C J Mieny, U Mennen: Principles of Surgical Patient Care - Volume II

Chapter 11: Vascular Trauma

Chapter 11.1: Principles and Management

J V Robbs

If diagnosed early and treated appropriately, the management of vascular trauma can prove an extremely rewarding clinical experience. The acute management of injured arteries may, however, present some of the most challenging problems that can be faced by the operating surgeon, particularly with regard to access to and control of active haemorrhage.

An understanding of the aetiology, mechanisms of injury and pathology are essential in order to deal adequately with these problems.

Mechanisms of Injury

The damage following a stab wound is usually confined to the track of the penetrating agent and most times will result in a simple laceration or perforation. In contrast, the damage caused by missile wounds depends largely on their velocity. This can perhaps be best illustrated by Einstein's equation which states that energy equals half the mass multiplied by the square of the velocity ($E=1/2mv^2$). With low-velocity missiles in which the bullet leaves the muzzle of the firearm at a speed of less than 600 metres per second, generally speaking the energy release is confined to an area which is little greater than the missile track. However, an added dimension, compared with stab wounds, is the ricochet phenomenon where the missile is deflected from its course by hard tissue, for example bone, or may itself fragment and cause additional damage in this way. Most handguns, the commonest of which in our experience is a .32 or .38 calibre, fall into this category. High velocity missile wounds are encountered in the military context, or occasionally are seen with certain hunting rifles. The missiles with muzzle velocity in excess of 600 metres per second generates enormous energy release in its passage through the tissues. This causes a major cavitation effect and far more extensive trauma than that previously described. There is total tissue necrosis along the missile track for a varying margin around it. Surrounding this area there is a far larger margin of tissue with doubtful viability due to damage to the small blood vessels. The pathology is further compounded by the suction effect caused by cavitation which tends to draw in extraneous matter such as clothing or other debris via the entrance wound. All these wounds are therefore contaminated by bacteria. In addition, any bone contracted is shattered and these fragments may act as secondary missiles further increasing the area of damage.

Blast injuries such as those caused by close range shotgun missile wounds are characterized by extensive local tissue destruction which contains a large number of foreign bodies, particularly if the smaller calibre birdshot has been used. In addition, clothing and even cartridge wadding may be found. An additional frequent finding is that there are multiple sites of perforation within a segment of blood vessel. Embolization of the small missiles distal to the site of perforation is occasionally seen.

Bomb blasts also result in extensive local tissue trauma, but the entire situation is compounded by the fact that many of the fragments are, in fact, high velocity missiles which, under certain circumstances, are hot. These injuries are threfore extremely complex due to a combination of high-velocity missile, thermal and local soft tissue injury.

Blunt trauma may be focal, for example following a direct blow such as a kick. However, the indirect injury which results from shearing and distraction forces, is more subtle. Examples of this are injuries sustained by arteries in proximity to joints which are dislocated, for example, the knee and elbow, or in association with long-bone fractures. More subtle causes of shearing forces are seen in the acceleration/deceleration type of trauma which results from high-speed motor-vehicle accidents, or a fall from a height. Classically these lesions occur in the aorta, at the site of insertion of the ligamentum arteriosum into the proximal descending thoracic aorta, which is approximately opposite the site of origin of the left subclavian artery. At this site the aorta is fixed and relatively immobile. If forward motion of the patient is arrested as in a motor vehicle collision, forward momentum continues to carry the untethered part of the aorta forward, causing it to tear at the fixed area. Less commonly, the ascending aorta may tear at the aortic valve ring. This usually follows a fall from a height and under these circumstances the aortic arch is tethered by the great vessels and the heart continues to move downwards by ongoing momentum.

Iatrogenic injury warrants totally independent consideration. The most frequently encountered is that caused by catheterization of the artery whether during diagnostic procedures, for example cardiac catheterization, or for monitoring using arterial pressure lines. The important point to emphasize is that the pathology is always organic related to local trauma and the development of an ischaemic limb following arterial catheterization is never due to spasm in isolation. An additional important cause of iatrogenic injury is that resulting from over-distension of the vessel by a balloon embolectomy catheter. This is invariably a result of using fluid to distend the balloon, which creates a hydraulic system which is capable of generating high pressures. It is therefore preferable to inflate this balloon with air during the procedure. An additional important cause of iatrogenic injury is inadvertent intra-arterial injection of noxious substances. Important agents are those which are lipid soluble and thus become fixed in the small vessels causing local endothelial damage with superimposed thrombosis. Diazepam and Thiopentone are examples of these agents. The lesions so caused are not amenable to surgical correction.

Pathology

The various mechanisms described which lead to partial or total disruption of the blood vessel may result in a variety of pathological situations. Partial or complete severance of the artery may result in ongoing free haemorrhage. However, if the haemorrhage is contained by the surrounding tissues, a false aneurysm or pulsating haematoma is formed. Total interruption of the vessel may result in spasm of the ends with superimposed thrombosis. Under these circumstances there may be initial haemorrhage which subsequently arrests. However, it is important to realize that the clots may subsequently lyse and a delayed haemorrhage occur. If this occurs within 24 hours, it is usually a result simply of spasm passing off and the thrombus being dislodged. More frequently low-grade infection of the thrombus may occur and a delayed or secondary haemorrhage take place between five and 10 days later.

Coincidental perforation of an adjacent artery and vein may result in the formation of an acute arteriovenous fistula. In effect a large fistula results in gross iminution of peripheral resistance as the peripheral arteriolar and capillary bed supplied by that artery is totally short-circuited. Generally, a hyperdynamic circulatory state develops where reduction in peripheral resistance results in a drop of the diastolic pressure with elevation of the systolic pressure and heart rate increases. With time, by a mechanism which is ill understood, blood volume increases. Locally there is marked venous hypertension, both proximal and distal to the site of the fistula. In addition, there is ischaemia of the tissue distal to the fistula due to the shunting away of arterial blood flow. Ultimately, after a variable period, progressive cardiac decompensation may occur with cardiac failure, cardiomegaly and in addition manifestations of venous hypertension such as gross dilatation of superficial collaterals of oedema, particularly when an extremity is involved. If this occurs in children prior to epiphyseal closure, general overgrowth of the tissues subjected to increased blood flow will occur.

The intima is the most inelastic component of the artery when compared to the media and adventitia. Distraction or shearing will result in the artery tearing from the inside to the outside. The range of injury therefore varies from isolated intimal disruption, leaving an intact media and adventitia to total disruption of all layers of the vessel wall. This partial disruption exposes an area of denuded intima which is highly thrombogenic upon which thrombus will form. The condition may be compounded by the development of an intimal flap. This partial disruption may also weaken the vessel sufficiently to allow a true aneurysm to develčop with time.

Iatrogenic catheter trauma may result in all the consequences of penetrating injury described earlier. In addition, retrograde passage of the catheter may result in dissection between layers of the arterial wall with the formation of an intimal flap, which acts in turn as a nidus for thrombus formation. In a diseased vessel an atherosclerotic plaque may be dislodged or lifted resulting in thrombosis or distal embolization. Post-catheterization thrombosis is often misdiagnosed as "spasm" or "embolism" and treatment frequently is either expectant, or simple passage of a balloon embolectomy catheter is performed, which either aggravates the clinical situation, or results only in temporary improvement. Correct management requires a full understanding of the underlying pathology with appropriate local surgical correction. Mismanagement often results in considerable long-term morbidity, especially when the brachial vessels are involved, with propagated thrombosis into the smaller sital arteries.

Spasm

Spasm in an artery complicates any trauma to the vessel. This occurs at the site of injury, and also in the arterial tree distal to the injured area. This is due to the unopposed action of the normal sympathetic vasomotor tone, which is no longer countered by intraluminal pressure. However, spasm per se acting in isolation will very rarely, if ever, result in peripheral ischaemic manifestations, and as a cause of an absent pulse is a retrospective diagnosis made at operation or on good quality angiography. In our own series of over a thousand patients, we have only made this diagnosis in seven cases (0.7%).

Clinical Presentation

In principle, arterial injuries present with haemorrhage whether contained or revealed, or ischaemia, either occurring in isolation or in combination with haemorrhage. True aneurysm may occur in the larger vessels. Following penetration injury, the presentation may be subtle especially if arterial disruption has occurred in an area with rich collateral flow, for example in the cervicomediastinal area. Haemorrhage may also be concealed in, for example, the thoracic or abdominal cavity, or in a large-muscle compartment such as the thigh. Acute arteriovenous fistula may be characterized by minimal manifest haemorrhage as the artery decompresses directly into the vein, and the diagnosis only becomes apparent on auscultation. It is important to detect these lesions early when operative treatment can be performed through relatively normal tissue planes, as after delayed presentation extensive fibrosis makes operative treatment far more difficult. Blunt injury may be focal or indirect and in any patient who has a long-bone fracture, or a dislocation, particularly of the knee or elbow, an arterial injury should be regarded as being present until disproved. In addition, in any patient involved in a motor vehicle accident, or having fallen from a height acceleration/deceleration type, tears of the aorta should be considered.

Clinical diagnosis depends on the alertness and awareness of the clinician and every injury should be considered on its individual merits.

Clinical Assessment

The clinical approach to the patient is outlined below, and the following features should be carefully elicited:

History

--> The nature of the wounding agent, ie, missile, stabo, etc.

--> Site and possible trajectory of the penetrating wound. Particular attention should be paid to the neck or other areas where the vessels are relatively superficial, for example, the femoral triangle and medial aspect of the upper arm over the brachial artery.

--> Observed haemorrhage at any stage from the time of injury, in particular whether the patient has been shocked at the time of admission to hospital.

Examination

--> Particular attention should be paid to the patient's haemodynamic status, whether there is shock or any evidence of ongoing haemorrhage.

--> The presence of a false aneurysm with or without compressive symptoms, in particular, stridor.

--> The presence of a bruit in the area of the wound is the most reliable sign of an arteriovenous fistula.

--> The presence of a pulse deficit. Pulses should always be assessed in patients who are at risk of arterial injury, and any evidence of a diminished or absent pulse indicates organic vessel injury until proved otherwise. Never accept spasm as a cause for this. It is extremely important to consider the onset of a delayed thrombosis in patients who have sustained blunt trauma, and the limb circulation should be assessed at regular intervals for at least a 24 hour period in this group.

--> Additional signs of limb ischaemia such as pallor, diminished temperature, sensory disturbances or compartment rigidity.

--> Other considerations suggesting possible vascular injury are trauma to anatomically related nerves, for examople, the brachial plexus.

Investigation

It must be re-emphasized that any form of investigation is only undertaken in the volume-resuscitated stable patient.

Doppler Ultrasound

This is useful in detecting flow in the impalpable artery. The presence of a Doppler signal does not exclude injury. Any diminution in the leg to arm pressure index which should exceed a numerical value of one, indicates an injury which should be confirmed on angiography.

Chest X-Ray

Valid conclusions may be drawn only by studying good quality erect films. In particular widening of the superior mediastinum should be sought which indicates a haematoma in this area. This is especially useful in the assessment of patients with penetrating wounds in the root of the neck, or those who have been subjected to the acceleration/deceleration trauma sustained as a motor-vehicle driver or passenger. In addition, other evidence of intrapleural haemorrhage such as haemothorax should be sought.

Angiography

Good quality angiography serves to make the final diagnosis in a patient with suspected arterial trauma. In those with suspected upper extremity, or cervico-mediastinal injury, an arch aortogram with selection of the appropriate vessels via a retrograde femoral route should be obtained. This procedure is not only invaluable in confirming the presence of vascular injury, but is also essential in planning the surgical approaches to what may be extremely inaccessible sites of injury. For injuries confined to the extremities, direct angiography can be obtained. However, in the presence of focal isolated trauma, as for example a stab wound or a fracture with absent pulses distal to the site of injury, exploration should be performed without angiography as this saves valuable time. Problems may, however, arise in patients with multiple fractures in the same limb, or with extensive soft-tissue injuries. Under these circumstances angiography should be performed and the entire artery imaged at every site of injury. If necessary this investigation may be carried out at

operation (intra-operative angiography). The indications for angiography based on suspicion of vascular injury are summarized in table 11.2.

Table 11.1.2. Indications for angiography in the haemodynamically stable patient

Suspected Cervicomediastinal Injury

--> Pulse deficit
--> False aneurysm
--> Haemorrhage
    - overt
    - unexplained shock
    - low haematocrit/haemoglobin value
--> Bruit
--> Injury to anatomically related nerves
--> First-rib fracture
--> Widened mediastinum on chest X-ray

Ischaemic Limb

--> Multiple fractures
--> Severe associated soft tissue injury (degloving)

Suspected Intra-Abdominal or Renal Arterial Injury

Management

The importance of a full general appraisal with a holistic approach to the patient's clinical status must be emphasized. The vascular injury must not detract from other possible injuries, and vice versa. Every individual injury must be considered on its merits and priorities of management should be decided upon. The first consideration is resuscitation which follows the basic principles of establishing an airway, placing wide-bore intravenous cannulae, and adequate monitoring (CVP, BP, urine output). Once stabilized, the patient may be fully assessed and investigated as described. There is a group of patients with large-vessel injury in whom operative intervention for control of haemorrhage constitutes part of the resuscitative process. Besides active haemorrhage an equally urgent situation is presented by the patient in whom an expanding false aneurysm constitutes a threat to life due to its compressive effects. In particular this applies to the trachea following injuries to the superior mediastinal vessels. Under these circumstances a patient with stridor requires urgent sternotomy with decompression of the haematoma and control of haemorrhage without delay.

Arterial Repair

In principle all injuries should be repaired even if there is no active haemorrhage or immediate limb threat due to the ever-present danger of delayed haemorrhage with exsanguinating bleeding or the development of a compressive haematoma which may result in respiratory embarrassment or compression of neighbouring nerves. This applies particularly to the brachial plexus. The consequences of the neglected arteriovenous fistula between major

vessels has been outlined. Contraindications to attempting vascular repair are presented by the patient with a severely traumatized limb with total disruption of the nerves and blood vessels. Under these circumstances it is preferable to perform primary amputation. However, this assessment really depends upon considerable experience and, as a general rule, if the tissues are deemed viable it is probably advisable to revascularize initially and consider amputation at a later date. A more difficult problem is presented by the question of limb viability when there has been some delay in instituting treatment. Limb-threatening ischaemia is represented by the signs of anaesthesia, total paralysis and muscle rigidity; of which muscle rigidity is the most significant sign. If this is confined to the extensor compartment of the leg, it does not preclude vascularization, but is a definite contraindication to restoring the circulation, and it is advisable to proceed directly to amputation. Obviously fixed-skin staining or frank gangrene also mandate immediate limb ablation.

Principles of Vascular Repair

The details of access to various vessels are beyond the scope of this chapter. It must be emphasized, however, that wide exposure of the traumatized vessels must be assured with adequate proximal and distal control. The procedure must be adequately thought out and planned prior to commencing the operation, and there is *no* place for "keyhole" surgery in attempting to expose the site of injury via pre-existing stab wounds. For example, attempting to control and repair vessels in the superior mediastinum via inadequately placed incisions in the neck invites, or indeed, ensures disaster. In the compound injured limb with associated orthopaedic trauma, the vascular injury should take precedence and the repair be completed prior to allowing the orthopaedic surgeons access to the injury. In our practice it is a rare occurrence for the orthopaedic manipulation to disrupt the vascular repair, although the vascular surgeon should remain in attendance until conclusion of the operative procedures.

Arterial continuity may be restored by simple lateral suture, patch angioplasty, end-to-end anastomosis or by the placement of an interposition graft. If any graft or patch is required autogenous vein, preferably the long saphenous, is the optimal material to use. Prosthetic material should be reserved for last resort situations and in our experience is very rarely necessary.

In order to adequately plan and repair thesxe injuries, it is essential fully to understand the reasons why attempts at repair occasionally fail. These can be summarized as follows:

--> tension at the anastomosis

--> failure of intimal apposition allowing a distal flap to form

--> incorporation of adventitia into the lumen. This acts as a nidus for thrombus formation.

--> narrowing at the suture lines

--> inadequate calibre graft

--> inadequate distal run-off due to propagated distal thrombus. This frequently occurs when repair has been delayed for some hours, and once this has spread into the venous system there is little hope of successful restoration of flow. An additional cause of compromised run-off may be the presence of an additional arterial injury in the distal system.

--> infection at the site of repair resulting in thrombosis or disruption of the suture lines. This usually occurs some days later.

In performing the repair and selecting the method of restoration of arterial continuity, these factors must be avoided. The edges of the arterial lacerations must be adequately debrided or the damaged segments of artery excised. It is important to repair the defect without tension, or allowing narrowing to occur. The decision as to precisely what method to use, depends on an appraisal of the situation with regard to avoidance of these adverse factors.

Ancillary Procedures

Venous injuries should be repaired whenever possible. This assumes particular importance in the lower limb where chronic venous insufficiency may follow venous disruption. In the upper limb interruption of major veins is far better tolerated and rarely causes long-term problems. The same principles outlined above apply to venous repair. The veins, however, are much more delicate and thinner-walled structures so that extremely gentle and meticulous technique is essential. However, the results of venous repair in terms of patency are not as good as those to be expected following arterial reconstruction.

It is generally considered important to separate the sites of repair of the arterial and venous components of an arteriovenous fistula by means of interposed fascia in order to prevent recurrence. In our experience it is often difficult to find appropriate tissue for this purpose and we have not done this as a routine. There have been no recurrent fistulae in this group of patients, and we no longer consider this to be necessary.

In the repair of bone and joint injuries, rigid fixation is desirable, using an intramedullary rod or a plating technique, or an external fixation device. Liberal use of the latter method has greatly improved management in patients with open wounds and when fasciotomy is done.

Raised intracompartmental pressure may occur in the presence of gross associated soft-tissue trauma with haemorrhage into the compartment, revascularization of a critically ischaemic limb, or in the presence of associated venous occlusion. This applies mainly to the lower limb, but may occasionally occur in the upper limb. The development of compartment syndrome should be anticipated and a fasciotomy performed prophylactically when necessary. The measurement of intracompartmental pressures may be of some value, but there is difficulty in obtaining reliable results, and indeed in interpreting the measurements obtained. As a general rule, intracompartmental pressures in excess of 30 mm of mercury are regarded as excessive and place the muscle at risk of necrosis. However, in the clinical situation it is preferable by far to be more liberal in one's indications and in essence, if on palpation the compartment is considered "tight" or there is extensive associated soft-tissue trauma or the limb is critically ischaemic at the time of reconstruction, prophylactic fasciotomy should be performed. Decompression of all four compartments is favoured in the lower limb, and the reader is referred to more comprehensive texts for detailed description.

In most injuries that have extensive associated soft-tissue trauma, or in which there has been a large false aneurysm or in which there has been considerabl delay before repair is effected, a policy of delayed primary closure of the wound should be followed. This entails loose approximation of the deeper structures with the skin either left open or loosely tacked together. Following a wound inspection 48 hours later, if this appears viable and healthy,

formal closure may be carried out. This policy of wound management has significantly reduced the incidence of infection and secondary haemorrhage in our own practice.

Postoperative Complications

Complications occurring within five to ten days of operation (early complications) may be local or systemic. The most frequent local complications are:

--> thrombosis
--> haemorrhage
--> compartment syndrome
--> infection
--> recurrent arteriovenous fistula.

Graft thrombosis usually results from a local mechanical cause, the result of technical misadventure, and is noted within 24 hours of operation. Haemorrhage occurring within hours of the procedure is due to inadequate haemostasis. It may, however, occur several days later due to infection with lysis of the thrombus; vein grafts tend to disintegrate if they become infected.

Compartment syndrome becomes apparent six to 24 hours after revascularization and must be anticipated particularly under the circumstances already outlined. True recurrence of an arteriovenous fistula is rare and persistence of the physical signs usually means that an additional fistula has been overlooked. This occurs particularly in the presence of missile wounds or shotgun blast injuries.

Systemic complications such as disseminated intravascular coagulation, adult respiratory distress syndrome and the multiple organ failure syndrome are the complications of massive blood transfusion and extensive multiple injuries and should be anticipated in patients who fall into this category.

The myonephropathic syndrome (crush syndrome) accompanies severe crush trauma or the restoration of circulation to necrotic tissue. Reperfusion under these circumstances results in flooding of the systemic circulation with the products of cell disruption and anaerobic metabolism. The nett effect is a profound metabolic acidosis, hyperkalaemia and the possible deposition of myoglobin crystals in the renal tubules which results in mechanical blockage of these tubules with the development of acute renal failure. With adequate anticipation and prophylaxis the complication should not occur. It is important to reperfuse the limb gradually by intermittent release of the clamps, to adequately treat the anticipated acidosis and to attempt to render the urine alkaline under which circumstances the myoglobin remains soluble. In addition, it is important to maintain a good urine flow in order to mechanically wash out the tubules; a flow of 50-100 mL per hour is adequate. It is advisable in susceptible patients to initiate diuresis with Mannitol infusion on the operating table prior to limb reperfusion provided that the patient is normovolaemic.

During the postoperative period the blood volume, urine output and acid base balance must be meticulously maintained. In addition, the urine must be monitored for the appearance of the characteristic brown discoloration caused by the presence of free myoglobin.

While injury to the blood vessels may represent a simple and unesoteric problem in many cases, there are situations in which prompt decision-making and meticulous management are essential in order to save life and/or limb. It is only by a good understanding of mechanisms of injury, pathology, principles of repair and the anticipated complications that satisfactory results may be achieved.

Comment

Vascular Trauma

L J Levine

While it is convenient from a practical point of view to group missile injuries into low velocity (under 600 metres/sec) and high velocity (over 600 metres/second), it should be appreciated that tissue damage extends beyond the permanent local track in all buller and missile wounds. The extent of local injury is dependent not only on the kinetic energy of the missile, but also on the rate of energy transfer. Thus slower moving but more unstable or irregular missiles can cause considerable tissue necrosis. Blunt trauma such as a knee or elbow dislocation may also result in extensive soft-tissue destruction in addition to any vascular injury.

It is essential to the ultimate success of any vascular repair that all necrotic tissue is thoroughly debrided and that only healthy, well vascularized tissue is left behind at the time of the original vascular repair. Failure to adhere to this principle will result in infection in the remaining necrotic material with consequent secondary breakdown of the graft (vein) or anastomosis and secondary haemorrhage.

Venous injuries, particularly of the lower limb, should be repaired if possible at the same time as the arterial repair. Although venous repair does not guarantee vein patency, venous repair does diminish the venous pressure distal to the site of the injury, particularly at the time of restoration of flow, and it probably has a contributory role to play in the maintenance of the arterial repair patency as well as helping to diminish the oedema distal to the site of injury.

Liberal use of fasciotomy cannot be stressed too strongly. Waiting to see if a limb requires a fasciotomy until such time as muscle compartment tension is clinically obvious, is not advised. One would rather advocate performing a four-compartment fasciotomy *prior* to releasing the clamps of the arterial repair. This reduces bleeding from the fasciotomy considerably.

Chapter 11.2: Arterial Embolism and Thrombosis

J V Robbs

An embolus may be defined as an abnormal mass of undissolved material which is transported from one part of the circulation to another, whereas a thrombus is a solid mass in the circulation formed from the constituents of the streaming blood. The context of acute vascular emergencies discussion is confined to embolism or thrombosis occurring in the large

vessels with exclusion of micro-emboli impacting or thrombi forming in "end arteries", for example the digital, ophthalmic or intracranial vessels distal to the circle of Willis. In general surgical practice the most frequently encountered problems related to acute ischaemia occur in the extremities and in particular the lower limbs. Therefore, although the principles of pathology and pathophysiology apply universally to occlusion in large vessels, more specific reference will be made to the limbs and in particular the lower limbs. In general, embolism occurs acutely in a previously asymptomatic limb. Thrombosis usually occurs in association with underlying arteriopathy and in general will occur in a patient with previous chronic ischaemic symptoms. However, it is not uncommon for a patient with a chronic problem to have a superimposed acute embolus. In addition, thrombosis may supervene on asymptomatic chronic occlusive disease. Although from the pathological point of view it is convenient to consider the entities of embolism and thrombosis separately, from the practical clinical point of view the differentiation is not always clear.

Aetiological Considerations

Embolism

Thrombus forming in the circulatory system and then dislodging constitutes the majority of emboli occurring in the larger vessels. Far less frequently atheromatous debris or foreign bodies may be responsible. The latter are most commonly iatrogenic following fragmentation of catheters during diagnostic or monitoring procedures. Also not uncommon, small missiles such as birdshot which cause self-sealing perforations in, for exmple, the aorta, may embolise into the distal artery system.

The most frequent source of thrombotic emboli is the heart (85%). Clot may form in the atrium as a complication of atrial fibrillation which in turn may result from valve disease, ischaemic heart disease or thyrotoxicosis among other causes. Diseased or prosthetic heart valves may be a focus for thrombosis but these emboli are often small and tend to impact in digital vessels. The ventricle may form a focus for thrombus formation on an area of endocardial damage due to myocardial infarction, the so-called mural thrombus. Within the great vessels aneurysm or large calcified ulcerating plaques may form a focus for the formation of thrombotic emboli. A cardiac tumour, most frequently a myxoma arising from the atrium, is an extremely rare cause of peripheral embolism. The embolus is usually thrombus that has formed around the tumour. However, it has been reported that fragments of the tumour itself may break off and embolise distally.

Important ancillary intraoperative manoeuvres should be aimed at preventing episodes of hypotension which may follow the entry into the systemic circulation of the metabolic products of anaerobic metabolism described earlier. If there has been prolonged ischaemia and the muscle is of doubtful viability, special prophylactic measures should be taken against renal failure caused by myoglobinuria. Under these circumstances the patient would be rendered alkalotic by sodium bicarbonate infusion in order to alkalinize the urine. In addition, an osmotic diuresis should be generated by the administration of Mannitol in a dose of 2.5 g/kg. It is important to initiate these manoeuvres prior to release of the arterial clamp. It is also advisable to release the clamp gradually, and reclamp if hypotension occurs so as not to totally flood the circulation. The aim is to achieve a urine output in the region of 100

mL/hour. During the postoperative period the urine output should be maintained at a level of 100 mL/hour until such a time as the myoglobinuria, if it occurs, has cleared.

It is advisable to heparinize these patients routinely until such time as they are able to walk and for the first 24 hours to administer low molecular weight dextran in a dose of 500 mL 8-12 hourly in an attempt to improve capillary bed circulation. Most of the manoeuvres described are mainly applicable to the patient with grave ischaemia.

Postoperative Complications

Local complications include haemorrhage form the operative site or reocclusion. This may result from repeat embolisation or rethrombosis, either due to extensive intimal damage from the catheter or to failure to recognize the underlying chronic pathology.

Revascularization oedema may result from restoring flow to a critically ischaemic extremity. The mechanism have been discussed at length. These patients are at risk of developing a copmpartment syndrome and require fasciotomy. This should be performed by the open technique and it is advisable to perform the operation at the time of initial revascularization in those at risk.

The myonephropathic metabolic (crush) syndrome may occur in the early postoperative period if necrotic muscle has been revascularized. The mechanisms and consequences of this have been discussed earlier. The most important facet of treatment involves prophylaxis and awareness of the possibility of its development. With adequate prophylaxis and maintenance of a good diuresis, this complication should not occur.

Infection may occur within fasciotomy wounds especially if there is residual tissue of doubtful viability. Of particular import is the possible development of gas gangrene due to clostridial infection or other anaerobic synergistic infections. The wound should be inspected within 24 to 48 hours and debridement carried out to remove slough and necrotic tissue. The procedure should be repeated at 24 to 48 hourly intervals until there is no further evidence of necrotic tissue.

Other systemic complications result from the underlying disease processes, for example, cardiac decompensation, etc.

Medium and Long-Term Management

It is of vital importance to elucidate the cause of the acute occlusion. In particular acute embolic occlusion should be regarded as a symptomatic part of some underlying disease process. Once the patient has recovered from the acute episode, a full assessment should be undertaken during the same hospital admission. A cardiac cause should be sought by echocardiography and electrocardiography.

In principle, embolic material tends to impact at bifurcations. Cardiogenic emboli will lodge in the circulation to the lower extremities in 80-85% of cases. Ten to 15% impact in the upper extremities and the remaining 4-5% will enter the extracranial cerebral vessels or the abdominal visceral arteries. In our own experience of those involving the lower limbs,

almost half impact at the common femoral bifurcation. The next most frequent site is the popliteal trifurcation followed by the common iliac and aortic bifurcations respectively. Aortic bifurcation emboli affect both limbs and the phenomenon is known as saddle embolism. If the source of embolus is a focal lesion such as an aneurysm or a plaque, the embolus will obviously lodge in the arterial tree distal to the source.

Thrombosis

The triad of stasis, intimal damage and increased coagulability described by Virchow, usually acting in concert, have stood the test of time. For the initiation of the thrombotic process in the arterial tree the prerequisite is intimal disease, such as an ulcerating arterial plaque, an aneurysm or intimal disease caused by an arteritis. The precipitating factor for thrombosis to occur may be a critically stenotic lesion with a consequent low flow state. Low flow states within the diseased arterial tree may also be contributed to by acute periods of cardiac decompensation such as myocardial infarction or arrhythmia which may potentiate an otherwise compensated stenosis. Blood coagulability may be affected by increases in viscosity such as during periods of dehydration or by polycythaemia or thrombocytosis. These may themselves constitute part of a systemic blood dyscrasia or may be a secondary phenomenon caused by underlying lung or renal disease which is further aggravated by cigarette smoking.

Primary thrombosis occurring in an apparently healthy arterial tree does occur but is an extremely rare phenomenon. We have seen it as a complication of severe thrombocytosis or extreme polycythaemia.

The most frequent underlying arteriopathy is atherosclerosis of which the classical distribution in the extremities involves the aortoiliac, femoropopliteal or tibioperoneal segments. The coronary and cerebral vessels are also common sites for thrombotic complications, but specific problems relating to these areas will not be discussed in this chapter.

Pathophysiology of Acute Arterial Occlusion

Once the embolus has lodged or thrombosis has been initiated and the vessel lumen becomes occluded, thrombus propagates proximally and distally to the site of occlusion to the level at which the proximal and distal collateral vessels enter, at which point active flow is occurring. The immediate pathological response is an acute inflammatory reaction within the wall of the artery resulting in oedema which is well established within two to three hours. If the thrombus remains, the full sequence of the inflammatory reaction continues to organization and fibrosis within seven to 10 days. The longer the inflammatory process is allowed to continue, the more adherent the thrombus becomes to a friable oedematous intima. At that stage attempts to remove the thrombus result in denudation of the endothelial surface of the vessel thus creating a thrombogenic surface which may initiate a recurrent thrombotic process. The reduction in the intraluminal pressure distal to collateral flow in the arterial tree results in an initial "clampdown" of muscular arteries and arterioles due to unopposed sympathetic tone. This further reduces distal flow because of an increase in peripheral resistance thus setting up a vicious cycle. Eventually, however, the products of anaerobic metabolism in the ischaemic tissue, which are powerful vasodilators, result in local vasodilatation with consequent reduction in peripheral resistance.

There is an increasing appreciation and awareness of the damage caused by free radicals generated during the reperfusion of ischaemic tissue whether by collaterals or otherwise. Free radicals are created by the process of gaining or losing an electron. Once this occurs, the compound becomes unstable and highly reactive as an oxidizing or reducing agent. The need to acquire an extra electron and regain stability is urgent and the molecule will pull these from adjacent molecules. These radicals thus have the ability to participate in chain reactions and create general molecular chaos with loss of function. Oxygen is particularly susceptible to free radical conversion. Gaining one electron convers it to superoxide, the second to hydrogen peroxide, the third forms a hydroxyl group and the fourth forms water. Superoxide and hydroxyl are powerful oxidizing agents and are responsible for the cellular damage.

It is hypothesized that the process is initiated when the decrease in blood flow to tissue is sufficient to limit oxygen availability for the production of ATP, the major energy source for cellular metabolism. Depletion of ATP results in elevated levels of AMP which is catabolized to hypoxanthine which accumulates in the tissue. Simultaneous with this ATP depletion, the cell is unable to maintain normal ion gradients across membranes. Calcium accumulates in the cell cytoplasm and activates a protease which activates an enzyme, xanthine dehydrogenase to xanthine oxidase. When oxygen is reintroduced into the system by reperfusion, the xanthine oxidase mediates the production of free oxygen radicals which then potentiate tissue damage. Naturally occurring defences against the activity of the free oxygen radicals are certain enzymes (superoxide dismutase, catalase and glutathione peroxidase). Other radical scavengers are iron-binding proteins, vitamin E, beta carotene and ascorbic acid. Mannitol has also been shown to modify the rate at which this process occurs. The susceptibility of various tissues to ischaemia seems to be proportional to the rate at which the inactive xanthine dehydrogenase is activated prior to reperfusion.

During the ischaemic period with minimal collateralization a combination of anaerobic metabolism, failure of the sodium potassium pump at cell membrane level and muscle necrosis, the venous effluent from an ischaemic limb contains a high level of lactic acid and an increased PCO_2 resulting in a fall in pH. Potassium levels are also high as well as various intracellular enzymes (CPK, LDH and SGOT). Myoglobin is also released from the necrotic muscle cells. Following reperfusion of an ischaemic limb this bolus of "metabolic debris" enters the systemic circulation and the patient may develop acidosis, hyperkalaemia and myoglobinuria. The myoglobin crystals precipitate out in the renal tubules, particularly in the acid urine, and block them causing oliguria and it may lead to renal failure (myonephropathic metabolic syndrome or crush syndrome). Profound hypotension may also occur due to a combination of diminution in cardiac contractility and peripheral vasodilatation resulting from the acidosis and hyperkalaemia. Within the limb itself endothelial damage results in severe interstitial oedema, the extent of which depends upon the degree and duration of the ischaemia. Oedema within the muscle compartments may result in compartment syndrome. The most vulnerable muscle group is the ankle and toe extensors and the peroneal group of muscles.

Factors Influencing Limb Survival

In the progression of an ischaemic process the blood supply is adequate at rest in the initial stage, but inadequate on increasing the demand during exercise (claudication). The

following stage is ischaemia at rest which is reversible (postpain but viable tissue). The final stage is irreversible tissue damage or necrosis. The time scale for this sequence is dependent on three major factors, namely:

--> the development of collaterals
--> the ability of the blood to deliver oxygen to the tissues and
--> the metabolic demands of the ischaemic tissue.

The development and efficiency of collaterals depend upon the rate at which occlusion occurs and the extent of occlusion, ie, the propagation of thrombus. The more rapid this process, the less time there is for collaterals to develop. Other important factors are the cardiac output, the state of patency of the inflow vessels, the outflow vessels and the collaterals themselves. The cardiac output in turn depends on myocardial contractility and the circulating blood volume.

Oxygen delivery depends on the efficacy of the haemoglobin and the flow characteristics of blood. Anaemia, for example, will result in diminished oxygen transport and delivery. The most important factor in blood flow is viscosity. Marked increase in viscosity results from polycythaemia for whatever cause and a diminished plasma volume resulting from, for example, burns or dehydration.

The metabolic rate of the tissues and hence their demand for oxygen and nutrients vary from tissue to tissue, with nerve tissue having the highest demands and skin the least. These metabolic demands may be increased by the stresss of exercise or infection and decreased by cooling or rest.

The vulnerability of different tissues within a limb to ischaemia, varies. Peripheral nerves are most vulnerable and cease to function within 10 to 15 minutes. Muscles will become irreversibly damaged within three to four hours and the least vulnerable is the skin and appendages which will show irreversible changes only after about 12 hours. It is not known in the human situation at what stage the totally ischaemic peripheral nerve becomes irreversibly damaged as all these statements are based on clinical observation.

It is thus clear that limb survival following an acute ischaemic insult depends upon a large number of interacting factors. No absolute time interval can therefore be given within which revascularization should not be attempeted.

Each individual case must be carefully assessed on its merits whatever the duration of the acute ischaemia.

Clinical Considerations

Diagnosis and Assessment

It is important to decide whether the occlusion has followed an acute embolic or thrombotic episode as the approach to management differs. Embolism is treated primarily by catheter thromboembolectomy while thrombosis usually involves more extensive reconstructive surgery. Embolism is classically characterized by a dramatic acute onset with

no antecedent history of limb ischaemia. Cardiogenic causes are evident if a history of previous myocardial infarction, valve disease or replacement is obtained or physical signs of cardiac arrhythmia are present. In contrast, thrombosis often occurs superimposed upon a background of chronic limb ischaemia, has a more gradual onset over a period of hours and there is often evidence of chronic occlusive disease in the opposite limb. However, embolism may occur in a patient with chronic peripheral ischaemia. The situation may be further confused by thrombosis occurring in a patient with hitherto asymptomatic chronic disease who develops an episode of hypotension, particularly following myocardial infarction.

The acute onset of ischaemia is characterized by pain, paraesthesia and motor weakness or even paralysis as nerve conduction is disturbed. Examination reveals a cold limb which is pale with absent pulses distal to the site of the occlusion. This can classically be summarized as the six p's: pain, paraesthesia, paralysis, pallor, pulselessness, perishing with cold.

If the ischaemia progresses and the situation deteriorates, signs of increasing interstitial oedema in the deep muscle compartments of the leg develop. The most vulnerable in this respect is the extensor compartment. This is characterized by increasing tissue turgor with tenderness on palpation. Eventually muscle necrosis may supervene with the development of muscle rigidity which is as hard as wood. The latter situation may pertain even in the presence of viable skin. The onset of skin necrosis is manifested by the presence of fixed-skin staining which results from the rupture of dermal capillaries leading to intradermal haemorrhage. Frank gangrene eventually follows. While total anaesthesia and paralysis indicate grave critical ischaemia, these manifestation do not necessarily indicate that this is irreversible. In terms of restoring circulation skin staining or gangrene are absolute contraindications. Viable skin with muscle rigidity confined to the extensor compartment are compatible with limb salvage. Once muscle necrosis has extended into the flexor compartment of th eleg it would be folly to attempt revascularization.

The diagnosis and assessment is essentially based on clinical parameters. If the features for acute embolism are classical, it is advisable not to waste time but to proceed directly to embolectomy. Preoperative angiography should be reserved for patients in whom doubt exists, and in whom it may be necessary to perform an emergency bypass procedure. In these patients attempts at catheter embolectomy may cause further damage to collaterals and aggravate a potentially salvageable situation.

Management

The priority is to restore flow to a salvageable extremity as soon as possible. However, attention must be given to the general condition of the patient, in particular to the cardiac status, ie, whether the patient is in congestive cardiac failure and in particular whether a silen myocardial infarct has occurred. This is a notorious problem in diabetic patients. In addition, the state of hydration should be carefully assessed as dehydration may further aggravate the local circulatory impairment. As far as possible these general problems should be corrected before surgery. Unless operative management will be delayed for some hours, anticoagulation is not advisable at this stage. *Adequate* analgesia is of extreme importance, as the pain of acute ischaemia is extremely severe. Opiates are usually necessary for this purpose, which also serves to sedate an almost invariably anxious patient. With regard to local treatment of

the limb, local pressure to the heels or the sides of the feet should be avoided, they should be maintained at room temperature without attempts at cooling or particularly warming, and should probably be maintained in a horizontal position and not be allowed to hang down.

The surgical options are dictated by whether the limb is salvageable or not. If the ischaemia is deemed irreversible, primary amputation at the appropriate level should be carried out as soon as possible. If the line of demarcation is uncertain, guillotine amputation should be performed without wound closure as a primary procedure with revision and secondary closure once viability is established. We have found transcutaneous oxygen measurement extremely useful in this decision-making process.

Classical acute embolism in a viable limb should be treated by balloon catheter embolectomy. Although this procedure can be performed under local anaesthesia it is preferable to use a general anaesthetic. Access to the arterial system is via the femoral artery with proximal and distal passage of the catheter. It is advisable to inflate the balloon with air rather than liguid in order to avoid creating a hydraulic system which is capable of generating considerable pressures and may damage the artery by overdilatation. In addition, copious regional irrigation of both the proximal and distal arterial tree should be performed using a diluted heparin saline solution (10000 units per litre isotonic saline). Prior to commencing this procedure, facilities should be made available for intraoperative angiography. On completion of the thrombo-embolectomy an angiogram should be done to ensure that adequate patency has been restored. Should any difficulty be encountered in initial passage of the catheter the problem should be further elucidated by angiography. If a preoperative diagnosis of thrombosis is made, the patient requires emergency bypass. In that group of patients in whom the clinical diagnosis is uncertain, a judicious attempt may be made to pass an embolectomy catheter. If this proves unsuccessful the appropriate bypass procedure should be performed. Deatils of these operative techniques are byond the scope of this chapter.

A routine pan aortogram should be performed in a search for a local cause and a routine full blood coagulation profile should also be performed to ascertain whether there are any aggravating factors. Long-term definitive treatment depends on these findings and the final diagnosis. If a specific local aortic lesion such as an aneurysm is found, this should be dealt with surgically. Long-term anticoagulation therapy is reserved for patients with a non-remediable cause such as myocardial damage, atrial fibrillation, etc. There remains a group of patients, approximately 10% in our own series of fully investigated patients, in whom no cause can be found. In these careful follow-up should be maintained and it is probably advisable to systematically anticoagulate this group.

Acute arterial occlusion may present an extremely challenging clinical problem, from the point of view of both diagnosis and treatment, and great discretion and judgement will be required to ensure that a good result is achieved.

Comment

Arterial Embolism and Thrombosis

L J Levine

Embolic disease causing major arterial occlusion arises from the heart in 85% of cases. Such cardio-arterial emboli arise most commonly from mitral stenosis, atrial fibrillation, mural thrombus following myocardial ischaemia or infarction, or from a prosthetic cardiac valve. Arterio-arterial emboli most commonly arise from a large aneurysm. Smaller arterio-arterial emboli may arise from an area of complicated atheroma, but usually present with micro-embolisation manifested by digital gangrene or "trash foot". The majority of the cardiac or arterial conditions which give rise to peripheral emboli are therefore usually readily diagnosed. In the absence of one of these conditions, it is advisable not to diagnose an embolic cause for acute arterial insufficiency, but rather to obtain angiography as a prelude to exploration in case a bypass is required to restore snormal arterial perfusion.

From a practical standpoint it is out policy to immediately anticoagulate any acute arterial insufficiency. Should an embolic source be evident (cardiac lesion or proximal arterial aneurysm) an embolectomy will be performed provided the patient is fit for the procedure. If no embolic source is evident, the timing and urgency of further therapy depends on the results of a period of conservative treatment, including anticoagulation. If the limb remains critically ischaemic, urgent angiography and exploration are warranted within hours. However, should a satisfactory restoration of collateral flow occur during the period of conservative treatment and anticoagulation, so that the limb is no longer critically ischaemic, angiography and possible surgery can be delayed until the patient has been fully investigated and his condition optimized.

Chapter 11.3: Aneurysms and Their Complications

J V Robbs

By definition, an aneurysm is a localized or diffuse dilatation in an artery. This is a vast subject and this chapter aims at outlining the general principles with emphasis on the complications and specific details relating to regional problems will not be described other than as illustrative examples.

The original pathological classification described the morphology and structure of the aneurysm and are illustrated. A true aneurysm is one in which all the layers of the arterial wall constitute the wall of the aneurysm. In a false aneurysm there has been a breach in the wall of the artery with the formation of a perivascular haematoma contained by the surrounding tissues. This wall, with time, becomes fibrosed and organized. With a dissecting aneurysm there is a focal breach in the intima and blood tracks down between the layers of the arterial wall. Further descriptive terms applied to the above relate to the form of the aneurysm which may be saccular in which the aneurysm has a narrow neck, or fusiform, in which it assumes a more diffuse, elongated form. The anatomical position, ie, central or peripheral, splanchnic, renal or cerebral, is also used in classifying aneurysms.

There is a great degree of overlap in specific aetiological entities which may result in the above morphological descriptions. There is no clear-cut specific classification that embraces all possibilities. It is therefore necessary to adopt a combined classification which embraces aetiologies as well as pathological descriptions.

Aetiology and Pathogenesis

The aetiology of aneurysms is summarized in table 11.3.1. The development of an aneurysmal process is determined by a disturbance of the balance between the physical integrity of the arterial wall and the mechanical stresses placed upon it, resulting from the intraluminal blood pressure. In virtually all the causes listed the *initiating* factor is a weakness in the wall of the artery, whether by some destructive process or by degeneration of the collagen and elastic tissue which are largely responsible for the integrity of the arterial wall. Intraluminal pressure is then sufficient to cause and maintain progressive dilatation. Hypertension acts as an accelerating factor. The exception to the above is the entity of post-stenotic dilatation in which the wall of the aneurysmal area is not intrinsically pathological. This occurs under the circumstances of a focal stenotic area in an otherwise normal vessel. Examples are coarctation of the aorta or cervical rib impinging upon and compressing the artery. Once a stenosis of this nature becomes haemodynamically significant Bernoulli's principle applies which states in essence that there is slow flow lateral to the central jet stream distal to the stenotic area with release of kinetic energy and a greatly increased lateral pressure on the arterial wall. With time, dilatation therefore results. This phenomenon is complex and ill understood as it has not been possible to clearly demonstrate this increased lateral pressure in a biological system.

Once dilatation has been initiated, this tends to progress. Circumferential stress applied to the arterial wall is a function of the transmural pressure (the intravascular minus extravascular pressure), the inside radius of the artery and the wall thickens. This is expressed as:

Circumferential stress = Transmural pressure x (Radius/Wall thickness).

It can thus be seen that the stress on the wall increases as the radius increases. In addition, the wall thins out progressively as it stretches and in this way a vicious progressive accelerating cycle is produced. As always, however, in any biological system there are many unaccountable variables and there is no specific prediction of the rate at which this process will progress.

As alluded to earlier, any one of the aetiological factors outlined may result in any morphological type described although some entities are more likely to result in true aneurysms, others in false aneurysms or in dissecting aneurysms. In addition, certain pathological types are found more frequently in certain anatomical areas, for example, non-specific aorto-arteritis will more frequently affect the aortic arch and its branches than will arteriosclerosis.

Arteriosclerosis is the commonest single cause of true aneurysm. At the other end of the spectrum, penetrating trauma will cause a false aneurysm, whereas blunt trauma may be responsible for arterial rupture or sufficient damage to the media to lead to the eventual

formation of a true aneurysm. Similarly, anastomotic aneurysms are equally divided between false aneurysms resulting from suture line leakage and true aneurysms forming in the arterial wall adjacent to the suture line.

Dissection requires focal disruption of the intima by the circulating blood into the media where it tracks down between the vessel layers. The most frequent aetiology of this entity is cystic mucoid degeneration of the media, although it is also frequently associated with atherosclerosis andx the congenital anomalies of elastic tissue, such as the Ehlers Danlos syndrome or Marfan's syndrome.

Complications

The discussion which follows pertains to any aneurysmal process, but special consideration will be given to the entity of dissecting aneurysm which has certain specific complications.

Most aneurysms remain totally asymptomatic and only declare themselves once complications develop. These manifestations may be of gradual onset, and steadily progressive or they may be extremely dramatic in their onset.

Expansion with Compression

As previously discussed, expansion of the aneurysm is a progressive phenomenon and all aneurysms will continue to dilate at a variably increasing rate, and cause symptoms by compression of surrounding anatomical structures. Nerve and nerve roots are characteristically compressed by posterior expansion of an atherosclerotic aortic aneurysm leading to referred pain in the lumbosacral distribution. Other frequent manifestations include compression of the posterior tibial nerve related to popliteal aneurysms and various cranial nerves as enlarging aneurysms on the Circle of Willis progressively impinge upon them. Venous occlusion is characteristically seen in association with popliteal aneurysms in 75% or more of these patients. Iliac vein occlusion is infrequently associated with abdomino-iliac aneurysms. Other characteristic symptomatology may result from compression of the ureter or duodenum by abdominal aortic aneurysms. Much less frequently bony erosion is noted, in particular with long-standing aortic aneurysms. The symptomatology produced obviously depends upon the anatomical location of the aneurysm and the structures compressed.

Rupture

Probably all aneurysms would eventually rupture if observed for long enough. Rupture may be free with haemorrhage or it may be contained. Probably the best documented of all aneurysmal processes is that of atherosclerotic aneurysm of the infrarenal aorta and in various series between 10 and 30% of these patients present with this complication. In contrast, of arteriosclerotic popliteal aneurysms presenting for treatment, only about 3% have ruptured. There is little objective data pertaining to predictive factors with regard to the likelihood of rupture and what there is once again refers to infrarenal atherosclerotic abdominal aortic aneurysms. The single most important factor is size as measured by the widest diameter. The risk of rupture within a five-year period of an aneurysm of less than 4 cm in diameter is 10-15%.

In contrast, once it reaches a diameter of 8 cm, the risk of rupture exceeds 75%. There is a sharp increase in the risk of rupture once a diameter of 6 cm is attained. Infrarenal aortic aneurysms may rupture freely into the peritoneal cavity or leak into the retroperitoneal space and be tamponaded by the overlying peritoneum. Rarely rupture may occur into the surrounding viscera such as the duodenum resulting in gastrointestinal haemorrhage or the inferior vena cava causing acute lower extremity venous hypertension with oedema, a loud abdominal arteriovenous bruit and in most cases, acute congestive cardiac failure. In general, the symptoms and signs of a ruptured or leaking aneurysm depend on its site. Classically this is a dramatic, if not a catastrophic event with signs of acute haemorrhagic shock. Locally there is almost invariably a large tender pulsatile mass.

Thrombo-Embolism

The passage of circulating blood from a normal calibre artery into the dilated aneurysm sac results in the phenomenon of a central jet stream through the aneurysm with much slower and turbulent flow around the aneurysm wall. It follows that the wider the aneurysm, the more sluggish is the flow at its periphery. This sluggish flow results in thrombus formation which lines the wall of the aneurysm for a varying depth, which in turn may dislodge and result in peripheral embolisation in the arterial tree distal to the aneurysm. This is a relatively uncommon complication in aortic aneurysms. It is far commoner with peripheral aneurysms, particularly popliteal aneurysms in which 20-30% show evidence of distal embolisation at the time of diagnosis. In addition, in our experience, aneurysms related to the aortic arch branches frequently manifest themselves with cerebral or upper limb embolisation.

Complete thrombosis with occlusion of the aneurysm occurs uncommonly but we have seen this on occasion with infrarenal aortic, iliac and especially popliteal aneurysms. This phenomenon is always associated with an element of pre-existing outflow stenosis with compromised distal runoff.

Infection

Secondary infection of thrombus in a pre-existing aneurysm is surprisingly rare. In many patients this may be asymptomatic and in our own study, in which routine cultures were taken from aneurysm clot at the time of surgery, of a total of 34 patients, 10 (29%) had positive cultures. Organisms encountered included *Bacillus sp, Klebsiella sp* and *Staphylococcus epidermidis.*

None of these patients developed wound or graft infection over a two-year follow-up period. It must be emphasized, however, that *Staphylococcus epidermidis* has been implicated in low-grade prosthetic infections. The organism is a skin commensal and not usually a pathogen, but once it becomes established in prosthetic material it is particularly resistant to antibiotic therapy by virtue of its ability to secrete a protective mucoid coat ("slime factor").

When overt invasive infection of aneurysmal clot occurs, it probably follows bacteraemia due to invasive procedures, such as arterial cannulation or urethral catheterization. Some organisms such as *Salmonella sp* have a predilection for arterial walls and the entity of primary infective aneurysms due to salmonella infections is well described. *Staphylococcus*

aureus has been reported as the most frequent gram-positive infecting agent under these circumstances.

Most false aneurysms following penetrating trauma will yield positive cultures, mainly due to commensal skin organisms. However, the sepsis rate following surgical repair is surprisingly low.

Complications of Dissecting Aneurysms

Dissecting aneurysms are most frequently encountered in the aorta and the aetiology has been discussed. De Bakey has described three pathological subgroups. The dissection invariably produces an expansile mass lined by a varying thickness of media and adventitia which may eventually rupture into the pleural cavity, mediastinum, pericardium, retroperitoneal space or peritoneal cavity. Presumably the pathological principle relating to expansion and rupture are similar to those which dictate expansion of other aneurysms. Unfortunately, objective data is sparse in this regard. Under certain circumstances the aneurysm may re-enter the true lumen by rupture through the intima.

More specific complications relate to proximal and distal progression of the dissection process. Retrograde dissection may result in aortic valve incompetence and/or occlusion of the coronary arteries. Distal dissection may occlude the ostia of major branches or progression of the dissection along these branches may lead to compromise of the lument by compression caused by the intramural haematoma. We have encountered acute presentations due to cerebral ischaemia, mesenteric infarction, anuria due to renal artery occlusion, and acute lower extremity ischaemia due to occlusion of the ileofemoral arterial segment.

The complications of dissecting aneurysms therefore result in varied and dramatic presentations and this should always enter into the differential diagnosis in any patient presenting with acute chest or abdominal pain or ischaemia of the upper or lower extremities. This is particularly important if the patient is significantly hypertensive or shows evidence of a deficiency in elastic tissue such as hyperextensible joints or the typical bodily habitus associated with Marfan's syndrome. Further evidence for this diagnosis is provided by the presence of a widened superior mediastinum or thoracic aortic shadow on chest radiography. Diagnosis is confirmed on CT or NMR scanning followed by pan aortography.

In summary, aneurysmal disease is one of multiple aetiologies and protean pathological and clinical manifestations. The complications are life, organ and limb threatening. The true prevalence of various aneurysms and incidence of the complications described are difficult to ascertain in view of the fact that most aneurysms declare themselves with evidence of these complications. It is probably a valid statement that, in general, aneurysms of any type require surgical treatment at the time of diagnosis, even if asymptomatic, and that good reason should be found for adopting a conservative approach.

Comment

Aneurysms and Their Complications

L J Levine

Rupture of thoracic and abdominal aneurysms continue to be responsible for about 2% of all deaths in a Western society. The results of emergency repair of a ruptured aneurysm have improved little over the past 15 years, whereas the results of elective repair, particularly if coronary artery disease is correctly treated, has fallen to under 2% mortality. On this basis most major vascular surgical centres have advocated the elective repair of abdominal or iliac aneurysms even under 5 cm in size, provided the patient has a reasonable life expectancy.

Abdominal aneurysms grow at about 4 mm per year on average, but it appears as if this growth rate is not constant, but rather occurs in fairly short bursts of growth. Symptoms of abdominal and back pain, as well as possible rupture, may be related to the periods of rapid growth.

Thoraco-abdominal aneurysms, previously regarded by many as inoperable, can now be corrected with an overall mortality of 10% and a morbidity of 7-10%.

Femoral and popliteal aneurysms may occasionally rupture, but far more commonly present with distal embolic phenomena, presumably because the sites of these aneurysms are far more mobile than the aorta. Particularly the popliteal aneurysm has a tendency to destroy the distal circulation, presumably by repeated emboli. By the time the patient with a popliteal aneurysm presents witgh a threatened leg because of the absence of a distally reconstructible vessel, the limb loss rate is high. For this reason, large popliteal aneurysms, particularly if they are saccular or contain significant quantities of clot, should be repaired electively.

Chapter 11.4: Compartment Syndromes

J A Shipley

Introduction

In 1900 Littlewood drew attention to the soft-tissue "effusion" developing in a traumatized limb and suggested that the pressure generated by the constricting deep fascia could obstruct the circulation in the limb. He advocated fasciotomy to relieve this pressure. Despite this early appreciation of the pathogenesis and treatment of compartment syndromes, and despite the use of fasciotomy after surgery for vascular injuries during World War II, only in the last decade has the concept of acute compartment syndromes gained wide recognition. The pendulum has now swung to the other extreme. The uncritical use of compartmental pressure measurements would appear to pose a real danger of overdiagnosis and overtreatment of the condition.

Definition

A compartment syndrome is a circulatory disturbance caused by a rise in the interstitial pressure in a closed osteofascial compartment to the point where tissue perfusion is reduced. There is resulting loss of function and ultimately ischaemic necrosis of the muscles and nerves enclosed by the compartment, unless the compartmental pressure is relieved by fasciotomy.

Pathogenesis

Table 11.4.1. Causes of Compartment Syndromes

Reduced Compartment Size

--> Tightly sutured fascia
--> Circumferential burns
--> Constrictive dressings

Increased Size of Compartment Contents

--> Oedema
- Trauma - especially diffuse, high-energy injuries
- Crush injuries
- High-velocity gunshot wounds
- Burns and frost-bite
- Acute muscle over-exertion
- Deep venous thrombosis

--> Haemorrhage
- Trauma
- Surgery, ie, osteotomy
- Haemophilia and other bleeding tendencies
- Anticoagulant treatment

--> Postischaemic swelling
- Restoration of circulation after acute arterial obstruction
- Prolonged limb compression, ie, unconscious patient
- Prolonged tourniquet application

--> Extravasation of IV infusions.

The syndrome is precipitated by interstitial swelling secondary to a variety of causes (table 11.4.1), the commonest of which are trauma and acute arterial obstruction. When swelling occurs in a closed anatomical compartment it inevitably leads to an increase in interstitial pressure which in turn causes partial or complete collapse of the venules and capillaries. To a variable degree arteriolar vasodilatation can reverse this collapse by increasing the capillary perfusion pressure, and the haemodynamic abnormality may stabilize. However, if the interstitial pressure continues to rise due to continued haemorrhage, or

increased capillary permeability and accumulation of oedema fluid, this compensatory mechanism may fail, the microcirculation will collapse and tissue perfusion will cease. As diastolic blood pressure will exceed the capillary closing pressure by up to 30 mm Hg, there will be no interruption of flow in major arteries. Normal pulses and perfusion will continue beyond the limits of the compartment.

Because patients vary in their ability to tolerate increased interstitial pressures, no critical pressure can be identified which is diagnostic of a compartment syndrome. Factors which influence the patient's susceptibility to increased compartment pressures are listed in table 11.4.2. The most important is hypotension which, by limiting the capillary perfusion pressure, allows the onset of ischaemia at relatively low interstitial pressures.

Table 11.4.2. Factors Predisposing to Compartment Syndrome

Reduced Perfusion Pressure

--> Hypotension
- Systemic, ie, hypovolaemia
- Local, ie, arterial injury, vascular disease

--> Elevation of limb

Tendency to Oedema

--> Reduced plasma osmotic pressure, ie, crystalloid overload
--> Increased venous pressure, ie, venous obstruction

Traction

Intact Fascial Envelope

Compartment syndromes may develop in two ways following arterial occlusion. It should be noted that complete arterial occlusion will result in gangrene rather than in a compartment syndrome. However, if the obstruction is incomplete, or a collateral circulation exists which is sufficient to fill and pressurize the microcirculation without relieving anoxia, the capillary leak which results will cause swelling and raise the pressure in the compartment. Restoration of arterial flow after complete obstruction may also cause a compartment syndrome (the revascularization syndrome). The vessels are maximally dilated, with high perfusion pressures; the capillaries are excessively permeable, and interstitial swelling occurs rapidly. In both of these situations the primary problem is arterial is arterial insufficiency, but failure to anticipate or treat a complicating compartment syndrome will perpetuate the state of ischaemia in that compartment despite arterial reconstruction.

Occasionally a reduction in the size of a compartment may contribute to the increased pressure. This occurs most frequently when the deep fascia is sutured tightly following an operation, or following circumferential burns of a limb with contraction of the eschar.

It must be emphasized that the fascia is the main constricting element in most cases of compartment syndrome. Mubarak has shown that little reduction of intracompartmental

pressure is gained by releasing the skin or epimysium, while a dramatic drop in pressure is achieved by fasciotomy. A patient with a severely lacerated limb is to a degree protected against a compartment syndrome by loss of continuity of the fascial envelope, but this is seldom complete, and it may not extend to all compartments at risk.

Pathology

When the nerves and muscles enclosed by the compartment become hypoxic, their function is impaired. As sensory nerve fibres are more sensitive to hypoxia than muscle, sensory symptoms and pain develop early in the course of the condition. Motor fibres are more resistant and paralysis of muscles supplied by the compartmental nerves is a later phenomenon. Active contraction of the compartmental muscles is lost relatively late, and muscle necrosis follows soon afterwards. Muscle necrosis becomes evident after six hours of total ischaemia, and the majority of muscle fibres are necrotic after eight hours.

Necrotic muscle is repaired by fibrous tissue, which causes contractures and limited excursion of the muscle-tendon units. The final result is the classical picture of Volkmann's ischaemic contracture.

As skeletal muscle does have a limited potential for regeneration, and some muscle fibres may survive in the fibrosed muscle bellies, a return of function is possible. Unless the compartmental nerves are completely necrotic, they have a rather better capacity for regeneration, and as the axon sheaths often survive intact, reinnervation of the axon's original end organ may occur.

Matsen noted that compartmenty syndromes treated in less than 12 hours from onset of symptoms had a 70% chance of complete recovery, while this figure dropped to 10% if more than 12 hours had elapsed before treatment started. Time is not the only factor in the prognosis; the pressure generated in the compartment must also be considered. High pressure for a relatively short time may cause severe permanent loss of function, while a low pressure persisting for a long time may also cause tissue necrosis.

Surgical Anatomy

Although compartment syndromes have been described in virtually every muscle group, the commonest sites are the forearm and leg.

The Forearm

The forearm has three fascial compartments:

1. Superficial flexor compartment containing:

Muscles

- Flexor digitorum sublimis
- Flexor carpid radialis
- Flexor carpi ulnaris
- Pronator teres

Nerves

- Radial
- Median
- Ulnar

2. Deep flexor compartment containing:

Muscles

- Flexor digitorum profundus
- Flexor pollicis longus
- Pronator quadratus

Nerves

- Anterior
- Interosseous

3. Extensor compartment.

The deep flexor compartment is the most prone to develop increased intracompartmental pressure. Rorabeck maintains that the extensor compartment seldom requires decompression, which should be performed only if pressure remains elevated after both flexor compartments have been released.

Although flexor compartment fasciotomies through an ulnar approach are popular, decompression of both compartments through the anterior Henry approach is familiar to most surgeons as the standard anterior approach to the radius, and it can be extended proximally and distally as circumstances dictate. It should always extend from the cubital fossa, with

division of the lacertus fibrosus, and include a carpal tunnel release at the wrist. The figure shows the recommended skin incisions which will cause the least skin morbidity.

The extensor compartment can be released through a mid-dorsal incision which can be extended into the hand.

The Leg

Although the anterior and deep posterior compartments are most commonly affected by compartment syndrome, the general experience is that all four compartments are usually simultaneously involved to a greater or lesser degree. For this reason, four compartment decompression is usually performed as a routine if fasciotomy of one compartment is needed.

Four-compartment decompression through the single incision parafibular approach is popular and effective. Care is essential to avoid the peroneal nerve in the proximal part of the incision. Where a limb is mutilated and the anatomy distorted, or in a severely swollen limb where the skin is very tight and may itself cause constriction, it may be safer to perform a double-incision fasciotomy. Here, the posterior compartments are decompressed through an incision posterior to the medial border of the tibia, and the anterior and peroneal compartments through an antero-lateral incision.

Fibulectomy is unnecessary and may lead to ankle instability.

Clinical

Compartment syndrome can usually be diagnosed on clinical grounds alone. An aggrressive diagnostic approach is necessary to identify patients at an early stage when the pathological changes are still reversible, and specific attention should be given to patients at high risk.

The clinical features are:

--> Pain over the affected compartment. It is typically severe, unremitting, and out of proportion to the underlying injury.

--> Paraesthesiae and sensory loss in the sensory distribution of nerves traversing the compartment.

--> Pain on stretching muscles in the affected compartment is intense.

--> Paralysis of the muscles in the affected compartment occurs late, and is a poor prognostic sign.

--> Palpation of the compartment shows it is tense and tender.

--> Discoloration and blistering of the skin overlying the compartment are late phenomena.

It must be emphasized that absence of distal pulses is a sign of arterial injury, not a compartment syndrome, where there is usually no evidence of ischaemia of the skin or structures distal to the compartment.

The four compartments of the leg are:

1. Anterior compartments containing:

Muscles

- Tibialis anterior
- Extensor hallucis longus
- Extensor digitorum longus

Nerve

- Deep peroneal

2. Peroneal compartment containing:

Muscles

- Peroneus longus
- Peroneus brevis

Nerves

- Superficial
- Peroneal

3. Superficial posterior compartment containing:

Muscles

- Triceps surae

Nerve

- Sural

4. Deep posterior compartment containing:

Muscles

- Tibialis posterior
- Flexor digitorum longus
- Flexor hallucis longus

Nerve

- Tibial.

Diagnosis

Compartment syndrome must be differentiated from:

--> Acute arterial occlusion where distal pulses are reduced or absent, and the skin and distal structures are ischaemic.

--> Nerve injury where pain is not a feature, sensory and motor loss are restricted to the anatomical distribution of that nerve, and passive stretching of paralysed muscle is painless.

--> Reflex sympathetic dystrophy. This form of nerve injury may present within a few days of an insult, and can be confused with an acute compartment syndrome. Pain can be severe and unremitting in this condition, and paraesthesia, pallor of the distal parts, swelling and hyperaemia may occur. Pain in the fingers can cause the unwary to interpret a stretch test positive. The compartments concerned will, however, remain non-tender and soft on palpation.

Obviously combined injuries may occur, especially in a badly injured limb.

Special Investigations

Much of the recent literature on compartment syndromes has been preoccupied with the measurement of intracompartmental pressures and the practical significance of these pressures. The present feeling is that pressure measurements are a useful diagnostic aid, but that they cannot be considered in isolation from the clinical findings. In most patients, a clinical decision on the need for decompression will be possible, and this can be confirmed by measuring the intracompartmental pressures, although this is not essential. However, in the following situations pressure measurements are much more important and may provide the only objective information on which decision can be made:

--> **The uncooperative or unresponsive patient.** In head injuries, drug overdosage, children and multiple trauma cases, clinical evaluation is often confusing or impossible, and pressure measurements in limbs at risk of a compartment syndrome are essential.

--> **The borderline case.** Where the clinical signs are equivocal, pressure measurements may help. A single measurement may be difficult to interpret, but continuous or repeated measurements showing sustained or rising pressures may indicate the need for compartment decompression.

--> **Diagnosis of nerve lesions.** In polytrauma cases, differentiation of a peripheral nerve lesion from a compartment syndrome may be difficult. Compartment pressure measurements may clarify the picture.

--> **Burns,** where clinical findings may be impossible to interpret due to pain from the injury.

Intra-operative monitoring has also been used to confirm that adequate decompression of all muscles and compartments at risk has been obtained.

Normal intracompartmental pressure is 4 ± 4 mm Hg. Much of the confusion surrounding intracompartmental pressure measurement arises from the widely differing pressures regarded as significantly elevated by different authors. Mubarak and Rorabek regard pressures of 30 mm Hg or above as an indication for decompression, while Matsen accepts pressures up to 45 mm Hg. Considering the multiple factors other than absolute pressures that determine the onset of compartment ischaemia, Whiteside has the most logical approach. He regards a compartmental pressure of 25-30 mm Hg below the diastolic blood pressure as an indication for decompression. Repeated or continuous pressure monitoring is becoming increasingly popular in borderline situations, or compartments at high risk of increased pressure. The usual method is to measure the pressures of all compartments at risk and continue to monitor the compartment with the highest pressure. Rising or persistently high pressures would suggest the need for fasciotomy.

To obtain a more direct assessment of tissue function, investigators have used nerve conduction studies, mostly on an experimental basis, and have found that they correlate well with the developing ischaemic nerve deficit. Although most compartments have a motor or mixed nerve passing through them that can be evaluated, the technique is specialized and not readily available in an emergency. Its practical value is therefore limited. It may be of diagnostic help in differentiating a nerve injury from a compartment syndrome.

Technique of Measuring Intracompartmental Pressures

The technique used by Whitesides is described because it does not require specialized equipment. An 18 gauge needle is connected via an extension line to a three-way stop-cock and a 20 mL syringe. Sterile saline is drawn up into the needle, and partway along the extension line, ensuring that no bubbles are present in the saline column. After cleaning the skin, the needle is pushed into the compartment to be evaluated. With the syringe full of air, the stop-cock is connected to a manometer, and with the tap of the stop-cock turned to connect the syringe, needle and manometer, the plunger of the syringe is slowly depressed. A pressure is gradually developed in the system, which can be read on the manometer. At the point where the pressure in the system equals that in the compartment, saline will start to flow along the extension line. The manometer reading at this moment will indicate the intracompartmental pressure. Two readings should be made at different sites, and both readings should agree; if not, a third reading is necessary. The common mistake is to depress the syringe plunger too rapidly, which gives an artificially high reading. The technique is accurate to plus minus 3 mm Hg. More sophisticated arrangements involve the use of:

--> **Wick catheters or slit catheters,** which are less likely to be blocked by blood clot or soft tissue than a needle, and which can be left in sity for pressure monitoring.

--> **Low-volume infusion systems** delivering 0.1 mL heparinized saline per hour. This allows continuous monitoring without introducing excessive saline into the compartment and artificially raising the pressure. Rorabeck maintains that a slit catheter will provide continuous accurate monitoring without the need for flushing or continuous infusion.

--> **Transducers** for more accurate and convenient pressure measurements instead of a manometer.

The basic principles of all these systems are the same.

Treatment

The most important emergency measures to be taken when a compartment syndrome is suspected or diagnosed, are to correct any tendency to hypotension, and to reduce a displaced fracture.

It is traditional to remove any potentially constricting dressing or plaster cast. This is of dubious value unless they actually do compress the limb, but provided any fractures are adequately immobilized, it can cause no harm. It may be beneficial to reduce the amount of traction used for a fracture.

No other treatment such as sympathetic block, steroids, or cooling is of proven value. Elevation of a limb has been shown to reduce arterial pressure and may prejudice the perfusion of the compartment, so at present it appears to be contraindicated. Hyaluronidase has been used experimentally to reduce intra-compartmental pressures, but no clinical reports are available.

The definitive treatment of a compartment syndrome is fasciotomy. The skin and deep fascia should be divided over the whole length of the compartment: there is no place for "blind" fasciotomy through a limited skin incision. Each muscle should be palpated to assess its tension (or its tissue-pressure measured) and if necessary the epimysium divided. Similarly, nerves in the compartment should be checked for areas of constriction by musculo-fascial bands, and these should be released.

Debridement of muscle at the initial operation should be extremely conservative: a more accurate assessment of muscle viability can be made at the routine wound inspection after 24 to 48 hours. Even then only obviously necrotic tissue should be removed, bearing in mind the possibility of muscle regeneration from residual viable fibres.

Fasciotomy usually leads to gross instability of any underlying fractures. It is essential to reduce and fix these fractures by internal or external fixation according to the nature of the injury. Although plaster casts may be used, they are very inconvenient for wound management and repeated debridements.

Attempts at skin closure are usually most unwise as further swelling is to be anticipated following relief of the ischaemia. The wounds should be covered with sterile dressings, and the limb re-examined under anaesthetic at 24 hours, and subsequently as deemed necessary.

Secondary skin closure can be attempted as swelling recedes, and any residual skin defect closed by skin-graft.

Splintage of paralysed muscles, and early active and passive mobilization of the limb are essential to prevent deformity and contractures, and may encourage muscle regeneration.

Prophylactic fasciotomy should be considered during surgery for:

--> arterial occlusion of more than four hours duration

--> when severe swelling has occurred or is expected in association with a fractgure needing internal fixation.

Complications

--> Infection usually occurs as a result of extensive muscle necrosis. Treatment is by aggressive debridement of infected and devitalized tissue, and administration of appropriate antibiotics. In this situation amputation is often necessary.

--> Hyperkalaemia and myoglobinuria occur to a varying degree after revascularization of ischaemic muscle. ECG and electrolyte monitoring may be necessary to avoid cardiac arrhythmias, and a high urine output and alkalinization of urine may help to prevent acute renal failure. Uncontrollable release of potassium and myoglobin is an indication for amputation.

--> Volkmann's ischaemic contracture

--> Posttraumatic ossification.

Prognosis

The earlier a compartment syndrome is diagnosed and treated, the better the prognosis. If less than 12 hours have elapsed between onset of symptoms and fasciotomy, the majority of patients will recover completely. A delay of more than 12 hours will leave the majority of patients with permanent disability. If a complete motor deficit has developed, the prognosis for complete recovery is very poor.

Comment

Compartment Syndrome

M Mars

Most of what the author has said encapsulates our own understanding, ideas and philosophy of the development and management of the compartment syndrome, the aim of management being to prevent the end sequelae of loss of nerve and muscle function by early identification, careful observation and prompt management of those patients who are at risk of having an increased intracompartmental pressure (table 11.4.2). While the use of intracompartmental pressure measurement may lead to the risk of overtreatment, it is our feeling that it is better to do one fasciotomy too many than one too few.

To put the incidence of treatment of acute compartment syndrome into perspective. Vascular surgical procedures now account for the majority of fasciotomies at our hospital, with 2.6% of all vascular admissions (± 15 patients per year) undergoing fasciotomy, as opposed to 0.75% of all ICU admissions (± 6 patients per year) and 0.1% of all orthopaedic

admissions (± 6 patients per year). The fasciotomy rate for vascular patients rises to 11% in cases of vascular trauma and acute arterial occlusion.

Constrictive dressings are mentioned in table 11.4.1 as being a cause of reduced compartment size. I feel they deserve further mention under the pathogenesis of compartment syndrome. Historically they are the most common cause of the compartment syndrome. Volkmann described the effects of the syndrome in 1872 and in 1881 attributed it to "bandages applied too tightly". Constrictive dressings influence the development of the compartment syndrome because they prohibit increases compartment sizes (a normal homeostatic mechanism), and they are a primary cause of raised intracompartmental pressure. This is based on the following observations:

If a tourniquet is applied to a limb, and the limb is then bandaged, we have shown that upon release of the tourniquet there is an immediate sustained rise in both the intracompartmental pressure and the pressure generated by the bandage on the limb, indicating that as the compartments fill with blood the intracompartmental pressure increases and the limb begins to swell. The same procedure repeated on unbandaged limbs resulted in a transient rise in intracompartmental pressure which returned to normal. Secondly, the average pressure generated by routine postoperative bandaging following hand surgery we found tobe about 23 mm Hg. We have confirmed Matsen's finding that the pressure generated on the skin by external compression is transmitted directly to the compartments and that the resultant intracompartmental pressure is the sum of the preexisting compartment pressure and the applied bandaging pressure. It is important to note, however, that if the applied bandaging pressure remains below 20-25 mm Hg, the homeostatic mechanisms for reducing intracompartmental pressure appear to be facilitated, and that at higher pressures they become inhibited and intracompartmental pressure rises still further. Garfin et al have shown in an animal model of raised intracompartmental pressure, that splitting and spreading of a plaster cast, followed by dividing the underlying orthopaedic wool, reduced the intracompartmental pressure by 50%. This work quantifies the standard orthopaedic practice of splitting the plaster of Paris if the patient complains of pain out of proportion to the injury after fracture reduction or if routine "circulation check" demonstrates any of the signs of raised intracompartmental pressure.

The pathogenesis of the revascularization syndrome, is now being attributed to the generation of superoxides, xanthine dehydrogenase being converted to xanthine oxidase during the ischaemic phase, and with revascularization and the reavailability of oxygen, xanthine oxidase converts xanthine in the presence of oxygen to uric acid and free oxygen radicals which attack the vascular epithelial cell membranes resulting in their increased permeability.

Finally, under pathogenesis snakes bites were the commonest reason for fasciotomy in our ICU and a contributing factor to the pathogenesis was the frequent use of a tourniquet prior to the patient reaching the hospital, adding the problem of revascularization syndrome to tahty of envenomation.

Elevation of the limb is mentioned in table 11.4.2 as a cause of compartment syndrome and again in the section on treatment which, while accepting that it is contraindicated, the sentiment is expressed that there may be some doubt over this. There is ample evidence using many different techniques to show that limb elevation diminishes the

arterial pressure in the limb, and that any factor that reduces the arterio-venous pressure gradient will reduce blood flow. Limb elevation causes in effect local hypotension and in the presence of a compartment syndrome any factor that diminishes limb blood flow is contraindicated.

The sections on pathology, surgical anatomy and clinical features summarizes these areas well. It should be re-emphasized that the often sought sign of capillary filling is not a sign of adequate circulation.

The use of intracompartmental pressure measurement as an indicator of when to do a fasciotomy has not been fully resolved as is shown by the "experts" advocating decompression over a wide range of pressures. A reason for this is that a high pressure for a short time is as dangerous as a prolonged increase of pressure at a lower pressure. It must be stressed that the diagnosis should be made on clinical grounds and that intracompartmental pressure measurement may assist in the borderline case, and in the unconscious, un-cooperative or burnt patient. It is my feeling that with repeated use of intracompartmental pressure monitoring, the user will develop a clinical feel for the interpretation of these values based on the observed relationship of these pressures and the condition of muscles seen at fasciotomy.

The management strategy is, as the author has stated, to remove any factor that may diminish limb blood flow and if this does not improve the situation, proceed to fasciotomy. It must be remembered that a rise in intracompartmental pressure is a dynamic situation in which the normal homeostatic mechanisms for reduction of compartment mechanisms for reduction of compartment pressure are being overcome by the factors raising the pressure.

Management of the raised intracompartmental pressure should consist then of correction of hypotension, reduction of fractures, release of any constricting dressings and preventing any elevation or dependency of the affected limb. Having done this, if a fasciotomy is still indicated clinically, it should be performed. However, if these measures improve the clinical picture, the patient should be reassessed regularly at short intervals and intracompartmental pressure monitoring may be useful as a trend indicator.

Our policy on fasciotomy is similar, there being no place for "blind" fasciotomy or "limited" fasciotomy.

While increasing awareness of the condition, its diagnosis and management has resulted in fewer cases of the end sequelae of the syndrome being reported in the forearm and leg, almost every muscle group is potentially at risk, and there are more cases of chronic compartment syndrome being reported in the hand.

The author's message on compartment syndrome needs to be repeated:

An aggressive diagnostic approach is necessary to identify patients at an early stage, while the pathological changes are still reversible, and specific attention should be given to the patients at high risk.