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CHAPTER 24

VASCULAR ABNORMALITIES ASSOCIATED WITH THERMAL AND ELECTRICAL TRAUMA

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Although direct injury to large vascular structures is rare following thermal injury, microvascular changes in both the wound and remote sites, such as the lungs or gastrointestinal tract, contribute to the morbidity and mortality associated with the underlying injury.

VASCULAR CONSEQUENCES OF BURN INJURY

Exposure of tissue to temperatures in excess of 43° C results in cell injury and cell death due to the direct effects of heat as well as the effects of humoral mediators secreted by the endocrine system and liberated by the injured tissues. The net result of these changes is edema formation due to an increase in capillary filtration representing the summation of altered microvascular pressures and increased microvascular permeability.

The magnitude of edema formation secondary to these microvascular changes is large enough to result in significant hypovolemia in burns that involve more than 25% of the total body surface area. Pitt et al., in studies of microvascular, hemodynamic, and permeability changes in a canine hind paw model of scald injury, found that, during the first 3 hr following burn injury, increased capillary pressure, as indexed by a 74% decrease in pre- to postcapillary vascular resistance, accounted for 53% of the increase in capillary filtration. In those studies, increased permeability accounted for 47% of the increase in early postburn capillary filtration, with 49% of the microvascular filtrate passing through large, 400-A nonsieving pores.[1]

The duration of this response remains uncertain and may be related to burn size. Brouhard et al., using radioisotopes and autoradiographic techniques, noted that transcapillary sieving of albumin molecules had essentially ceased by 8 hr afterburn injury.[2] Conversely, Brown et al., using a murine model of a more extensive burn injury, found that the rate of entry of albumin into the burn wound was increased throughout the first postburn day, and that burn wound water and albumin content were maximal 24 hr following injury.[3] The total albumin pool size stabilized between 24 and 72 hr afterburn injury, indicating a state of dynamic equilibrium between the burn wound and plasma albumin pools, with the quantity of albumin transferred out of the wound equal to that entering the wound. Those investigators also observed that postburn hyaluronidase treatment resulted in a lesser increase in the albumin and water content of the burn wound, a finding implicating changes in the biochemical and perhaps even physical characteristics of interstitial tissue in the dynamics of burn wound edema. Lund et al. reported that interstitial fluid hydrostatic pressure becomes strongly negative in burned dermis within 1 hr of injury and causes tissue imbibition.[4]

In addition to direct thermal injury, a variety of humoral factors have been implicated in the pathogenesis of edema in both the burn wound and remote tissues. These factors include products of complement activation, arachidonic acid metabolites (principally thromboxane A_2 and leukotrienes), histamine, fibrin degradation product D, activated proteases, and the lysosomal enzymes and oxygen radicals released by activated neutrophils.[5–10] Laboratory studies have demonstrated that plasma histamine concentration increases within 1 min of thermal injury, and the increase is proportional to the extent of surface area injured. A partial-thickness burn evokes a single time-related peak of plasma

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histamine concentration, in contrast to the biphasic, more prolonged elevation of plasma histamine concentration after a full-thickness injury of equal size.[11]

Boykin et al., using a hairless mouse model, found that a scald burn of one ear was associated with a delayed (2 to 6 hr) increase in water content of the contralateral, unburned ear.[12] That edema could be inhibited by cold water treatment, high doses of cimetidine, and preinjury histamine depletion by polymyxin B. Those investigators interpreted their findings to implicate histamine as a mediator of delayed edema formation in unburned tissue because the cold water treatment appeared to reduce histamine release from the burned tissue. Additional studies by Yurt et al. have demonstrated that preburn administration of polymyxin B degranulated mast cells and caused a significant reduction in the number of mast cells per vessel in the burn wound. Those animals had no associated decrease in wound edema.[13] These studies suggest that elevated circulating levels of histamine contribute to the systemic vascular response to burn injury but are of little importance in terms of early edema formation in the burn wound.

The effects and importance of various humoral factors on postburn alterations of vascular permeability have been studied by several investigators. Arturson and Jonsson were able to decrease lymph flow by 40% in the scalded paw of a dog when the animal was pretreated with indomethacin.[14] Studies from that same laboratory showed that substance-P-like immunoreactivity in dog paw lymph significantly increased within 5 to 10 min of scalding.[15] Additionally, Lundberg et al. reported that systemic pretreatment with a substance-P antagonist significantly reduced local extravasation of Evans blue following scald injury.[16] Because tissue response to substance-P was resistant to histamine-blocking agents, its effect did not appear to be mediated by histamine release.

The local effects of thromboxanes have been studied by Heggers et al., who demonstrated that postburn topical application of thromboxane synthetase inhibitors reversed progressive dermal ischemia in the zone of tissue injury and produced what was considered earlier healing in comparison to the control group.[17] In contrast to these studies, Harms et al., using an ovine model of a 30% burn injury, identified postburn increases in lymph flow from burned tissue, unburned tissue, and the lung that occurred in temporal association with an increase in the lymph content of prostacyclin metabolites but no increase in thromboxane concentration in the lymph from any of those tissues.[18] To the contrary, Herndon et al., in clinical studies, found markedly elevated plasma levels of thromboxane B_2 during the first 3 days following burn injury and secondary elevations thereof during subsequent septic episodes. Prostacyclin metabolite levels were not elevated at any time in these patients.[8] Alexander et al., using a murine model of scald injury, found that various inhibitors of the arachidonic acid system including a cyclooxygenase inhibitor, a leukotriene receptor antagonist, an inhibitor of thromboxane synthetase, and a calcium-channel inhibitor all significantly reduced extravasation of Evans blue dye.[5] Wound water content was significantly reduced only in the animals treated with the leukotriene receptor antagonist and the inhibitor of thromboxane synthetase. The antiedemic effect was evident with either pre- or postburn treatment with those two agents.

The area of immediate cell death resulting from burn injury has been termed the zone of coagulation. The vascular structures in this zone are irreparably damaged and thrombosed. Surrounding this area of cell death is the zone of stasis in which the blood flow is initially attenuated and then usually returns to normal following injury (Fig. 24–1). Progressive endothelial injury has been noted by Ham and Hurley over 24 hr in this area.[19] Failure to restore tissue perfusion by adequate resuscitation may result in further decreases in blood flow in the zone of stasis, eventually yielding devitalized tissue similar to the zone of coagulation.

Circumferential full-thickness burns of an extremity may compromise blood flow to underlying or distal unburned tissue. As edema formation progresses beneath the inelastic eschar, tissue pressure may in-

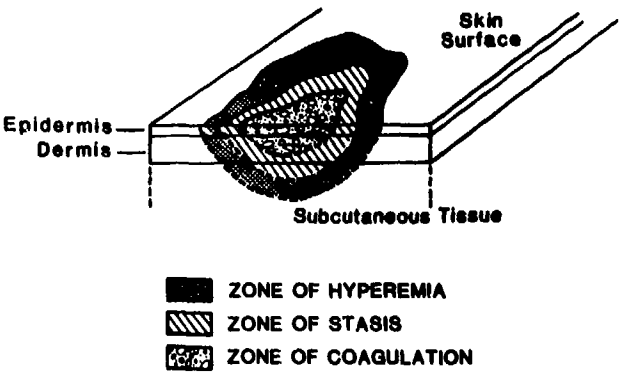

Fig. 24–1. The tissue injury typically seen following thermal trauma is concentric in nature. The zone of coagulation or the area in which immediate cell death occurs following burn involves the entire epidermis and dermis in full-thickness burn injury. In partial-thickness burn injury, the zone of coagulation involves only part of the epidermis and dermis, as pictured here. The surrounding zone of stasis may convert to a zone of coagulation yielding full-thickness injury if tissue perfusion is not restored.

crease until it exceeds venous and capillary pressure. The increase in tissue pressure will restrict blood flow to this area, and if sufficient pressure is transmitted to the underlying muscle compartments, the blood supply to distal unburned tissues may be impaired. Edema formation may be exaggerated by administration of excessive resuscitation fluid and by dependency of the burned part. Edema formation should be minimized by judicious resuscitation, constant elevation of burned limbs, and active exercise of the extremities for 5 min every hour. Although the value of escharotomy in relieving compression and thus restoring blood flow is well established, the clinical indications for escharotomies are uncertain. Pallor and coolness to the touch of unburned skin are common in extensively burned patients and hence of little value in assessing the need for escharotomy. The signs and symptoms of elevated compartmental pressures, such as cyanosis, delayed capillary refilling, paresthesias, abnormal pain, loss of sensation, and decreased motor function are difficult to evaluate in the badly burned limb.

Doppler ultrasonic flow meter examination to detect pulses in the palmar arch of the upper extremity and posterior tibial artery in the lower extremity is the most widely used method for determining when escharotomy is needed. An absence of pulsatile flow and progressive diminution of the auditory signal on serial examination are indications for escharotomy.[20] In an effort to identify limbs requiring escharotomy at an earlier stage in the process, Saffle et al. advocated the use of compartmental pressure measurements.[21] An absolute compartmental pressure of 30 mm Hg was proposed by this group as the critical level above which escharotomy should be performed. Because this pressure coincides with capillary pressure, a higher pressure causes cessation of flow through the capillaries. These investigators contended that use of compartmental pressure measurement allowed earlier identification of limbs requiring escharotomy than the conventional Doppler ultrasonic flow meter and thus decreased the period of time during which underlying tissue was subjected to ischemia. Direct measurement of an elevated compartmental pressure following escharotomy also enables one to identify those patients in whom fasciotomy is required.

Escharotomy can be performed as a ward procedure using either a scalpel or electrocautery. Anesthesia is not necessary because the incision is made in the area of insensate full-thickness burn. The escharotomy incision is made in the midlateral or midmedial line in the involved limb or digit, and it should extend from the distal to the proximal margin

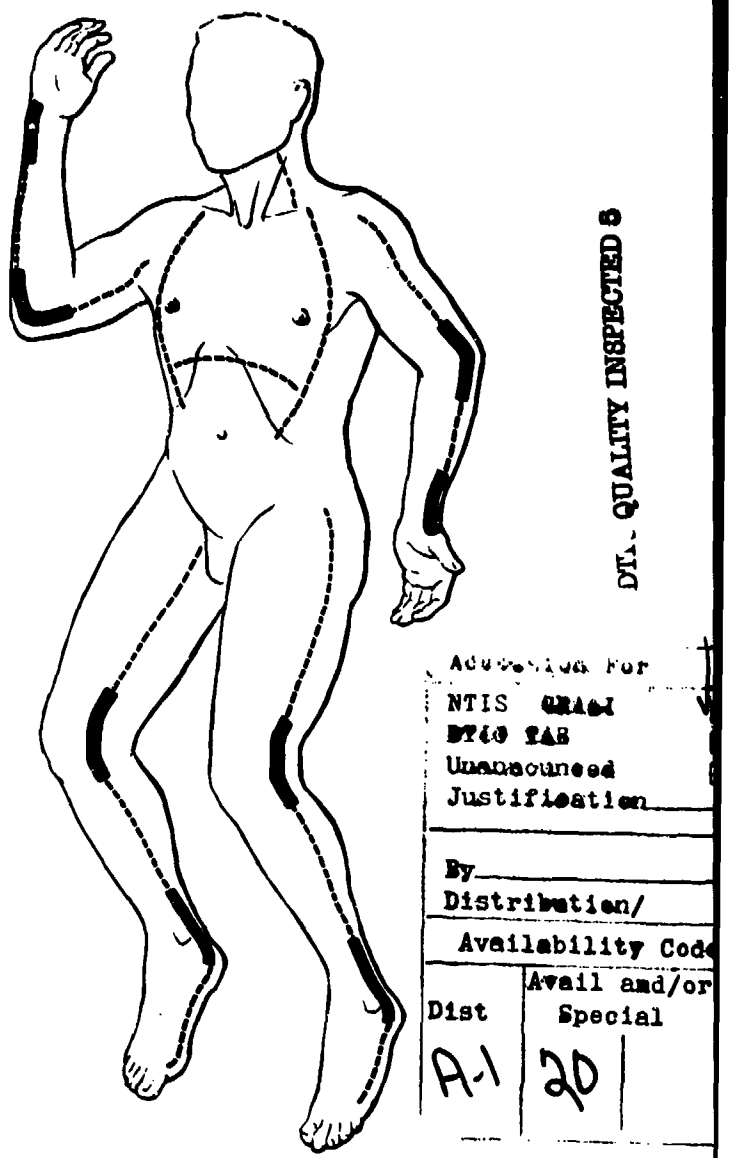

Fig. 24–2. The preferred sites for escharotomy are depicted in this diagram. If the burn extends across a joint, the incision should be carried across the joint lines, as emphasized here by the thickened lines.

of the encircling full-thickness burn (Fig. 24–2). The excision should extend only through the eschar and the immediately underlying superficial fascia (Fig. 24–3). Deeper incisions only increase bleeding and expose more subcutaneous tissue. Exposed unburned subcutaneous tissue rapidly becomes colonized by bacteria, the proliferation of which should be controlled by generous application of topical antimicrobial agents to the wounds.

Escharotomy commonly suffices to restore the circulation in a limb with cutaneous burns, but fasciotomy may be required to restore limb circulation in patients with: (1) high-voltage electrical injury; (2)

Fig. 24–3. The circulatory embarrassment caused by circumferential full-thickness burns in both lower extremities has been relieved in this patient by escharotomy incisions placed over the midmedial aspect of each leg. Note that the excision has been extended only through the eschar and the underlying superficial fascia exposing subcutaneous fat. In an effort to delay proliferation of bacteria in the exposed subcutaneous tissue, a topical antimicrobial agent has been generously applied to the left lower extremity.

associated skeletal trauma; (3) associated soft tissue damage from crush injuries; (4) reperfusion injury following vascular trauma; and (5) burns involving the muscles themselves, i.e., injuries that cause edema formation beneath the investing fascia. When the anterior compartment of the lower limb is involved, clinical manifestations include loss of sensation of the skin on the first pedal web space and loss of dorsiflexion of the foot. Fasciotomies should be performed in the operating room using general anesthesia. In patients with full-thickness burns of both surfaces of the hand, dorsal fasciotomies of the interosseous muscle compartments have been recommended to prevent the occurrence of ischemic necrosis of these intrinsic muscle groups, but the effectiveness of such procedures remains unconfirmed.

The net effect of burn injury on vascular permeability in specific tissues remote from the wound remains undefined. Montero et al., in an ovine model, have demonstrated a fourfold increase in lymph flow and a twofold increase in the lymph: plasma protein ratio from an unburned extremity following thermal injury; these findings suggest an alteration in vascular permeability.[22] Data from our institution have suggested that burn injury alone does not cause a generalized systemic increase in vascular permeability.[23] Carvajal et al., in a scald burn model, demonstrated maximal burn wound edema at 3 hr following injury but documented neither albumin extravasation nor water accumulation in the unburned tissues.[24]

Studies by Goodwin et al., Herndon et al., and Tranbaugh et al. have indicated that lung water is little changed during the first 24 hr afterburn injury, even when the patient is resuscitated with large volumes of crystalloid solutions.[25–27] Goodwin used a soluble gas rebreathing technique, whereas Herndon and Tranbaugh used a thermal-green dye double indicator dilution technique. Both techniques have been documented to underestimate the actual amount of extravascular lung water, especially in the presence of lung perfusion abnormalities.[28,29] In contrast, Sharar et al., using a goat model, found a 30% increase in gravimetrically measured extravascular lung water with a threefold increase in lung-lymph flow.[30] The gravimetric lung weights were corrected for increased pulmonary blood volume. Immediately following thermal injury, pulmonary vascular resistance has been noted to be increased disproportionately with respect to systemic vascular resistance, and that increase persists after the systemic vascular resistance has returned to normal. The immediate elevation of pulmonary vascular resistance is thought to protect the lung from pulmonary edema during the resuscitation phase because of the precapillary nature of this alteration.[31]

Several humoral mediators have been implicated in the microvascular changes of remote tissue following burn injury. Luterman et al. related acute postburn pulmonary dysfunction to the microvascular effects of elevated circulating levels of fragment D produced by the coagulation system in response to severe burn injury.[32] Gelfand et al., using a mouse model of scald injury, identified massive postburn activation of the alternate complement pathway associated with an increase in neutrophil aggregation within the lungs, increased pulmonary vascular permeability, and increased lung edema.[33,34] Those changes were significantly reduced in mice with a genetic C5 deficiency and by preburn cobra venum factor decomplementation. Local and remote edema

formation has been attributed to activated neutrophils and their metabolic products such as lysosomal enzymes and excited oxygen radicals. The importance of neutrophils in local microvascular changes has been questioned by studies in which neutropenia induced prior to burn injury did not significantly alter Evans blue dye extravasation or wound water content. The limited effectiveness of individual agents used to prevent, attenuate, or counteract permeability changes is probably explained by the predominance of pressure-related alterations in capillary filtration in the early postinjury period. The many circulating substances that can affect vascular permeability also make it unlikely that any one inhibitor or antagonist is totally effective in preventing edema formation following burn injury.[10]

Alterations in microvascular blood flow occur in other organs following thermal injury of which the gastrointestinal tract is typical. Asch and associates, using radiolabeled microspheres in a canine model of burn injury, showed that total gastric blood flow was little affected in the first 5 hours afterburn injury.[35] However, more recent studies have identified significant shock-related diminution of gastric mucosal blood flow.[36] This leads to histologically identifiable ischemic changes of the upper gastrointestinal tract that are focal in nature and result in mucosal cell injury. These small erosions in the stomach and duodenum may progress to frank ulceration of the upper gastrointestinal tract if not protected from further acid injury by the administration of antacids or H_2 receptor antagonists.

VASCULAR INJURIES FROM ELECTRICAL TRAUMA

Vascular injury may be caused by the direct effect of high-voltage electricity or by impairment of capillary flow as a result of elevated pressure in muscle compartments due to postinjury edema. Vascular injury normally leads to thrombosis of the involved vessels, with secondary damage to tissue supplied by these vessels. Arteriography may be helpful in determining when significant deep tissue damage is present even when large vessel pulses are intact. Arteriographic evidence of small vessel occlusion may be useful in determining the need for an operation and the extent of the procedure required (Figs. 24–4 and 24–5). Small vessel occlusion may be due to elevated compartmental pressure caused by edema of injured muscles within that closed space. Immediate decompression relieves the pressure and prevents progression of tissue ischemia within a burned extremity. The absence or progressive diminution of distal pulses, the loss of sensation of

Fig. 24–4. Extensive charring in obvious full-thickness injury is evident on the lateral aspect of this patient's foot, which had made contact with a high-tension line. Arteriography or radiolabeled technetium pyrophosphate scanning may be of use in evaluating the extent of tissue injury in this extremity.

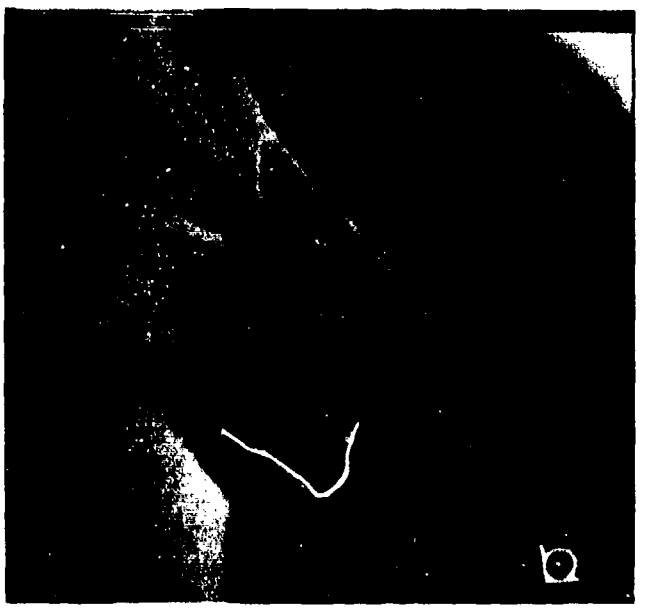

Fig. 24–5. The lack of arborization of nutrient vessels is typical of damage secondary to electrical injury. In this arteriogram, the proximal profunda brachii artery demonstrates filling of nutrient vessels, whereas the distal vessel shows a lack of such branches. Also absent is filling of the superior and inferior ulnar collateral arteries. Evidence of small vessel occlusion indicating nonviable tissue can be helpful in planning the required operative procedure.

distal unburned skin, a decrease in motor function of the involved limb, and a compartment pressure of more than 30 mm Hg are all indications for immediate decompression. Radiolabeled technetium pyrophosphate, used as described by Hunt et al., may be helpful in determining the extent of muscle injury.[37] "Cold" or nonperfused areas on the scan of a limb correspond to tissues that have been irreversibly necrosed and are nonviable. Tissue with normal uptake can be reliably presumed to be well perfused and undamaged. "Hot" areas of increased uptake on the scan correspond to muscles in which 20 to 80% of fibers are necrotic.

At surgery, obviously nonviable tissue should be debrided. Because heat dissipates from deeper tissues more slowly following cessation of electric current flow, periosseous tissues may be more badly damaged than more superficial tissues. Consequently, periosseous tissues underlying viable superficial muscles should be examined to ensure adequate debridement.

Proper wound care after fasciotomy is important in an effort to preserve exposed tissue and to conserve limb length and function. The wound should be loosely packed with gauze sponges soaked with mafenide acetate solution to prevent desiccation of exposed muscles, tendons, and nerves. Alternatively, a biologic dressing such as cadaver allograft or porcine xenograft can be used for coverage of exposed tissues. Adjacent cutaneous burn should be treated with a topical antimicrobial agent. The patient should be returned to the operating room 24 to 48 hr following the initial procedure for repeat exploration and debridement if necessary. The wound should be closed only when the viability of the remaining tissue is certain. Early flap coverage of exposed vital structures has been reported to be useful in selected patients.

Delayed hemorrhage from moderate-to-large blood vessels may occur after high-voltage electric injury. Although such hemorrhage is reported to be caused by arteritis secondary to the injury, inadequate debridement and vascular necrosis due to exposure-related desiccation are more likely causes.

IATROGENIC VASCULAR INJURY IN BURN PATIENTS

Iatrogenic catheter-related vascular injury may be divided into mechanical and septic complications. The necessity for long-term venous access in a severely burned patient will normally require placement of catheters into central veins. Reported mechanical complication rates of central venous access range from 0.4 to 22%.[38–41] Internal jugular and subclavian lines have the highest complication rates, with the majority of complications occurring at the time of insertion. Recognized complications include hematoma formation, carotid or subclavian artery laceration, traumatic arteriovenous fistula, pleural injury resulting in either pneumo- or hemothorax, air embolism, and neurologic deficits due to injury of either the phrenic nerve or brachial plexus. Femoral venous lines have a lower mechanical complication rate, with a 1.3% incidence of major hematomas that compares favorably to a 1.6% rate for peripheral venous access.[42] Strict adherence to proper technique should keep the rate of mechanical complications secondary to intravascular cannula insertion under 5%.

Quadruple-lumen pulmonary artery catheters are used frequently in critical care settings to monitor cardiac output and right and left heart filling pressures. The most common complications associated with their use are related to insertion techniques used to gain central venous access. Additionally, pulmonary infarcts secondary to improper positioning, pulmonary hematomas due to overzealous balloon inflation and subsequent pulmonary artery rupture, and air emboli caused by injection of air into a ruptured balloon all may occur. Knotting of a catheter around itself is most likely to occur when loops form in the right ventricle and the catheter is repeatedly withdrawn and advanced. Knotted catheters can usually be removed transvenously, but occasionally extensive surgical procedures are necessary. Mechanical damage to the tricuspid or pulmonary valves has been reported as a consequence of withdrawing inflated balloons through valves, prolonged catheterization, and preexisting myxomatous degeneration. Right-sided endocarditis secondary to pulmonary artery catheterization has been reported but is rare.

Nonseptic complications related to placement of arterial lines for hemodynamic monitoring or prolonged arterial access are normally related to thrombosis of the catheterized vessel. The incidence of vascular thrombosis is dependent upon the patient's hemodynamic stability, intrinsic vascular disease, and the relative sizes of vessel and cannula, as well as the duration of catheterization, the catheter material, and whether the catheter is intermittently or continuously flushed.

The complication rate associated with repeated percutaneous arterial punctures is normally greater than that associated with prolonged arterial catheterization. This is especially true in young children in whom repeated punctures may result in hematoma formation that may impede blood flow. Approximately 5 to 8% of all arterial catheterizations

result in thrombosis of the vessel, but only 1% are symptomatic.[43–45] Use of the radial or dorsalis pedis artery is preferred because both the hand and the foot have a dual circulation. Prior to radial artery puncture, an Allen test should be performed to confirm good collateral flow through the ulnar artery. Femoral arterial catheters may be safely used if the cannulation site is placed below the inguinal ligament. Puncture of the vessel above the ligament may result in significant retroperitoneal hemorrhage that is not easily diagnosed. Femoral cannulation should be avoided in children and adults with significant peripheral vascular disease because thrombosis may occur in a significant percentage of these patients. The brachial artery should only be used when other sites are unavailable because embolic occlusion of either the radial or the ulnar artery occurs in 10 to 40% of cases when this vessel is used for long-term access.[46] After placement of any arterial catheter, distal perfusion should be checked at least every 8 hr. Signs of poor perfusion, difficult blood withdrawal, or prolonged dampening of the pulse tracing will necessitate catheter removal.

Catheter size is clearly related to the incidence of thrombotic and embolic complications. Arterial catheters should not be larger than 20 gauge in size. The use of an 18-gauge catheter in a radial artery will result in a greater-than-50% thrombosis rate between 20 and 40 hr following placement.[47] The thrombosis rate drops to less than 11% when a 20-gauge catheter is used. For neonates, a 22- to 24-gauge catheter should be used, with 22-gauge catheters used preferably for children between 1 month and 3 years of age. A percutaneous route should be used for placement whenever possible because access gained by arterial cutdown has a greater risk of thrombotic complications. Ischemic necrosis of the skin overlying a radial artery cannulation site occurs in approximately 3% of patients and is secondary to thrombosis of the vessel at the point of puncture. Retrograde embolus resulting in a cerebral vascular accident may occur when a radial vessel thrombosis occurs following use of the vessel for long-term access. The incidence of retrograde embolus is related to the method in which the patency of the catheter is maintained. Continuously flushed catheters have the lowest thrombosis rate. Intermittent flushing of the catheter results in higher thrombosis rates. If the volume of fluid used to flush the catheter is greater than 5 ml, the risk of retrograde embolism is significantly higher.

Septic thrombophlebitis from long-term venous access remains a common complication in burn patients. Injury to the vein wall, catheter composition, and microbial seeding resulting from contamination of the infusion sets or bacteremia secondary to wound manipulation all play a significant part in the pathogenesis of suppurative thrombophlebitis. In an attempt to prevent suppurative thrombophlebitis, the duration of cannulation at any given site should be limited to 72 hr. Strict enforcement of such a regimen resulted in a decrease in the incidence of suppurative thrombophlebitis from 6.9 to 1.4% in a large series of burn patients[48] (Table 24–1).

Suppurative thrombophlebitis should be considered as a diagnosis when no other source of sepsis or infection can be found in a burn patient with positive blood cultures, hematogenous pneumonia, or acute bacterial endocarditis. Local signs of infection are present in fewer than half the patients, and thus every previously cannulated vein must be evaluated and the site of the cannula tip residence examined. Brisk flow of unaltered blood from the proximal end of a vein excludes it from further consideration. If intraluminal pus is identified, the diagnosis of suppurative thrombophlebitis is confirmed. If clot is present without suppuration, a section of the vein and clot should be cultured and studied histologically. The presence of microorganisms in the clot or vein wall supports the diagnosis of intraluminal infection.

The treatment of suppurative thrombophlebitis includes systemic administration of antibiotics to

Table 24–1. Changing Incidence and Location of Suppurative Thrombophlebitis, 1969 to 1978

		Patients with Suppurative Thrombophlebitis		Patients with Central Vein Disease	
Time Period	Burn Patients Admitted	Number	Percentage of Admissions (%)	Number	Percentage of Patients with Disease (%)
1969–1970	626	43	6.9	9	21
1971–1972	568	29	5.1	9	31
1973–1974	505	18	3.6	3	17
1975–1976	502	20	4.0	7	33
1977–1978	506	7	1.4	4	57

Fig. 24–6. Extensive mural thickening and inflammatory changes as evident in this cephalic vein are characteristic of suppurative thrombophlebitis. The venous puncture site is obvious at the level of the forceps. Venectomy should be extended proximally and distally until a grossly normal vein with brisk back bleeding is encountered.

which the infecting organism is sensitive and the excision of the infected vein. Proximal excision should continue to the point where the vein becomes a tributary of the next larger order of veins or to that level where the vein wall shows no mural thickening and brisk retrograde flow of blood is noted (Fig. 24–6). Venectomy wounds should be loosely packed and either grafted or secondarily closed at a later date when the local infection has resolved. Failure of venectomy to result in eradication of the septic episode may indicate inadequate excision or the presence of suppuration within another vein.

The recommendation to limit cannulation time for venous access to 72 hr in thermally injured patients is supported by the documented finding of bacteremia in approximately 20% of burn wound debridement procedures, with the incidence of positive blood cultures directly related to the extent of the burn wound manipulation.[49] Such recurrent bacteremias are associated with a time-related increase in the risk of microbial seeding of the fibrin sleeve that forms around the cannula. In addition, patients with extensive thermal injury have wounds that are heavily colonized by the endemic flora of the burn unit. Because long-term access is required in such patients and the cannulation site must be changed every 3 days, it is often necessary to perform venipuncture through those colonized wounds, thus increasing the risk of cannula contamination. This situation is distinctly different from the average patient in an intensive care unit in whom bacteremic episodes and the existence of heavily colonized wounds are less common.

SUPERIOR MESENTERIC ARTERY SYNDROME

The superior mesenteric artery syndrome or Wilkie's syndrome was first described by Rokitansky in 1861.[50] The classic syndrome is due to an obstruction of the third portion of the duodenum by the root of the small bowel mesentery at the level of the superior mesenteric artery where the duodenum crosses the spine. Normally, the angle between the superior mesenteric artery and the aorta is between 38 and 50°. With loss of mesenteric and retroperitoneal fat, this angle may be decreased to less than 20°, and the weight of the mesenteric tissues compress the duodenum when the patient is in a supine position. Thermally injured patients who experience weight loss are at particular risk for developing this syndrome. The syndrome has been described in various other groups of patients, including patients with cancer, severe head trauma, and dietary disorders, and orthopedic patients who have been placed in whole body casts, all of whom have significant weight loss in common. It is usually a disease of young adults, with females predominant over males at a ratio of 1.5 to 1.

Diagnosis of the superior mesenteric artery syndrome is best made by fluoroscopic examination of the upper gastrointestinal tract in patients who have postprandial vomiting associated with progressive weight loss. Fluoroscopic findings of obstruction of the third portion of the duodenum, dilation of the proximal duodenum, and apparent retrograde peristalsis confirm the diagnosis (Fig. 24–7). The treatment of patients with a short duration of symptoms should be nonoperative. Nasogastric decompression of the stomach and alimentation provided either by the parenteral route or enterally via a small bowel feeding tube placed past the point of obstruction are the mainstays of treatment. After stabilization of weight loss and with roentgenographic evidence that the obstruction has resolved, multiple small feedings may be begun, with the patient placed in the left lateral decubitus position following each meal.

Operative treatment may be necessary for patients with longstanding weight loss and persistent symptoms of duodenal obstruction. Duodenojejunostomy has been the most commonly performed procedure. It has an 80 to 90% success rate in alleviation of symptoms.[51] Gastrojejunostomy has been used in a few series, but because of its ulcerogenic potential, vagotomy must also be performed. Strong's operation, in which the right colon is mobilized, the ligament of Treitz is lysed, and the duodenum is mobilized, has gained increasing popularity. Its success

Fig. 24–7. The diagnosis of superior mesenteric artery syndrome was confirmed in this patient by this barium study, which shows complete obstruction of the third portion of the duodenum. Note the catheter in the superior mesenteric artery that implicates this vessel as the cause of the obstruction.

rate rivals that of duodenojejunostomy, and no gastrointestinal anastomosis is required. Progressive nonsurgical management utilizing gastric decompression and vigorous nutrition has reduced the need for operative intervention from 42 to 11% in a current series.[52,53] This syndrome has become increasingly less common as better understanding of postburn metabolic requirements has led to improved nutritional support of the thermally injured patient.

DISCLAIMER

The opinions or assertions contained herein are our private views and are not to be construed as official or as reflecting the views of the United States Department of the Army or the Department of Defense.

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