulted from a burrowing infection beneath dressings which were left in place too long. The thenar and hypothenar spaces were liquefied by infection. Below elbow amputation was necessary.

1956, leading to varying degrees of limitation of motion but usually resulting in a rather marked impairment. The surfaces of the joints do not appear to be involved nor do the bones suffer from immobilization except for the osteoporosis attendant upon inactivity. The most characteristic finding is a faint periarticular calcification along fascial planes or in the region of the joint capsule. This condition is more and more evident on serial x-ray examinations; it may progress over a period of time to solid union between opposing bones as illustrated in the case of L. W. (Fig. 16). Admittedly, this is an unusual case because involvement of most joints does not progress to such a marked degree. The most common involvement is about the elbow. However, no survey of joints has been made. It may be that the apparent predilection for the elbow is because any impairment of this somewhat complicated joint is more readily appreciated. Figure 17 shows a typical, although rather indistinct, periarticular calcification that occurred about the elbow in one of the immobilized burned patients. Logically, it would seem that the earlier the mobilization of the joint, the less opportunity there would be for complications of this type. However, patients manifesting this involvement had immobilization of the elbow joint for periods varying from 21

TABLE 15. *Major Tendon Destruction, 1950–1956*

Year	Patient	Burn Agent	Involvement	Treatment	Result
1950	J. B.	Gasoline	Loss of extensors of dorsum of hands due to burn and infection.	Fusion of joints in position fo 30° flexion.	Marked impairment of function.
	T. M.	Grease	Destruction tibialis tendons due to burn and infection.	Attempted fusion of ankle failed. Low leg amputation.	Good stump.
	H. L. M.	Electricity	Destruction all flexors and muscles of left forearm.	Amputation.	Good stump.
1952	C. C. S.	Jet fuel	Destruction of extensors on dorsum both hands due to burn and infection.	Tendons excised. Skin graft. I. P. fused at 90° and M. P. at 30°.	Marked limitation of function.
	M. W.	Gasoline	Destruction of all forearm tendons and exposure all joints.	High forearm amputation and secondary closure.	Fair stump.
1953	J. F. M.	Flame thrower	Thermal destruction all finger extensors and joints exposed.	Graft coverage and splinting.	Severe deformity and ankylosis.
	A. O. M.	Jet fuel	Thermal destruction all finger extensors and joints exposed.	Amputation at proximal I-P joints and graft.	Markedly diminished function.
1954	S. D. O.	Gasoline	Thermal destruction of Achilles and tendons of dorsum of foot.	Graft coverage and later triple arthrodesis.	Able to walk with cane.
1955	G. B.	Electricity	Destruction of 3 and 4 flexors at wrist.	Graft coverage.	Only 30° flexion. May repair later.
	O. U.	Gasoline	Loss of peroneal tendon due to burn and infection.	Graft coverage.	Fusion later.
1956	P. R.	Hot metal	Thermal destruction flexor carpi ulnaris and radialis.	Graft coverage.	No limitation function.
	A. L.	Jet fuel	Loss of all flexors at wrist due to secondary infection present on admission 30 days postburn.	Mid-forearm amputation.	Fair stump.

to 210 days. Obviously, in cases similar to that of L. W., nothing short of surgical intervention is of value. Surgery was markedly successful in the case of L. W.

In patients having joint involvement, active motion and physiotherapy should be instituted initially and continued for as long as improvement occurs. No operative procedure should be contemplated unless no further improvement occurs in the range of motion in the joint. Surgical intervention is resorted to only if there is a good chance for improvement in the range of motion and the patient manifests a strong desire. The precise operative procedure undertaken will be dictated by the conditions found at the operating table. They may include removal of portions of abutting bone surfaces, capsulotomy or capsulectomy, and excision of the fibrous and calcified adhesions. In this group of major joint involvements, as in the hands, it is important that active physiotherapy be instituted as soon as possible in order to prevent the loss of the increased range of motion gained by the operative procedure.

Tendon Destruction

The joint surfaces and the periarticular structures of the major joints are not the only elements that suffer as a result of thermal injury and subsequent infection. Table 15 shows types of major tendon destruction that occurred from 1950 to 1956. The only superficial tendon structures escaping involvement to any significant de-

gree were the wide, thick tendons of the patella and triceps. The relatively superficial tendons of the hand, wrist, ankle and foot showed extensive destruction as a direct result of thermal injury and/or subsequent infection. The deep localized third-degree burns following electrical contact caused immediate destruction by coagulation necrosis at the time of the passage of electrical current. Other types of thermal injury, such as those caused by gasoline or fuel of jet aircraft, produce a partial destruction of the tendons and their paratenon and tendon sheath. The already tenuous blood supply to the tendon is destroyed, thus laying the groundwork for complete destruction by the infection that invariably ensues. The loss of tendons by bacterial autolysis is more frequent than by direct thermal destruction.

If at the time of initial examination (whether this be immediately after thermal injury or at a later date) the destruction of tendons is so extensive as to preclude any chance of functional activity, or if the structures supplied by the tendons such as the digits and hand are destroyed, no attempt should be made to preserve tendon function. Amputation is probably the treatment of choice. This is particularly true if widespread infection has destroyed the surrounding muscles and blood supply so that in all probability avascular necrosis of the tendons will occur (Fig. 18). Further delay in amputation may result in amputation higher up on the extremity possibly above a major joint and thus diminish the chances of adequate prosthetic substitution.

When the tendons are exposed, even though the burn destroys the normal sources of blood supply such as paratenon and tendon sheath, there is an occasional chance of maintaining the viability of the structure through granulations that may penetrate between the individual fibers of the tendon from adjacent muscle. Consequently, a surgeon should be in no great haste to remove what appears to be a nonviable tendon. This is particularly true if it is the only hope of retaining function to the distal organ of insertion. A surgical approach to tendons that have been exposed by thermal injury should be conservative as the primary object is to retain as much function as possible without jeopardizing the survival of the patient.

Soft Tissue Contracture

There can be significant limitation of functional activity and range of motion of any joint due to keloidal skin contracture in the area of burn, even if a joint is not affected by thermal injury, or if no periarticular involvement or interference occurs in tendon function. A contracture can also occur, as well as fibrosis of muscle and the connective tissue exclusive of joint capsules. Table 16 tabulates the occurrence of significance soft tissue contractures from 1950 through 1956. No joint appears to be immune to involvement. Some patients are more likely to have keloidal skin contracture than others; this is particularly true of the Negro race. In the Caucasian race, the clinical impression is that keloid formation is much more frequent in the upper extremities, trunk, face and neck than it is in the lower extremities. Also the burns caused by fuel from jet aircraft tend to produce more of a vesiculating, keloid-like scar.

Skin contractures are more likely to occur when mixed second-degree and third-degree burns involve areas about the joints and particularly when they cross flexion creases and lines of stress. Such a burn heals spontaneously by epithelization from the remaining skin islands and such healing is more likely to cause skin contracture than a graft. Indeed, if grafts are placed over flexion creases and folds and about joints in a manner so that the margins of adjacent strips of skin are at right angles to the long axis of the limb, there is little, if any opportunity for scar tissue to limit joint activity.

TABLE 16. *Significant Soft Tissue Contractures, 1950–1956*

Year	Patient	Burn Agent	Involvement	Treatment	Result
1952	H. B.	Gasoline	Thenar web contracture with adductor fibrosis.	Physiotherapy.	Marked limitation thumb abduction.
1953	P. E. A.	Flame thrower	Keloid flexion contracture of fingers.	Physiotherapy to keep joints mobile.	Correction upon maturation scar tissue.
	V. L. J.	Clothing fire	Severe neck contracture.	Early excision and graft.	Failed. Contracture rapidly recurred.
1954	L. E. B.	Gasoline	Flexion contracture right elbow and axilla.	Physiotherapy.	Minimal limitation of motion.
	J. R. P.	Phosphorus	Flexion contracture right elbow.	Physiotherapy.	No improvement.
1954	L. W.	Clothing fire	Flexion contracture, groin, bilateral.	Excised and grafted.	Good release and good range of motion.
	T. B.	Clothing fire	Adduction contracture hips, bilateral.	Physiotherapy.	Marked limitation.
1955	P. A. B.	Clothing fire	Keloid flexion contracture index finger.	Physiotherapy.	Joint mobile. Contracture remained.
	E. S. C.	Clothing fire	Flexion contracture both elbows.	Physiotherapy.	Joint mobile at 130°.
	K. O. R.	Hot water	Flexion contracture left popliteal.	Early excision and graft.	Rapid recurrence.
	J. W.	Gasoline	Severe neck contracture.	Early excision and graft	Failure with recurrence of contracture.
1956	M. B.	Clothing fire	Flexion contracture right antecubital.	Early excision and graft.	Good initially but some recurrence with 15° limitation of extension.
	A. W.	Gasoline	Thenar web contracture with adductor fibrosis.	Physiotherapy.	Marked limitation thumb abduction.
	N. H.	Gasoline	Thenar web contracture with adductor fibrosis.	Physiotherapy.	Marked limitation thumb abduction.
	J. H. S.	Gasoline	Flexion contracture left popliteal.	Early excision and graft.	Good with minimal limitation.
	H. P.	Gasoline	Thenar web contracture with adductor fibrosis.	Physiotherapy.	Marked limitation thumb abduction.
	A. S.	Gasoline	Moderate neck contracture.	None.	Revision later when scar tissue mature.
	V. F.	Clothing	Posterior axillary fold.	Physiotherapy.	Marked improvement with only 30° limitation motion.

The thenar space of the hand is unique in its involvement with fibrosis and contracture of the muscles secondary to deep burns of the dorsum of the hand. This woody induration and fibrosis results in an adduction of the first metacarpal and the medial rotation of the first metacarpal with marked limitation of motion. The exact cause of this is not known, although prolonged interference with lymphatic drainage and dependency aggravate and protract any hand difficulty. It may be that, as the deep burn on the dorsum of the hand interferes with the lymphatic drainage from the palm, it causes a precipitation of protein immediately after thermal injury. Later, due to prolonged stasis of the lymphatic drainage, the precipitate organizes and severely limits the function of the thenar muscles by fibrotic contracture. Microscopically there is considerable fibrosis of the adductor muscles and, even if skin contracture in

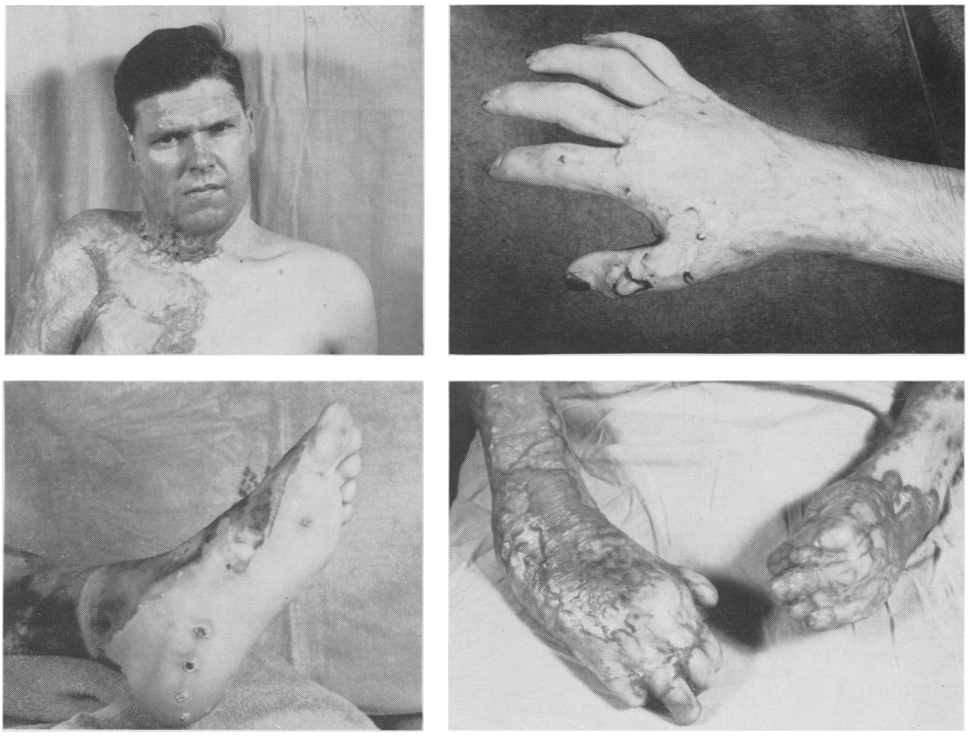


FIG. 19. (*Upper left*) Neck contracture. This resulted from phosphorus burn. The beard in the adjacent areas of deep second-degree burn caused repeated abscess formation.

FIG. 20. (*Upper right*) Thenar web contracture. In this case muscle fibrosis and not skin contracture is the cause. Note adduction and beginning medial rotation of first metacarpal. There is minimal functional impairment.

FIG. 21. (*Lower left*) Electrical burn. Points of exit of current through nails in shoe heel is readily demonstrated.

FIG. 22. (*Lower right*) Severe deformity and loss of substance from phosphorus burn. Chronic infection persists in these burns for months preventing any early functional reconstruction.

the thenar web is completely relieved, little increase in the range of motion can be obtained.

It is best to treat these soft tissue contractures expectantly with early active physiotherapy in an attempt to increase the range of motion of the involved joint or at least to prevent further decrease in the range of motion. As noted in Table 16, early excision and the grafting of soft tissue contractures is to be avoided if at all possible since the procedure is usually a failure because of rapid recurrence of the contracture. Failure is due primarily to the lack of a maturation of the scar tissue that obscures differentiation between the scar tissue and adjacent normal tissue. The incomplete removal of the scar tissue causes a rapid recurrence of the contracture beneath the graft.

Usually it requires from 12 to 18 months after thermal injury for a complete maturation of the scar tissue to take place. Maturation is manifested by the disappearance of the vascularity, loss of the blanching on stretching, softening of the scar tissue itself, and a ready differentiation between the scar tissue and adjacent normal tissue. Dictated by the conditions, a good result may be expected both cosmetically and functionally after surgical excision and re-

TABLE 17. *Conversion of Donor Sites and Burns by Infection, 1950–1956*

Year	Patient	Complication	Treatment	Result
1952	J. E. E.	Donor sites converted to full thickness loss by infected dressings in transit.	Cleaned with wet soaks and grafted.	Good coverage.
1954	F. G. M.	Donor sites converted to full thickness loss by infected dressings in transit.	Cleaned with wet soaks and grafted.	Good coverage.
	M. M.	Donor sites converted to full thickness loss by infected dressings in transit.	Cleaned with wet soaks and grafted.	Good coverage.
	J. M. P.	Conversion of 2d degree to 3d degree by infection due to "open neglect."	Cleaned with wet soaks and grafted.	Good coverage.
	R. S.	Conversion of 2d degree to 3d degree by infection due to "open neglect" and conversion of donor sites by neglect in transit.	Cleaned with wet soaks and grafted.	Good coverage.
1955	R. P. C.	Conversion of 2d degree to 3d degree by infection due to "open neglect" and conversion of donor sites by neglect in transit.	Cleaned with wet soaks and grafted.	Good coverage.
	A. J. S.	Conversion of donor sites and 2d degree burn to full thickness loss by infrequent dressing changes in transit.	Cleaned with wet soaks and grafted.	Good coverage.
	O. U.	Conversion of donor sites and 2d degree burn to full thickness loss by infrequent dressing changes in transit.	Cleaned with wet soaks and grafted.	Good coverage.
1956	M. B.	Conversion of donor sites and 2d degree burn to full thickness loss by infrequent dressing changes in transit.	Cleaned with wet soaks and grafted.	Good coverage.
	J. C.	Conversion of 2d to full thickness loss on back due to inability to turn patient because of respiratory difficulty.	Cleaned with wet soaks and grafted.	Good coverage.
	V. F.	Conversion of 2d to full thickness over 15% of body due to "open neglect" at previous hopsital.	Debridement, homograft than autograft.	Good coverage. Homograft survived 57 days.
	J. F.	Conversion 2d degree to full thickness by "open neglect" at previous hospital.	Cleaned with wet soaks and grafted.	Bilateral ectropion.
	J. C. H. B. R. S. W. H. V. F.	Spotty conversion of donor sites and recently grafted areas to full thickness loss due to *B. strep* infection.	Cross contamination from hubbard tank, P-T discontinued. I. M. penicillin, wet soaks.	Spontaneous epithelialization.
	L. H.	70 year old. Conversion of 2d to full thickness by *B. strep.*	Cleansed with soaks and grafted.	Good coverage.

placement by grafts of varying thicknesses. Figure 19 shows a scar tissue contracture of the neck occurring at the junction of graft and mixed second-degree and third-degree burn. Some increase in motion was obtained with physiotherapy. No surgical intervention will be carried out on this patient until further maturation of the scar tissue takes place.

The fibrosis of the adductor muscles of the thenar web space do not lend themselves to easy correction. Active physiotherapy should be instituted as early as possible to increase the range of motion and decrease the amount of contracture. Skin contracture of the thenar web should be corrected. Although release of this will not affect the muscular contracture if it is present, if the skin contracture exists alone considerable increase in function and range may be obtained. The woody, indurated sensation one elicits upon palpation of the

thenar space gives a good indication that release of any skin contracture will be of no avail. Figure 20 illustrates a thenar web contracture resulting from a deep burn of the dorsum of the hand. Some skin contracture of the thenar web was released by excision and grafting, but the procedure did not increase the range of motion as the limitation was due primarily to fibrosis of the adductor muscle. Some surgeons have advocated that a deep V-wedging of the adductor muscle be carried out in order to free the first metacarpal and widen the thenar web space. However, if flexion of the interphalangeal and metacarpophalangeal joints and the carpometacarpal joint is adequate and no marked degree of medial rotation of the first metacarpal is present, it is unlikely that widening of the thenar space by this procedure would add much functional improvement and it would weaken adduction. Actually, it is unlikely that any improvement other than that gained by active physiotherapy would be of any real functional value.

Wound Infection and Conversion

Control of infection in a burned patient is of primary importance in order to prevent the development of septicemia. Treatment of infection on a superficial burn surface is of great importance from the standpoint of avoiding conversion of second-degree burn to third-degree burn. Unless infection is controlled, it increases the amount of deformity, morbidity, and the length of hospitalization. Table 17 shows the important instances occurring from 1950 through 1956, in which conversion of donor sites and burns was caused by infection. The most important microorganism involved in this surface infection is Group A beta hemolytic *Streptococcus,* a species sensitive to penicillin. It is fortunate that only this species of *Streptococcus* is a pathogen; therefore only small amounts of penicillin need be given systemically as a prophylactic measure (300,000 units daily or b.i.d.). If this micro-organism is cultured from the surface wound, penicillin administered systemically in the same dosage schedule will usually cleanse the wound of a penicillin-sensitive *Streptococcus* within 48 to 72 hours. It is impossible to sterilize the burn wound completely by systemic or local antibiotics. If good surgical care of the burn wound is carried out, the physician need not be concerned over other micro-organisms on the surface of the wound, no matter how prolific they may be.

Conversion of donor sites and second-degree burns to full-thickness skin loss is primarily due to poor surgical management of the wound. If dressings over donor sites or second-degree burn wounds are allowed to remain in place for more than four or five days, the bacterial autolysis taking place beneath the burn dressing will convert partial- thickness loss to full-thickness loss within 12 to 24 hours. It is to be noted from Table 17 that only one patient treated in this installation from almost the beginning of his injury suffered conversion of a donor site or a second-degree burn to full-thickness skin loss. In other cases, wounds were converted as a result of neglect of the dressing or exposed burns during the period of initial hospitalization elsewhere or during the period of transit which on some occasions spanned five to ten days.

It is extremely important in the treatment of second-degree burns by the so-called open method that one does not allow the routine treatment to become "open neglect." The exposure method must be abandoned whenever infection occurs beneath the crust of a second-degree burn. The area is best treated by applying wet saline soaks and changing them as frequently as necessary. Whenever an area of softening occurs or purulent material appears beneath the margins of the crust, the wound must be treated promptly. Failure to perform immediate therapy causes the disssection to

continue beneath the crust. The superficial appearance of an intact, uninfected crust often hides this dissection while, at the same time, a bacterial autolysis is destroying small islands of epithelium that would otherwise regenerate without difficulty. This same process occurs beneath burn dressings and dressings of donor sites that are not changed frequently and causes marked increase in morbidity, mortality, deformity, and work for every one concerned. The exposure method of training donor sites minimizes the chance of infection.

Beta hemolytic *Streptococcus*, sensitive to penicillin, can be a considerable problem in a burn ward if one is not aware of the possibility of its occurrence and the rapid development of complications due to its presence. It is important that a culture of the surface of the burn wound be taken prior to grafting because this particular microorganism will markedly decrease the percentage of graft take and destroy the epithelium of recently placed grafts, healing second-degree burns, and donor sites. In aged patients having thin atrophic skin that lacks dermal papillae, even superficial second-degree burns can be and have been converted to areas of full-thickness skin loss by the presence of beta hemolytic *Streptococcus* sensitive to penicillin.

One of the most dramatic episodes occurring in this installation involved five patients who were receiving physiotherapy and being exercised in a large Hubbard tank. One of the patients developed a streptococcal infection of the healing and grafted burn surfaces. This infection was transmitted to the other four patients via the Hubbard tank in spite of thorough cleansing of the tank and scrubbing with zepheran between the treatment of each patient. All five patients developed spotty areas of full-thickness skin loss of recently grafted areas and healing second-degree burns. This was detected immediately on the Ward, physiotherapy was discontinued, and patients were placed on 300,000 units of penicillin twice a day. Culture did not reveal streptococcus on the wound surfaces after 48 hours and epithelization was occurring quite rapidly. Five to seven days after the onset of the infection, with its attendant destruction of the surface epithelium, the epidemic was controlled and re-epithelization of the areas of skin loss occurred.

Electrical Burns

Burns resulting from high voltage electrical contact are quite different from the usual thermal injury. If the patient does not succumb immediately from respiratory arrest and cardiac failure, the physician will find that the resulting burn is extremely deep. Sometimes it is widespread, but usually it is well localized. It is common to see burns occurring only at the points where the current enters and leaves the body. Typical of such a burn is a deep third-degree burn across the palm of the hand where the individual has touched a high voltage wire as the point of contact. He may also sustain deep punctate third-degree burn over the tips of all the toes and a horseshoe-like distribution of these same deep punctate burns about the heel where the current has passed through the foot via the nails attaching the heel to the shoe. This type of burn is illustrated in Figure 21.

The small localized areas of deep third-degree burn are of no great consequence as far as survival of the patient is concerned. Considerable disability may result, however, if the burned areas involve regions over superficial joints or areas that are critical from the standpoint of function. Although localized, the burn may cause destruction of underlying bone, tendon, nerve, and blood vessels, and it may be of such depth and severity that any organ involved may be completely deprived of its functional use, innervation, or blood supply.

TABLE 18. *Complications of Electrical Burns, 1954–1955*

Year	Patient	Complication	Treatment	Result
1950	H. B.	Osteo of occipital bone due to deep burn with secondary meningitis.	Sequestrectomy, tantalum cranioplasty, graft, and drainage dural abscess.	Good coverage. Meningitis cleared rapidly.
	H. L. M.	Fourth degree burn of hands with loss of tendons of forearm.	Amputation.	Good stump.
1952	D. G. B.	Aneurism right radial artery with rupture.	Amputation below elbow.	Good stump.
	W. D. J.	Ulnar hypesthesia with elbow burn.	Grafting.	Slow, incomplete return with pain on use of hand.
1953	L. W. C.	Deep muscle burn with clostridium butyricum cultured.	Debridement and antibiotics.	Cleared rapidly.
1955	G. B.	Osteo first metacarpal and exposure first carpal metacarpal joint.	3 graftings failed.	Finally covered from margins. Stiff wrist.
	R. R. W.	Severed brachial artery and nerves of arm.	Amputation.	Good stump.
	L. G. M.	Thrombosis right popliteal and left bracheal arteries with gangrene of extremities.	Right mid-thigh and left mid-arm amputation.	Died of septicemia.

Frequently amputation of the involved limb may be necessary. These possibilities are adequately demonstrated in Table 18, a tabulation of the severe complications of electrical burns occurring from 1950, through 1956.

Frequently, when an electrical current passes through the body and particularly when the burn is widespread, the subcutaneous tissue appears to explode through the overlying skin and the skin itself appears somewhat inspissated either as a result of electrical current or the tremendous heat generated. The vessels may have their continuity actually disrupted, the vessel wall may be weakened with resultant aneurysm formation, or as most frequently happens, the blood within the lumen of the vessel is immediately clotted with resultant gangrene of the extremity distal to the obstruction.

The treatment of an electrical burn will vary with the depth and extent of the burn, the area involved, and the immediate complications resulting from the electrical current. The small localized areas of third-degree involvement may be excised almost immediately or in the first few postburn days and then be grafted immediately. The burn wound may appear well circumscribed; and an obvious line of demarcation between burned and unburned tissue may appear to be present. However, this may be an invalid assumption. Frequently, further necrosis of surrounding skin takes place and grafting immediately after excision of the burned area may not be successful.

In the early excision of localized areas of third-degree electrical burns it is necessary to be assured that a wide margin of excision is performed in order to prevent the failure of the graft take. Obviously, if these small areas overlie important functional structures such as flexor tendon of the hand, it is better to excise early; then the wound is covered with a clean surgical dressing. The wound is watched carefully for several days to determine whether further necrosis of surrounding tissue takes place; and debridement is performed accordingly. The necessary grafting can then be carried out with much less chance of functional impairment. Extensive areas of third-degree electrical burn should be treated essentially as extensive areas of third-degree burn from any cause. Complications may result that

TABLE 19. *Complications of Phosphorus Burns, 1950–1956*

Year	Patient	Complication	Treatment	Result
1950	O. C. H.	Bilateral corneal burn. Cataract of right lens due to rupture of lens.	Local only. Immobilization.	Light perception only. Slight improvement.
	R. C. H.	Fibrosis all I-P and M-P joints.	Physiotherapy.	Ankylosis all joints.
1952	R. J.	Corneal ulcer and leukoma, O. S. Ectropion.	Local ointment and irrigation. Overcorrected.	Ulcer healed. Vision diminished. Good.
1953	B. M. T.	Bilateral ectropion.	Overcorrected.	Cornea well protected.
	W. A. E.	Deep joint burns index and middle digits.	Amputation through proximal I-P points.	Good healing—poor M. P. motion.
	H. E.	Bilateral ectropion.	Overcorrected.	Cornea well protected.
1954	L. C. W.	Phosphorus poisoning with icterus and RBC hemolysis.	Bed rest and diet.	Cleared in 15 days.
	P. A. Y.	Bilateral corneal burn.	Local.	Bilateral scarring with blurred vision.
1955	D. M.	Deep joint burn I-P joints all digits right hand.	Amputation through proximal I-P joints	Markedly limited function.
	D. W.	Deep joint burns I-P joints all digits bilaterally.	Amputation through proximal I-P joints.	Markedly limited function.

are peculiar to electrical burns, such as extensive thrombosis of deep vessels, direct destruction of the nerves and blood vessels, and incineration of muscle groups, frequently requiring amputation of the extremity distal to the point of maximum injury.

A severe electrical burn in Patient L. G. M., who contacted an 8,000-volt electrical high tension wire, resulted in a thrombosis of the right popliteal and left brachial arteries and gangrene of the extremities distal to these points of obstruction. Right mid-thigh and mid-arm amputations were required, as well as a trans-metatarsal amputation of the left foot. The patient developed a septicemia caused by *Pseudomonas,* and a massive, bleeding Curling's ulcer, and died.

Phosphorus Burns

Chemical burns resulting from white phosphorus are rarely seen in civilian practice, but they constitute the major type of chemical burns among military personnel. These burns result not only from combat injuries but also from accidents caused by the explosion of grenades and shells containing white phosphorus. Table 19 tabulates the important complications of phosphorus burns observed from 1950 through 1956. White phosphorus is the most important single cause of burns of the cornea. In spite of early treatment, invariably such a burn causes diminution in visual acuity and frequently produces complete blindness.

The immediate treatment of a phosphorus burn consists in the application of saturated copper sulphate solution to inactivate the phosphorus and meticulous removal of all fragments of phosphorus. In soft tissue burns, but not in corneal burns, treatment consists of debridement of the area to assure complete removal of any remaining fragments of phosphorus. Subsequent treatment is directed toward the prevention of any significant infection and the correction of deformities that occur (Fig. 22). In burns of the cornea, it is extremely important that local irrigation and application of antibiotic ointments be carried out in order to protect the cornea from further damage.

If this is done and the initial burn has not been too deep, blurring of vision will be the usual extent of damage. It is possible that corneal grafts may be applied at a later date, provided Descemet's membrane and the lens have not been involved.

The complications of intravenous therapy utilizing polyethylene tubing in the femoral vein are detailed elsewhere in this series. The problems of septicemia have been fully described by Hummel and Artz and are partially discussed in another section to follow.

Summary

The complications encountered in the treatment of 1,000 burns are described. These resulted from both the thermal injury and the treatment of the burn. Present methods of treatment are described along with the early follow up. It is emphasized that there is no difference between the burn wound and any other wound; both must be kept as surgically clean as possible. Awareness of possible complications may minimize their future incidence and allow for early recognition and prompt treatment.

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