Arterial Vascular Occlusion and Devitalization of Burn Wounds *

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ALTERED vascular permeability in tissue subjected to severe thermal injury has been demonstrated by a variety of dyes.[1, 3, 4, 10, 11] However the patterns of arterial occlusion and revascularization have not been delineated. In this study, standard third degree [14] and second degree [7] burns were inflicted on rats. Radiopaque injection technics were employed and the sequence of arterial occlusion and revascularization determined. Clinical fatalities with lower extremity burns were studied and the importance of vascular occlusion related to the devitalization of the tissue.

Methods

One hundred Sprague-Dawley rats were anesthetized with Nembutal, 25 mg./Kg., placed in a burning device which exposed 20 per cent of the total body surface on the dorsum of the back, and immersed in boiling water for 3 seconds or 10 seconds to create second or third degree burns, respectively.[7, 14] Animals were sacrificed immediately after burning, 24 hours postburn, 48 hours postburn and 1, 2 and 3 weeks postburn. Following sacrifice the descending thoracic aorta was cannulated and saline injected at 120 mm. mercury pressure and the thoracic inferior vena cava severed. When clear fluid emanated from the vena cava a modified Schlesinger mass,[9] which does not traverse the capillary bed, was injected at 120 mm. mercury pressure. After fixation of the animal in formalin, the burn eschar, subcutaneous tissue and panniculus carnosus were dissected from the deep fascia and x-rayed. Ten animals which were not burned were similarly injected as normal controls.

The femoral artery was cannulated in clinical fatalities with lower extremity burns. A similar injection procedure was performed. X-rays were taken of the extremities and of transverse cut sections which included eschar, subcutaneous tissue and deep fascia. In order to delineate bacteria, microscopic examination of Giemsa-stained sections was performed on all specimens.

Results

The normal arterial vasculature of the rat back contains four large arteries which penetrate from the deep fascia and arborize in a network of anastomotic branches similar to the subpapillary plexus in man [8] (Fig. 1a). This arterial plexus lies between the panniculus carnosus and deep subcutaneous tissue.

Experimental Third Degree Thermal Injury. In the immediate period following third degree thermal injury most of the arterial vascular network is occluded and only the large penetrating vessels with

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their major branches remain patent (Fig. 1b). Twenty-four hours later the entire eschar is devoid of patent arterial vasculature (Fig. 1c). Forty-eight hours postburn the devitalized eschar remains the same (Fig. 1d). Granulation tissue containing capillaries whose diameters are large enough to accept the injectate mass first appears 1 week after burning. At 2 weeks this vasculature has increased and at 3 weeks a great portion of the eschar contains a new vasculature architecture (Fig. 1e). However the transverse cut surface of the eschar reveals a lack of patent vasculature in the superficial regions of the eschar even at this stage (Fig. 1f).

Microscopy. Immediately following third degree thermal injury the arterial vasculature of the subpapillary plexus contains a dense red-cell mass which is adherent to the arterial intima (Fig. 2a) with many of the cells undergoing hemolysis (Fig. 2b). Some of the subpapillary plexus is occluded by blood coagulum (Fig. 2c) and a variety of stages between red-cell impaction and coagulate obstruction are present.

Later the red cells layer against the intima and fibrin strands interpose between the layers. The arteries demonstrating luminal coagulation, however, remain unchanged except for progressive necrosis of the arterial wall (Fig. 2d).

A parallel is seen in the capillaries surrounding the base of the hair follicle shaft where more intense coagulation and rupture, with extravasation of blood into Huxley's layer, are present.

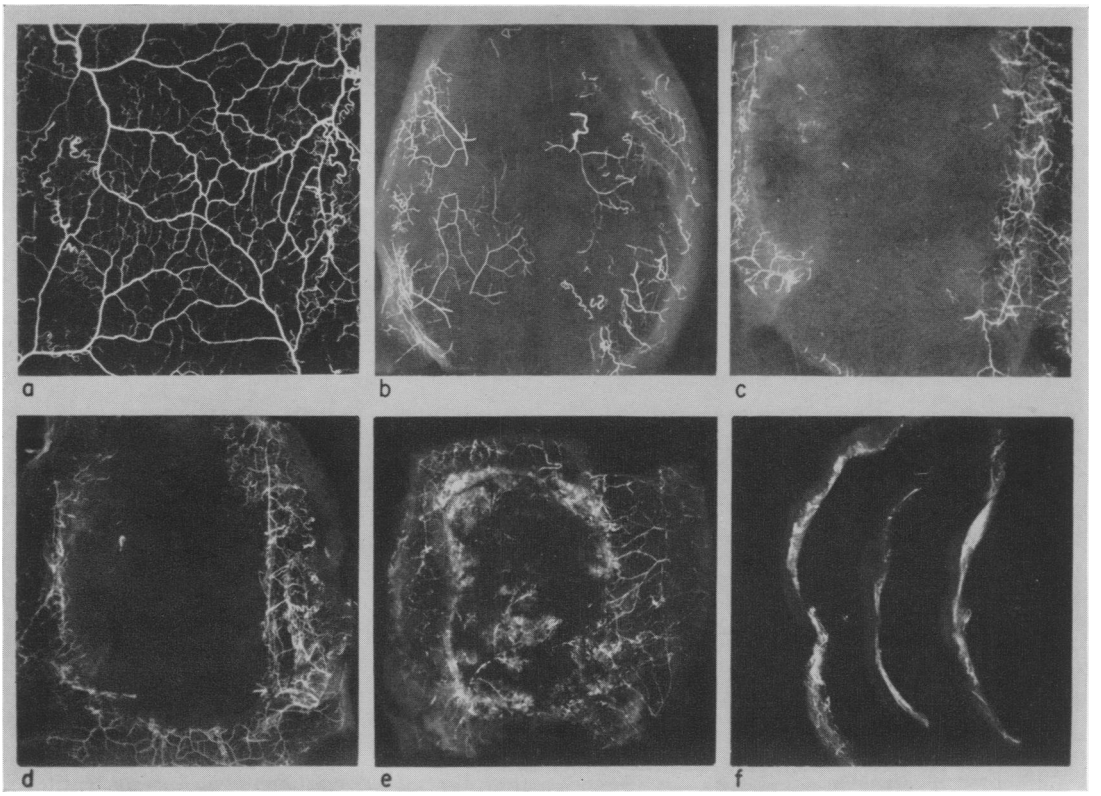

FIG. 1. a) Normal arteriogram—rat back (note the penetrating arteries at each corner). b) Following third degree burn. c) 24 hours post-third degree burn. d) 48 hours post-third degree burn. e) 3 weeks post-third degree burn with revascularization by granulation tissue. f) Cut surface of (e) with eschar (left) and revascularization beneath.

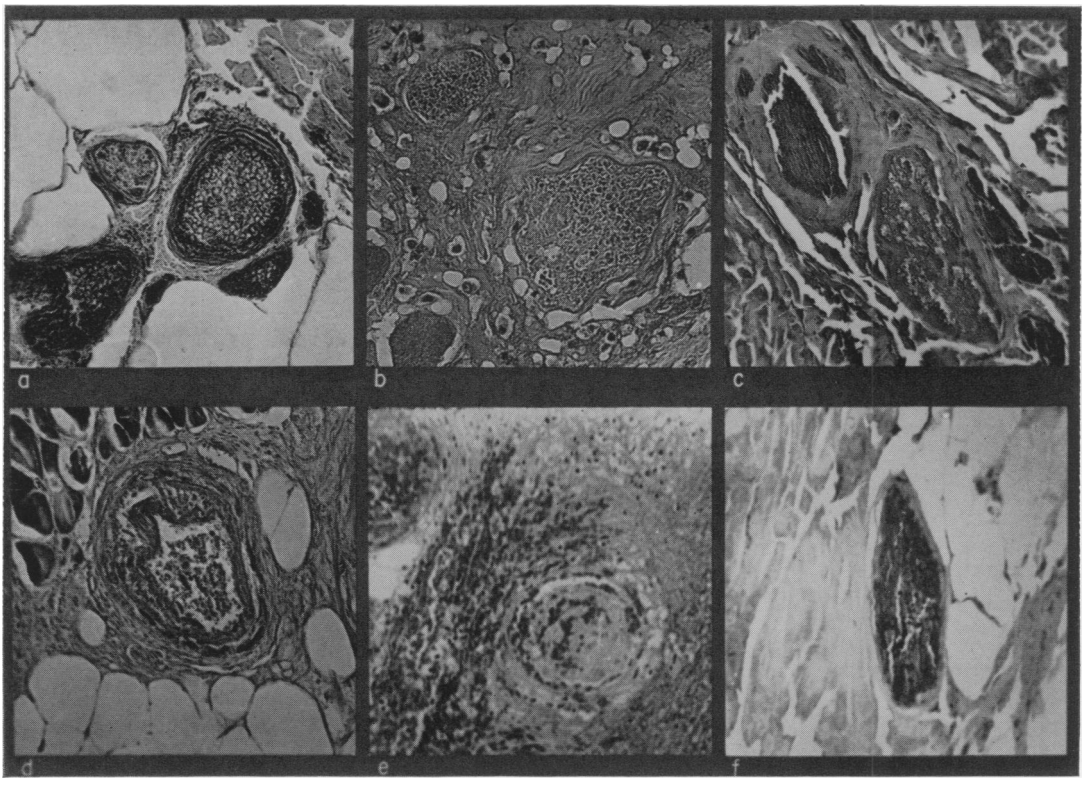

FIG. 2. a) Red-cell mass adherent to arterial intima following third degree burn. b) Hemolysis of red cells at a later post-third degree burn period. c) Thermal coagulation of blood within an artery immediately after third degree burn. d) Arterial necrosis following third degree burn. e) Clinical fatality with red-cell masses and hemolysis. f) Clinical fatality with thermal coagulation of blood.

The inflammatory reaction is well established 48 hours postburn and is limited to the connective tissue layer beneath the panniculus carnosus. No inflammatory reaction is seen in the dermis except in a few focal regions, usually at the edge of the burn where some of the arteries are patent. At the end of the first week the inflammatory process has progressed and granulation tissue is present but the magnitude of revascularization is not pronounced.

Three weeks after third degree burn the granulation tissue with marked revascularization is primarily situated beneath the panniculus carnosus, much of which is necrotic. The eschar is devoid of a significant inflammatory response.

Second Degree Thermal Injury. Following second degree thermal injury arterial vascular occlusion is similar to but not as intense as third degree injury (Fig. 3a). During the 24 and 48-hour period following the injury the patency of some of the subpapillary arterial network is re-established (Fig. 3b, c). One week postburn total restoration of the normal pattern of the arterial vasculature and other vascular channels associated with foci of granulation tissue are present (Fig. 3d).

Microscopy. In the immediate period following second degree thermal injury the vascular occlusion is similar to that seen in third degree injury but not as diffuse and many arteries never become occluded. The capillaries surrounding the hair follicles vary and are both patent and occluded.

The inflammatory response is not limited to deep fascia but is seen in many regions

in the dermis and sometimes in the upper stratum of the eschar. Granulation tissue appears at the same time postburn as in third degree injury; however, revascularization is accomplished by an inapparent mechanism whereby the original arterial vasculature is re-established at 1 week. The magnitude of capillary vasculature associated with granulation tissue at this stage is not great.

Clinical Thermal Injury. In clinical thermal injury the components of second and third degree injury are mixed. The arteriogram of an extremity does not always suggest arterial vascular occlusion (Fig. 4a); however, transverse sections including eschar, subcutaneous tissue and deep fascia reveal a diverse pattern of arterial vascular disarray ranging from complete occlusion to focal alterations within the same wound (Fig. 4b, c, d). The persistence of arterial vascular occlusion in the third degree injury has been documented as long as 30 days after thermal injury.

The arterial and inflammatory pattern seen in both second and third degree experimental injury is present in a random distribution in clinical injuries. The lack of inflammatory response in the eschar is associated with occluded arteries. Both coagulated and thrombotic occlusions are present (Fig. 2e, f). In regions of second degree injury vascular patency is in marked contrast to the multiple occlusions in third degree injury. Avascular tissue extends to a depth of 1.5 to 2.0 cm. in many regions of a severe thermal injury.

Discussion

Experimental third degree burn injury produces a complete loss of arterial vas-

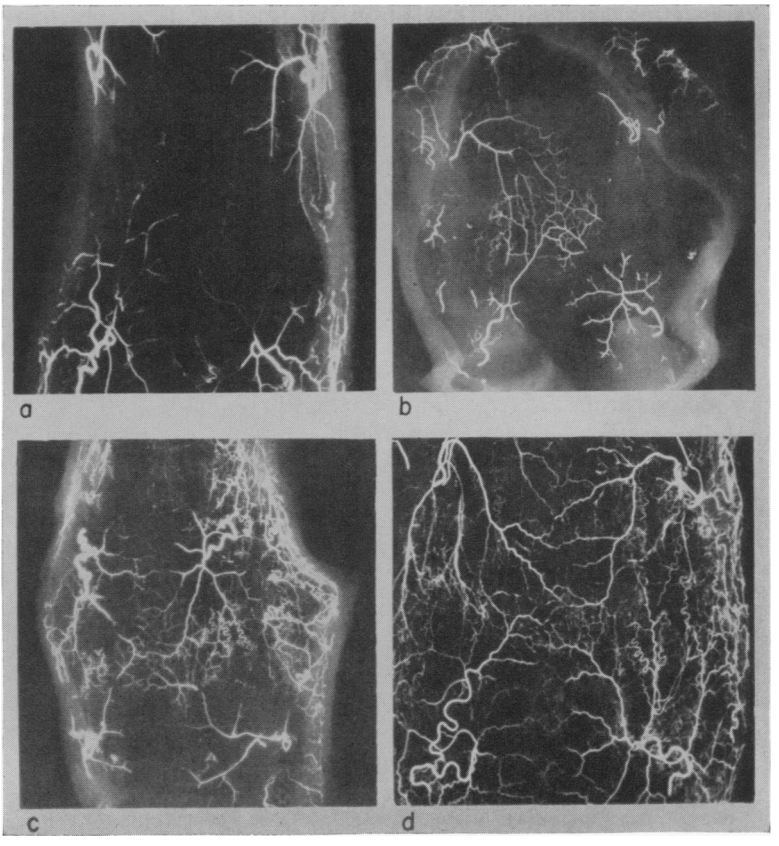

FIG. 3. a) Arteriogram, post-second degree burn. b) 24 hours post-second degree burn. c) 48 hours post-second degree burn. d) 1 week post-second degree burn.

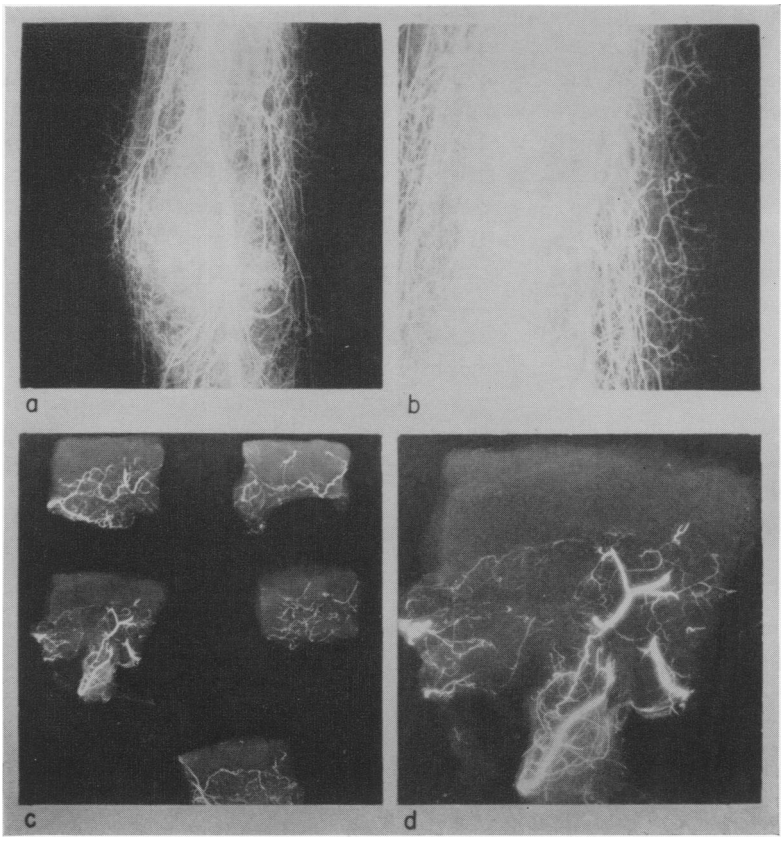

FIG. 4. a) Arteriogram of clinical fatality with third degree burned extremity. b) Soft tissue at edge of arteriogram suggesting arterial vascular occlusions (right). c) Dissection with eschar at top and fascia at bottom, demonstrating arterial vascular occlusion. d) Close-up to demonstrate depth of arterial avascularity.

culature at 24 hours postburn. The slow process of revascularization beginning at 1 week is incomplete as late as 3 weeks after injury. The inflammatory response is limited to the deep fascia as is granulation tissue which is its ultimate product.[2]

Experimental second degree thermal injury demonstrates a capacity to maintain an inflammatory response beneath the eschar and fully restore the arterial vasculature 1 week postburn.

Thus, the patient with second degree thermal injury exposes to the bacterial environment a tissue which has the capacity to produce an inflammatory response, preserve nutrition and support reconstruction of remaining cellular elements.

In contrast, the devitalized tissue of third degree thermal injury offers an excellent environment for bacterial growth and penetration. The lack of inflammatory reaction beneath the eschar is often directly related to thrombosis of vessels. Absence of granulation tissue beneath the eschar characterizes this type of injury. This absence of circulation and granulation tissue permits bacteria to proliferate in large numbers unassaulted by the host response and leads to burn wound sepsis in severe thermal injury.

Areas for clinical consideration implicit in the avascular lesion of severe thermal injuries are: 1) prevention of thrombosis, 2) early route for debridement of dead tissue, 3) risks inherent in the use of dressings and 4) appropriate antibiotic therapy in the prevention of burn wound sepsis.

The thermal coagulation of blood within the vasculature is probably irreversible. However, the red-cell mass conglomerates

(these later hemolyze) which occlude the circulation might be prevented by anticoagulant therapy.

Thrombo-emboli appeared in 28 per cent of 54 autopsy cases performed in thermally injured patients at this unit during 1963.[6] This incidence may be diminished by the same therapeutic regimen. To avoid both the problems of thrombosis and embolization, immediate utilization of preventive measures is suggested by the pathophysiology.

Burn wound excision as a one-stage procedure has as a foundation the removal of the devitalized tissue by debridement (thereby avoiding the template of infection) and homograft covering of a competent vascular bed.[12]

It is our clinical impression that dressings enhance the potential for burn wound sepsis. The common clinical practice of dressing burn wounds is not indicated in large injuries since the combination of enhanced potential and the mass of devitalized tissue may allow sepsis to become overwhelming. In second degree injuries or small areas of third degree injury, sepsis can usually be avoided while dressings aid in eschar separation for early grafting.

Parenterally administered antibiotics have failed to prevent burn wound sepsis.[5] Failure of diffusion due to local arterial occlusion may account for this in part. The evaluation of topical chemotherapy to prevent burn wound sepsis is justified by the pathologic state of burn wounds.

Summary and Conclusions

Experimental third degree thermal injury produces progressive arterial occlusion in the subcutaneous tissue which is complete 24 hours after injury and associated with a lack of inflammatory response. Inflammation is present in deep fascia and granulation tissue, its end product, later is apparent in this region. Revascularization of the devitalized third degree burn requires 3 weeks; the superficial regions of the eschar remain devoid of patent vasculature. The inability of the devitalized wound to deliver a host defense and the slow process of revascularization form the template for burn wound sepsis.[13]

Experimental second degree thermal injury produces a temporary arterial occlusive process. Arterial patency is restored 1 week after injury. The inflammatory reaction is present in the dermis and superficial regions of the eschar. There is subsequent appearance of granulation tissue. Delivery of host defenses, persistence of some patent arterial vasculature throughout the injury and early revascularization accompany the restoration of second degree thermal injuries.

Clinical fatalities with mixed second and third degree thermal injuries are analogous to the experimental models. Arterial occlusion, lack of inflammatory response and devitalization of third degree burn wounds has been demonstrated. A lack of granulation tissue beneath the eschar, the absence of host defenses and the slow process of revascularization of the devitalized tissue constitute the pathologic state of severe burn wounds. The demonstration of arterial vascular occlusion and devitalization of burn wounds suggest approaches to both clinical and experimental studies of burn wound therapy.

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