Complications of vascular conditions in the upper limb are rarely life threatening but can frequently be limb threatening. The vascular system is the lifeline through which travels the necessary ingredients to preserve the tissues of the upper limb. The system of arteries and veins in the proximal limb is very simple, because it is a one vessel system; but in the distal limb, it becomes more complex with a dual supply in most areas. Because of this dual supply, a significant interference with one leg of the dual supply is not tantamount to losing total blood supply to the involved digit or portion of the hand.

In looking at the natural course of vascular conditions of the hand, be they traumatic, occlusive, vasospastic, or compressive, the most frequent complication is ischemia of varying degrees. Ischemia can be manifested by an increased need for circulation, such as a very cold exposure or by increased demands of the muscles during extreme exercise. This is very mild ischemia and during the resting state is not manifested at all. As ischemia progresses to more total ischemia, one can experience pain, paresthesia, and absence of pulses, indicating a significant deprivation to the tissues of their blood supply. Ultimately, more profound ischemia can lead to tissue loss with gangrene. Many times in the natural course of vascular diseases, such as gradually occlusive or vasospastic diseases, one sees the ischemia syndrome progress from very mild ischemia to marked ischemia with tissue loss. However, in the traumatic and sudden occlusive or embolic categories, the ischemia is frequently total and sudden, and, if not diagnosed and treated appropriately, it can lead to massive tissue loss.

One of the other complications of vascular conditions and their treatment is bleeding. Bleeding can be the result of suture line leak; unrepaird vessels; coagulation deficiencies, either natural or induced by drugs; or unrecognized lacerated vessel branches which are masked because of hypotension and become manifest when the blood pressure returns to normal. On the other side, thrombosis is a standard complication of all vascular conditions and can be due to unrecognized vessel trauma with intimal damage; inadequate surgical technique; or hypercoagulable state, either natural or drug induced; or it may be accidentally produced by interruption of the normal arterial and venous ingress and egress of blood through the tissues.

Most of these complications are avoidable, but they must be recognized when they occur and dealt with appropriately. The scope of this article is to determine what complications arise in various situations and the appropriate treatment if they do arise.

When one thinks of the macroscopic circulation system, one should envision the heart as the central pumping system which sends out blood to various sites throughout the body through a system of channels called arteries. The pressure throughout the system should essentially be constant as it is the single most important ingredient of the fluid mechanics and is responsible for the constant delivery of the blood to the various sites that have continually changing demands. It is these changing demands that cause the shunting of blood from one site to another. The whole system is under a complex control mediated through the autonomic nervous system, which changes the di-
ameter of the various channels to supply the flow necessary to the various demand sites. The whole system is under the control of the basic laws of dynamics. If the diameter changes and the pressure remains constant, then the rate of flow is increased through the more narrow site. If the peripheral resistance increases, then the rate of flow is significantly less, and if the peripheral resistance equals the head of pressure, there is no effective flow. A basic understanding of these fluid dynamics is necessary to avoid and treat most of the complications that arise in vascular surgery.

Three major areas will be approached: first, the complications of incorrect diagnosis; second, the complications of surgery and surgical judgment; and third, the complications of postsurgical management.

**COMPLICATIONS OF DIAGNOSIS**

Choosing four separate categories of vascular injuries and disease—traumatic lesions, occlusive lesions, compression syndrome, and vasospastic disorders—we can now examine the complications produced by incorrect diagnosis in each one of these categories.

In dealing with the patient with acute trauma, many of the usual diagnostic maneuvers cannot be performed, but the examination should be as thorough as possible. It should include a determination of pulse distal to the area of trauma by clinical palpation and the use of ancillary devices such as the Doppler ultrasonic flow detector and pulse volume recordings. Visualization of the vascular tree by radionuclide angiogram, contrast angiogram, or digital subtraction angiogram will yield useful information. The patient with blunt trauma should also have evaluation of the compartmental pressure by clinical examination including digital flexion and extension and palpation of the area injured, plus compartmental pressure recordings by one of the available techniques if there is any doubt in the examiner’s mind. The areas of complication in dealing with the incorrect diagnosis of a patient with acute trauma are commonly errors of omission in that a vascular lesion is not suspected but does exist. Examples of such are in the blunt trauma with compartmental pressure, in which the diagnosis is missed, leading to muscle necrosis or even Volkmann’s ischemic contracture. This is easily avoided by recognition of the increased pressure within the closed fascial compartment, usually on the flexor surface, and appropriate fasciotomy.

Fasciotomy should also be considered in the intrinsic compartments of the hand when dealing with blunt trauma to the hand. Occasionally in blunt trauma, there can be a direct arterial injury with division of the main artery and extensive bleeding into the compartment that can only be determined by increased compartmental pressure readings and subsequent operation or contrast angiography. In the forearm and hand, direct visualization of the vessel at surgery is not difficult, and the need for contrast angiography in these patients is not essential. The main point is that suspicion and ultimate recognition of these lesions lead to the appropriate diagnosis and avoidance of the complication.

When faced with the patient having an open injury and profuse bleeding, the nonrecognition of the vascular injury will lead to complications such as ischemia or, more commonly, a false aneurysm of the injured vessel. The main reason that these lesions are missed is that the distal circulation is deemed adequate by the examining physician because there is adequate capillary refill and the hand appears viable; therefore, no attempt is made to explore the area of injury in order to determine if the bleeding is coming from a vessel. If there is profuse bleeding, the area must be examined by radionuclide angiography, contrast angiography, digital subtraction angiography, or direct surgery to determine if the radial or ulnar artery has been damaged. A partial laceration of such an artery will lead to profuse bleeding and the ultimate formation of a false aneurysm, which is a complication of laceration and will necessarily have to be surgically approached at a later time. Therefore, any patient with acute trauma and bleeding more substantial than one would expect from the local environment must be suspected of having an arterial injury and approached as such, either directly or with the various diagnostic tests.

Another complication which can arise in the area of acute trauma is the incorrect diagnosis of the level of arterial injury. If there is a significant traction lesion, in addition to the lacerating lesion, the level of arterial injury can in fact be much more proximal than the open injury. We have seen injuries at the wrist level with arterial injuries at the elbow or above. Unrecognized, this will lead to a significant complication of distal ischemia and tissue loss due to gangrene. If at the time of open exploration the proximal flow is not adequate, then the artery must be traced back to the point where the flow is adequate. In a traction lesion there could be skip areas of intimal damage.
producing proximal spasm and thrombosis that will go unrecognized by the unsuspecting operator. Therefore, when performing an operation on a patient with acute trauma and arterial injury, the tourniquet must be released and adequate proximal flow must be obtained before performing a vascular anastomosis.

In dealing with a patient who has an occlusive lesion, the most common incorrect diagnosis is a proximal source of the occlusive lesion. This is particularly true in a patient with an embolus. Any patient with a distal embolus must be examined more proximally to determine the source. There can be an occlusion with embolization from the subclavian artery due to the cervical rib or a proximal aneurysm. Axillary artery aneurysms should be suspected especially in crutch users as we have seen seven such lesions with distal embolization. In operating on these patients, one must approach the proximal lesion as well as perform the distal embolectomy. When performing a diagnostic evaluation on a patient with an occlusive lesion, visualization of the entire vascular tree in the upper extremity is usually necessary. Certainly pressure studies and pulse volume recordings should be done of the entire upper arm to insure proximal flow, and visualization of the proximal tree by digital subtraction angiography is extremely useful. The other common incorrect diagnosis in a patient with occlusive disease is a systemic problem such as clotting disorders or coagulopathies due to medications like ergot; ingestion of heavy metals or agents such as polyvinyl chloride; sickle cell disease and crisis or malignancy.

The third category of vascular diseases involves the compression syndrome. Incorrect diagnosis here usually leads to inappropriate surgical approaches. Because of the labile nature of the vascular tree some patients are thought to have shoulder girdle compression and are operated on for this when, in fact, they have segmental or regional spasticity of the vessel. First rib resection for shoulder girdle or thoracic outlet compression has led to significant complications, including both nerve and arterial damage. In a recent report, Dale\textsuperscript{a} reviewed his personal series of 76 patients treated with surgery, of whom 64 had transaxillary first rib resection. The results of 55 per cent were excellent, 35 per cent good, and 9 per cent failures. What was more important in this presentation is that Dale included results of a national inquiry made of vascular and thoracic surgeons performing the transaxillary first rib resection. Complete postoperative paralysis due to brachial plexus dysfunction was reported 102 times. Twenty-two of these patients did not recover any function. We have personally seen seven patients with major brachial plexus dysfunction following transaxillary first rib resection. An additional seven instances of partial neurologic deficit with 30 instances of failure of complete recovery were reported. Patients having first rib resection should be warned of the potential complications of brachial plexus neuritis, neuralgia, or even paralysis. Reviewing the operative approach, the significant traction and abduction that are necessary for the exposure through the transaxillary approach may lead to these lesions; therefore, we recommend careful positioning of the arm if this approach is used and a discontinuance of the abduction and traction every 20 minutes during the operative procedure. This syndrome can frequently be diagnosed incorrectly, and if one is contemplating surgery, corroborative evidence, including objective pulse volume measurements during the positional maneuvers or angiographic evidence of arterial occlusion during the positional maneuvers, should be obtained.

The last area of consideration is the vasospastic disorders. Incorrect diagnosis of these disorders can lead to unnecessary surgery plus the complication of the aggressive surgical debridement. The majority of people with vasospastic disorders do not need any surgery at all. Most can be managed with medication and local care. The diagnostic evaluation of these patients must be thorough and must include baseline vascular studies of some type. We currently use pulse volume recordings followed by cold stress and vascular recording. This will then give the managing physician an objective measurement of the circulatory picture following cold stress. The avoidance of tobacco is important in managing these patients; and during the diagnostic evaluation, the patient should be shown how the circulation is disturbed by smoking.

When evaluating a patient with a vasospastic disorder, one must first be sure that the spasm is primary and not secondary to a thromboembolic phenomenon or nerve lesion. The sympathetic nerves travel in the peripheral nerves, and a compressive nerve lesion can produce a vasospastic disorder as a part of its presenting symptoms. Therefore, the peripheral nervous system should be thoroughly evaluated and studied with electrodiagnostic studies if there is any question of a proximal compressive nerve lesion. A patient with occlusive arterial disease, including peripheral arteriosclerosis, arterial embolism, arterial thrombosis, and thoracic outlet syndrome, sometimes shows peripheral vasospastic symptoms. Asymmetry of the symp-
COMPLICATIONS OF SURGERY

The main complications of surgery are thrombosis, bleeding, and infection. Having just reviewed the complications of incorrect diagnosis, we will assume that the appropriate and complete diagnosis has been made before or during the surgical exercise for the purpose of this part of the discussion. Thrombosis is most commonly due to inadequate vascular anastomosis. One cannot overemphasize that there are no ancillary modalities to prevent thrombosis in an inadequate anastomosis or an anastomosis at the site of a damaged vessel. The adjuncts of anticoagulation—dextran, aspirin, sympathetic blockade—only serve to protect the perfect anastomosis from subsequent thrombosis. If thrombosis is detected, early thrombectomy or resection and reanastomosis can be beneficial. If thrombosis is undetected, however, there is no successful method of treatment.

The first sign of thrombosis is a sudden color change. If a previously pink hand becomes white or ashen, there has been a change in arterial flow. The absence of capillary refill after blanching confirms this color change, heralding arterial thrombosis. If, however, the distal part appears cyanotic and becomes tense to the feel with very rapid capillary refill after blanching, these signs indicate venous thrombosis. The fingertips can be punctured with an 18 gauge needle and if dark blood appears followed by bright red blood, one can then make a diagnosis of venous thrombosis with patent arterial inflow. If the diagnosis of postoperative venous or arterial thrombosis is made, immediate surgical correction is the only approach. Delaying surgery for hours only complicates the situation and may cause extension of the thrombosis from the venous side to the arterial side and vice versa. Local or proximal spasm can predispose the patient to thrombosis. The patient must be kept pain free and content, for it is apparent that emotion plays a tremendous role in the blood flow patterns of an extremity, especially after surgery. Pain can cause intense vasospasm, as can worry and anxiety. Therefore, if the patient can remain free of pain in a worry free environment, there will be less risk of vascular spasm and thrombosis. Smoking is prohibited during the postoperative period of a patient with vascular reconstruction. Many studies have shown that smoking can reduce the blood flow to a digit by approximately 20 per cent. I think that patients should refrain from smoking for at least two weeks postoperatively until the intima has re-endothelialized.

Bleeding is the second major surgical complication. The best way to avoid postoperative bleeding is to insure that the vascular anastomosis is complete at the time of surgery. The anastomosis should be inspected and any leaks closed. Wrapping an anastomosis with a piece of vein frequently can aid in the reduction of postoperative bleeding. If there is a persistent leak from a vascular anastomosis, a false aneurysm will form and then must be surgically removed at a later date. If anticoagulation medication is used, the patient must be monitored closely because adverse systemic effects can be dangerous and extensive bleeding can ensue. Most patients do not need systemic anticoagulation after vascular reconstruction. Local anticoagulation with heparin, or even one of the thrombolytic agents, at the time of operation can be useful. Another cause of postoperative bleeding is bleeding in the area distal to the revascularization. A careful check for bleeding sites should be made after revascularization. These sites will only become evident after the blood flow has stabilized. Especially in an area of trauma, muscle bleeders or small arteries can present postoperative hemorrhage problems if not coagulated or tied off. Therefore, after doing a revascularization procedure of any type, a careful check of the subcutaneous and soft tissue region distal to the area of revascularization must be made prior to closing the wound in order to prevent this postoperative complication.

The third major complication following surgery is infection. The area surrounding a vascular anastomosis, where there is invariably some hematoma, is especially susceptible to infection by blood-borne bacteria from any infection elsewhere in the patient’s body. Systemic antibiotics are therefore used in all patients undergoing vascular reconstruction particularly if an operation takes more than 2 hours. Bacterioidal, rather than bacteriostatic, drugs are recommended. The antibiotics should be given intravenously at the time of surgery and for approximately 72 hours following surgery. If there is drainage from the wound, the wound should be immediately opened. The late complication of infection is blowout of a vascular anastomosis. This is particularly dangerous because it often occurs in unprotected situations,
Complications of Vascular Conditions in the Hand

POSTSURGICAL MANAGEMENT

Many of the complications in the immediate postoperative period have been mentioned in the previous section; however, the major complications in the postoperative management are spasm, drug reaction and interactions, and swelling and edema from reperfusion. Spasm following the operative event can be a serious complication and can lead to eventual thrombosis and/or tissue loss. If the patient has a viable vascular system, spasm may play a role generally and peripherally, especially in those patients who are extremely anxious or who are experiencing a significant amount of postoperative pain. Therefore, in the postoperative period, pain and anxiety must be relieved by narcotic and/or mood-altering drugs. Smoking should be avoided in the late postoperative period as it has been shown to cause a reduction in digital flow. The patient experiencing spasm and eventual ischemia in the late postoperative period (1–2 weeks after repair) should be treated with anxiety-relieving medication and a warm environment. If the spasm is intense and unrelenting, an axillary block, which can be long-acting, will usually alleviate the situation. This will effectively relieve all spasms for a period of 36 hours. Proximal circumferential bandages can also induce the segmental spasm and should be avoided.

Anticoagulants have been used for the postoperative management of vascular reconstruction. Bell has shown that thrombocytopenia associated with the administration of heparin is fairly common. The thrombocytopenia characteristically occurred 2 to 10 days following administration of the drug, with the platelet count returning to pretreatment level in 3 to 5 days. Moore and Weiland presented a case of heparin-induced brachial artery thrombosis associated with thrombocytopenia. In their case, low-dose heparin therapy was used, and the patient developed problems on the fourth day after repair. The cause of the platelet thrombi is unknown, although an autoimmune mechanism may mediate the heparin-induced thrombocytopenia. Pre-existing arterial disease can play a role also. In the case presented by Moore and Weiland, the patient did exhibit fibromuscular hyperplasia. Therefore, there can be complications due to anticoagulant medication. This complication of platelet aggregation and thrombosis is treated by discontinuation of the heparin and rapidly restoring the circulating platelet volume. Other thrombolytic drugs such as streptokinase and urokinase can be associated with rather severe allergic reaction, especially in the case of streptokinase. The third complication which is sometimes seen is bleeding due to anticoagulant medication even in the late postoperative period. This is treated by discontinuation of the anticoagulant medication immediately. In our experience, there is no indication to use long-acting anticoagulant medication such as Coumadin in vascular reconstruction.

A third complication which can arise in the postoperative period is swelling and ischemia from the reperfusion syndrome. Muscle tissue that has been ischemic for a long period of time when reperfused undergoes rather marked swelling. The exact physiology behind this is now being actively investigated; however, it may be because of the fact that, during the ischemic phase, oxygen free radicals are produced that are destructive to local tissue. The current work on enzyme scavengers—such as superoxide dismutase, which eradicates the oxygen free radical—may indeed show that this reperfusion swelling and subsequent muscle necrosis may be prevented by the use of the enzyme scavengers. At any rate, there is a reperfusion swelling that must be observed and appropriate decompression carried out. If during the operative procedure distal fasciotomy...
has not been done, decompressive fasciotomy may be necessary, sometimes at 5 days when the swelling is maximum and the local inflammatory changes occur in the muscle tissue. A dreaded complication of a successful revascularization would be muscle necrosis because of swelling in the postoperative period. Of course, the easiest way to prevent such complications is to do the decompressive fasciotomy at the time of surgery. We strongly recommend that such procedures be carried out.

When revascularizing an individual with chronic obstruction and long-standing marginal ischemia, there is even more of a problem because it is unsuspected. Such an individual may undergo a revascularization in the shoulder region and may need a decompressive fasciotomy in the elbow and forearm postoperatively.

**SUMMARY**

Complications of vascular injuries, diseases, and reconstruction are all avoidable. Careful diagnosis and presurgical management, as well as surgical management and observation in the postoperative stage, will allow the treating physician to pick up impending complications and treat them accordingly. Close attention to detail in all three of these areas and close observation will prevent these complications.

**REFERENCES**