Arterial injuries and their management

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TRAUMA to a major artery is a disaster which may forthwith imperil the life of the patient or the viability of a limb. In the Second World War and the early phases of the Korean War ligation for major arterial injuries of the lower limb was associated with an amputation rate of 60-70%, but following the institution of a policy of performing arterial repair whenever possible this was reduced to 13% (DeBakey & Simeone, 1946; Jahnke & Howard, 1953; Hughes, 1958). Despite the increasing incidence and complexity of high velocity wounds in Vietnam the overall amputation rate for 500 cases of arterial injury remained initially at 12.7% (Rich & Hughes, 1969). Subsequent series have, however, reported amputation rates of 8% comparing favourably with those of 5% or so occurring in series of civilian arterial injuries of similar size (Morris *et al.*, 1960; Ferguson, Byrd & McAfee, 1961; Patman, Poulos & Shires, 1964; Cohen, Baldwin & Grant, 1969; Drapanas *et al.*, 1970; Feltis, 1970). Thus, it is evident that satisfactory results are feasible in a high proportion of cases but because early diagnosis and expeditious management are essential prerequisites to a successful outcome, arterial injuries now merit an importance out of all proportion to their frequency for the sooner effective treatment is instituted the greater the probability of a satisfactory result. However, as they are relatively infrequent in civilian practice, complicating less than 4% of skeletal trauma, individual surgical experience tends to be limited and arterial injuries have acquired an unnecessarily gloomy reputation both with regard to their management and results (Koskinen, 1962; Connolly, 1970). In fact, the majority of disappointing results are usually attributable to delays in diagnosis compounded by protracted, poor, and sometimes perfunctory, management.

Major arterial trauma may be caused by:

1. Deceleration injuries.
2. Penetrating wounds—clean
 - lacerated
 - missile.
3. Crushing injuries.
4. Fractures or dislocations.

5. Therapeutic and diagnostic techniques
 - angiography
 - intra-arterial injections
 - cardiac catheterization.

In recent years there has been a notable increase in deceleration and iatrogenic injuries (Morton, Southgate & De Weese, 1966). Several types of injury may occur and sometimes more than one mechanism may be operative. In the main, however, these fall into the following categories:

1. Complete arterial interruption due either to transection or actual tissue loss.
2. Contusion producing traumatic arterial thrombosis.
3. Partial division or laceration with subsequent formation of a false aneurysm.
4. Injury to neighbouring artery and vein with the formation of an arterio-venous fistula.

The contentious subject of traumatic arterial spasm merits special consideration and will be discussed later.

In the past several years we have dealt with ninety-seven civilian arterial injuries and the various types and aetiological factors involved are summarized in Table 1. Detailed descriptions of many of these have been reported previously (Slaney, Ashton & Abrams, 1966; Ashton & Slaney, 1970).

Deceleration injuries

These are usually caused by a combination of going too fast in the first place and stopping too quickly in the second for the stresses and strains produced in this situation may be very great indeed. It has been shown experimentally that acute deceleration from 120 miles/hr down to 0 miles/hr in 0.2 sec or so can produce rises in pressure of the order of a 1000 mmHg in the arch of the aorta (Cammack *et al.*, 1959). It is also of great importance to appreciate that complete transection of a major vessel may occur in a deceleration injury without any external evidence of violence whatsoever and certainly without any accompanying bony injury.

A classical example of a deceleration injury is that of traumatic rupture of the aorta, which though

TABLE 1.

	Total	Industrial accidents	Road accidents	Iatrogenic injury	Other causes
Traumatic arterial thrombosis	30	6	7	14	3
False aneurysm	15	—	4	7	4
Arterial transection sharp	12	6	—	4	2
Arterial transection blunt	12	2	8	2	—
Arterio-venous fistula	12	4	1	—	7
Rupture of aorta	8	—	7	—	1
Shot-gun wounds	3	—	—	—	3
Miscellaneous	5	—	1	4	—
Total	97	18	28	31	20

it may involve any segment of this vessel, usually occurs immediately distal to the origin of the left subclavian artery. Two varieties of lesion are seen; either a complete transection of the aorta, or a partial tear producing a localized haematoma which may rupture in hours or days or organize to form an aneurysmal sac, this being known as a traumatic aneurysm (Fig. 1). Traumatic rupture of the aorta usually complicates severe deceleration, as in aircraft or high-speed motor vehicle accidents, but it can occur following deceleration at speeds as low as 30 miles/hr or less (Rice & Wittstruck, 1951). Moreover, it may be accompanied by severe injuries to the heart, chest, abdomen, limbs or skull, which may be incompatible with survival but in a third of the patients may be the only injury present (Parmley *et al.*, 1958). Though instantaneous death may ensue, approximately 20% of patients survive to reach hospital alive and of these the majority survive for periods varying between 12 hr and 3 weeks after admission before final dissolution of the periaortic haematoma occurs (Slaney *et al.*, 1966). In fact, of those patients who reach hospital alive about half

will survive for a further 7–10 days before final dissolution occurs, so that if the diagnosis is borne in mind there is usually ample time to institute effective treatment. A few patients, usually those with a localized tear and the formation of a traumatic aneurysm, may survive for several years after the original accident before final rupture occurs. The most important single point in making the diagnosis is to suspect that aortic rupture may have occurred in any deceleration injury for there may be no external evidence of injury whatsoever. A chest X-ray is mandatory in every case, usually demonstrating widening of the superior mediastinum, or the presence of a hilar mass and if these are seen traumatic rupture of the aorta should be suspected especially when there is no evidence of injury to the sternum or the thoracic cage (Fig. 2). If there are accompanying signs such as an impaired blood flow to the left arm manifested clinically by a diminished or absent radial pulse and a difference in blood pressure between the recording in the left upper arm and that in the right, or evidence of ischaemia in the legs then the diagnosis becomes almost certain. In a few patients there may be signs of acute ischaemia to the spinal cord and this may lead to a misdiagnosis of traumatic paraplegia. The diagnosis can usually be confirmed by an aortogram and effective management instituted (Fig. 3). The management of the aortic injury should obviously be accorded a high priority but one must take into account the presence of associated injuries. An ECG should always be performed to exclude an associated contusion of the heart with a tracing resembling that of a myocardial infarct. If there is evidence that this has, in fact, occurred it is wise to try and defer operation for a few days rather than proceed to immediate surgery. Whenever other extensive injuries such as a flail chest, airway obstruction, intra-abdominal haemorrhage, multiple fractures and so forth are present these must all be taken into account when deciding

FIG. 1. The common site at which traumatic rupture of the aorta occurs. (a) Complete rupture; (b) incomplete rupture, 'traumatic aneurysm'.

FIG. 2. Mediastinal haematoma extending to the apex of the left lung following complete rupture of the aorta immediately distal to the left subclavian artery.

FIG. 3. Traumatic rupture of the aorta showing extravasation of dye below the aortic arch.

on priorities in management. Repair of the aortic injury usually requires simple left heart by-pass and replacement of the damaged segment by an appropriate length of Dacron or Teflon synthetic graft (Fig. 4). An increasing number of successful instances of repair of this type of injury are now appearing in the world literature and because of this it is well worthwhile bearing the diagnosis in mind.

A few fortunate patients may sustain a localized tear in the aortic arch with the formation of a false aneurysm and this may not present clinically until several years following the accident. Fig. 5 shows the chest X-ray of a young man who 7 years previously had been injured in a car crash as a result of which he had been unconscious for 1 week. During this time he had had an emergency splenectomy for traumatic rupture of the spleen and had been noticed to have a small haemothorax. A subsequent aortogram confirmed the diagnosis (Fig. 6); at operation the aneurysm was found to be lying in the classical position in the concavity of the aortic arch and it proved possible to excise it and repair the defect with a Dacron patch (Fig. 7). He remains well and is living a normal life 4 years postoperatively.

Although traumatic rupture of the aorta is perhaps one of the more dramatic examples of decelera-

FIG. 4. Resection of the left thoracic aorta with atrio-femoral by-pass.

tion injury it is important to remember that viscera such as the liver, spleen, small bowel and kidney can be similarly damaged and again there may be little, or no, evidence of external injury. Fortunately, deceleration injuries of major vessels or viscera are much less frequent than the traumatic injuries which occur to the peripheral vessels in the shoulder and groin or the most distal parts of the upper and lower limb.

FIG. 5. Left hilar shadow due to traumatic aortic aneurysm.

FIG. 6. Angiogram of the patient shown in Fig. 5.

FIG. 7. Traumatic aneurysm lying in the concavity of the aortic arch.

Peripheral arterial injuries

The diagnosis of these injuries may, of course, be obvious when there is an open wound associated with profuse arterial haemorrhage. In the majority of instances, however, this is not the case, but the diagnosis should always be suspected if a penetrating wound lies over the course of a major artery or when a closed injury is accompanied by a rapidly increasing haematoma (Fig. 8). The diagnosis of an arterial injury should *always* be anticipated if there is an associated skeletal injury and in some sites the probability of an arterial injury is so high that it should be assumed to be present until it has been clearly demonstrated to be absent. In situations such

FIG. 8. Large groin haematoma due to blunt trauma to common femoral artery.

as fractures of the neck of the humerus, supracondylar fractures of the humerus or femur and posterior dislocations of the knee the adjacent arteries are especially at risk (Lipscomb & Burleson, 1955; Hoover, 1961). The signs of distal arterial insufficiency such as absent pulses, pallor or patchy cyanosis, coldness, paraesthesiae, anaesthesia and paralysis frequently accompanied by severe ischaemic pain are usually quite evident if sought for. Rarely, however, distal pulses may remain palpable even in the presence of a proximal major arterial injury (Hewitt, Collins & Hamit, 1969). If signs of distal ischaemia are present and especially if good pulses are present in the contralateral limb of an otherwise fit and healthy person, the diagnosis of a major vessel injury becomes almost certain. A questionable pulse must be considered an absent pulse (Gaspar *et al.*, 1968). In spite of this, it is unfortunately all too common for these signs to be misinterpreted, disregarded or attributed to traumatic arterial spasm with further procrastination and conservative management, which frequently results in the loss of the limb. Awareness and suspicion that an arterial injury may have been sustained remain the two most important factors leading to the establishment of a correct diagnosis. In most instances the diagnosis can be made on clinical signs alone and recourse to complicated angiographic techniques is seldom required. Unfortunately, the putative need for angiography may be used as yet another excuse for deferring active surgical intervention. Similarly we would stress yet again the extreme dangers of attributing severe vascular insufficiency in an injured limb to traumatic arterial spasm, with the adoption of an unwarranted and very hazardous conservative policy. In our opinion the diagnosis of traumatic arterial spasm should never be made until an organic cause for acute limb ischaemia has been positively excluded by an exploratory operation.

Traumatic arterial thrombosis

In contrast to the usually self-evident arterial injuries complicating open wounds, traumatic arterial thrombosis may cause great diagnostic difficulty. It commonly occurs when a vessel is injured by being crushed against an underlying bone or the vessel wall is actually damaged in the process of a joint dislocation or fracture. It is an extremely dangerous and treacherous condition because apart from initial diagnostic difficulties occasionally the signs of acute vascular insufficiency may not develop until several hours after the accident. In this situation a small localized lesion, usually a fracture of the intima, occurs in the vessel wall and over the next few hours a layer of platelets and fibrin form upon it. Several hours later superadded secondary thrombosis occurs

on this area producing total occlusion of the affected vessel and until this event takes place signs of severe vascular insufficiency may not develop. By this time the initial treatment of the associated injury may have been completed and the signs of ischaemia may be concealed by dressings or plaster of Paris and ischaemic pain erroneously attributed to post-operative discomfort. These lesions may be very localized and although affecting no more than a few millimetres of the vessel wall may result in occlusion of the vessel just as securely as if it had been ligated. Such an injury in which the common femoral artery was crushed against the underlying pubic ramus by a heavy weight is illustrated in Fig. 8. On clinical examination a large haematoma was present and the distal limb was cold, pulseless and paralysed. It was not clear whether the patient had sustained a neurological injury in addition to the vascular injury but exploration disclosed a complete tear in all the layers of the common femoral artery except the adventitia which remained intact (Fig. 9). Following excision of this localized lesion and the insertion of a vein graft there was a full restoration of blood flow and complete recovery in the affected limb. In our experience where, in addition to the vascular insufficiency, there is an appreciable neurological deficit, especially of the stocking-glove distribution, this is nearly always secondary to ischaemia, though sometimes individual peripheral nerve lesions may be inflicted by the causative injury. Occasionally cases are seen where following fracture of a long

FIG. 9. Complete division of intima and media due to closed injury.

bone there is genuine doubt as to whether an arterial injury has been sustained or not. From a clinical point of view one can say that if the signs of arterial insufficiency persist for longer than 2 hr it is almost certain that a vascular lesion is present and the clinical rule should be to explore all such cases. If facilities for angiography are readily available this may occasionally obviate the need for such an exploration but in the vast majority of cases it merely confirms what the clinical diagnosis has already suggested (Fig. 10). Intimal lesions do not invariably proceed to total occlusion of the lumen and rarely angiography may demonstrate that an intimal lesion has occurred or a small vessel has been avulsed without secondary thrombosis occurring in the main channel. In our experience this is rare but it undeniably occurs and may well explain some cases in which the distal pulses are absent but the viability of the limb is not seriously in doubt (Fig. 11). In the past, many such cases have been erroneously attributed to arterial spasm (Gaspar *et al.*, 1968).

Traumatic arterial spasm

Reference has already been made to the extreme dangers associated with this diagnosis. In every case of so-called traumatic arterial spasm subsequently explored we have found a demonstrable organic lesion to be present. Rarely the artery itself may be found to be intact but flow obstructed by acute

FIG. 10. Angiogram demonstrating traumatic thrombosis of the superficial femoral artery due to fractured femur.

FIG. 11. Angiogram demonstrating intimal fracture without secondary thrombosis.

augmentation of the vessel or its compression by a haematoma or an adjacent fracture, and removal of these factors is followed by an instant return of pulsatile blood flow. These constitute instances of mechanical obstruction to blood flow and should not be cited, as is sometimes the case, as instances of arterial spasm. In the classical situation the vessel appears to pulsate down to the point of obstruction where it abruptly narrows and the distal arterial tree is usually visible as a narrow pulseless cord. It is most important to appreciate that soft, recently formed thrombus will still transmit pulsation and if the vessel is examined between finger and thumb it may appear to pulsate down to the point where it narrows even when the lumen is occluded by soft thrombus, as depicted in Fig. 12. If a small arteriotomy is performed at this point in the majority of instances an intimal tear, with superadded thrombus

FIG. 12. Traumatic arterial spasm—most cases due to clot. Remember: soft clot may pulsate, explore all persistent cases, and always do fasciotomy.

FIG. 13. Intimal tear with superadded thrombus.

upon it, will be found (Fig. 13). When the appropriate repair is performed, either by tacking down the intima, performing a patch angioplasty or a localized resection, and the clamps are removed the distal vessel previously thought to be in 'spasm' immediately dilates and pulsates. It is for this reason that we consider if traumatic arterial spasm exists at all it is an extremely rare entity and every case of persistent ischaemia in which this diagnosis is made should be subjected to surgical exploration without delay. To prevaricate using vasodilator drugs, reflex heating, local cooling and similar measures results in dangerous deception with the loss of more valuable time which may ultimately end in the loss of a previously salvageable limb. Similar views have been expressed on numerous occasions in the past decade but this topic continues to bedevil the management of arterial injuries (Hardy & Tibbs, 1960; Hoover, 1961; Bradham, Buxton & Stallworth, 1964; Eastcott, 1965; Goldman, Firor & Key, 1965; Gaspar *et al.*, 1968). If thorough exploration fails to demonstrate an organic lesion it is recommended that the vessel be painted with 2.5% papaverine or forcibly dilated by injecting saline between two vascular clamps. However, we have no personal experience of either of these procedures (Kinmonth, 1952; Mustard & Bull, 1962).

Peripheral traumatic aneurysm

These lesions are unusual but may give rise to considerable difficulty in diagnosis. In our experience they are most frequently associated with comminuted fractures of the tibia. This is usually treated on admission by some form of internal fixation and several days later the patient develops pain in the calf, associated with oedema and swelling of the leg. This is liable to be misdiagnosed as a deep

venous thrombosis and if anticoagulant therapy is commenced it is likely to have disastrous consequences. On examination there is usually marked swelling of the calf and quite severe pain, associated with evidence of distal arterial insufficiency. Auscultation over the affected area may reveal a bruit and sometimes detectable pulsation may be present (Ashton & Slaney, 1970). In such a case localized damage to a vessel, often the posterior tibial artery, results in the formation of a small pulsatile haematoma, which usually contains the situation for several days until it gives way, permitting free haemorrhage to occur into the gastrocnemius and soleus muscles. Angiography in these cases can be deceptive and may indicate that a total arterial occlusion is present but subsequent exploration usually reveals the main vessel to be patent but non-pulsatile due to the considerable pressure exerted on it by the haematoma (Fig. 14). Once the proximal vessel has been controlled evacuation of the haematoma usually reveals quite a small localized defect in the arterial wall which can usually be closed with a few interrupted arterial sutures. A traumatic aneurysm may occasionally result from infection at the site of injury and if this is the case the vessels should be ligated and no local repair attempted because of the virtual certainty of a secondary haemorrhage.

FIG. 14. Apparent occlusion of distal popliteal artery by large haematoma due to small tear in peroneal artery. Immediate return of circulation on evacuation of haematoma.

Arterio-venous fistulae

These occur when a localized arterial injury is accompanied by damage to an adjacent vein with the resultant formation of a communication between them. They usually complicate stab injuries or missile wounds inflicted by small high velocity fragments but may follow closed trauma. A distinct thrill is generally present on careful palpation and a bruit, which may develop in a matter of hours, is usually detectable on auscultation. The distal pulses, however, frequently remain palpable (Smith, Szilagyi & Pfeifer, 1963; Hewitt & Collins, 1969; Drapanas *et al.*, 1970). When discovered during a routine exploration of a fresh wound or injury, they should be dealt with there and then, but if the lesion does not become apparent until several days following injury it is preferable to temporize for a few weeks before resorting to surgical intervention. Where major vessels are involved the principle of management should be to repair the arterial injury always and the venous injury whenever possible (Hughes & Jahnke, 1958; Beall *et al.*, 1963). With small and relatively unimportant vessels quadruple ligation still remains an effective form of management. When a simultaneous repair of both artery and vein is performed, it is important to fashion a flap either of fascia or muscle to lie between the two sites of repair to prevent subsequent refistulation which can and does occur. A measure which we have found useful on a few occasions is to sew a piece of synthetic graft around the arterial repair in the fashion

TABLE 2. Arterio-venous fistula in twelve cases

Treatment	
(Arteriography demonstrated fistula in 7)	
Spontaneously closed (1 after arteriography)	2
Quadruple ligation (1 death)	3
Ligature of connection (1 uncured)	2
Formal closure of artery and ligature or suture of vein (1 death)	5
Total	12
Results	
7 Cured	
2 Deaths (cardio-renal failure and air embolus)	
1 Bruit still present	
2 Spontaneous closure	

of a gaiter, thus excluding the arterial suture line from its venous counterpart (Table 2). Although a few may close spontaneously, most traumatic arterio-venous fistulae should be repaired as they are slowly progressive (Schenk *et al.*, 1960). It is often stated in textbooks that the degree of shunting from peripheral fistulae distal to the root of the limb never produces secondary cardiac effects. This is most emphatically not the case and we have seen several patients in incipient or established heart failure from distal arterio-venous fistulae (Table 3). Moreover, it has recently been demonstrated by human flowmeter studies that flows of the order of 1.5 l/min may occur at rest through the latter type of fistula representing a 3–5 fold increase over normal limb blood flow (Lee, Madden & Hershman, 1970).

TABLE 3. Arterio-venous fistula

Site	Cause	Circulatory effects
Subscapular artery to axillary vein	Shell fragment	Cardiac output 13.76 l/min
Femoral artery to vein	Gun-shot wound	Cardiac output raised 25% over resting value
Femoral artery to vein	Gun-shot wound	Cardiac failure
Posterior tibial artery to vein	Flying steel fragment	Branham's sign positive 74 → 64/min
Lower brachial artery to venae comitantes	Stone fragment while pick-axing	None
Sural artery to vein	Pitch fork in popliteal fossa	None
Femoral artery to vein	Flying steel sliver in thigh	Cardiac failure. ? Bacterial endocarditis
Posterior tibial artery to vein	Fall through glass frame	None
Common carotid artery to jugular vein	Flying glass from fire-work in bottle	None
Peroneal artery to vein	Fractured tibia and fibula	None
Axillary artery to vein	Stab wound of shoulder	Rise in blood pressure of 15 mm on closing fistula
External carotid artery to external jugular vein	Stab wound	None

It is not surprising therefore that cardiac failure may sometimes supervene in a matter of days even in a previously healthy person (Smith *et al.*, 1963).

Principles of management of arterial injuries

Local haemorrhage can usually be controlled by firm pressure with a pad and bandage. If this fails and facilities are available temporary local control using a haemostat or vascular clamp is preferable to the application of a tourniquet, which not only devitalizes the whole limb but may also damage nerves in addition.

A plan of management should be formulated early. If the limb is grossly traumatized with multiple fractures accompanied by severe vascular and nerve damage or extensive infection, especially of the anaerobic type, primary amputation still has a place and may be the wisest and most effective procedure (Porter, 1967; Cohen *et al.*, 1969). Such instances apart, if the signs of arterial insufficiency persist a major vessel injury must be assumed to be present and the affected artery explored without delay. If strong distal pulses are present in the contralateral limb it is almost certain that arterial insufficiency in the traumatized limb is due to a major arterial lesion at the site of injury. In a badly shocked patient in whom there may be complete peripheral shutdown it may be impossible to diagnose an arterial injury initially on clinical grounds alone but following restoration of an adequate circulatory volume if the pulses return in the other limbs and remain persistently absent in the traumatized limb the same considerations apply and early exploration should be undertaken. Time should not be lost by resorting to conservative measures such as vasodilator drugs and reflex heating, since these are usually completely ineffective and merely delay the institution of adequate treatment for a further few vital hours. We would stress yet again the extreme dangers associated with the diagnosis of persistent traumatic arterial spasm and reiterate that from a clinical point of view if such a diagnosis is made this should be regarded as an indication for active intervention rather than further procrastination and ineffective conservative management which may have disastrous consequences. Better by far a negative exploration rather than a neglected one!

Early repair of the vascular injury not only restores the viability of the limb but determines, to a large extent, the degree of functional recovery likely to ensue. Operation should be undertaken as soon as possible, preferably in 8 and certainly within 12 hr of injury if good results are to be anticipated, but there is no hard and fast rule and good results may still be obtained in the late case (Table 4). Conversely even when distal pulses are restored an amputation may subsequently become necessary because of massive

TABLE 4. Acute peripheral arterial injuries

Function	Time to restoration of flow (hours)				Total
	0-6	7-12	13-24	24	
Good	15*	11	2	2	30
Impaired	1		2	3*	6
Major amputation			2	2	4
Deaths		1†		1‡	2

* Includes one transmetatarsal amputation.

† One death in a patient with traumatic thrombosis of the aorta and multiple injuries.

‡ One death from respiratory failure in a chronic bronchitic with iatrogenic traumatic brachial artery thrombosis.

muscle necrosis (Cohen *et al.*, 1969; Hewitt *et al.*, 1969; Rich & Hughes, 1969).

As regards technique, the incision should be long enough to afford adequate access to the affected vessel and it should ideally enable proximal control to be obtained before the actual site of the lesion is widely exposed. In practice, however, even if the artery is completely transected, haemorrhage can be controlled by firm digital pressure whilst the vessel is quickly isolated and vascular clamps or tapes applied. Coincident fractures or dislocations should be reduced and stabilized prior to attempting arterial repair as this may be jeopardized by subsequent manipulation. However, although internal fixation is preferable it is not essential as experience both in Vietnam and in civilian practice has proved (Cohen *et al.*, 1969; Rich & Hughes, 1969; Connolly, 1970). The vessel ends and especially the ragged adventitial sleeve may require trimming but this should not be over-generous. Wherever possible arterial continuity should be restored by simple end-to-end anastomosis using fine 5/0 or 6/0 silk or synthetic sutures. It is vitally important to make sure that both the proximal and the distal arterial tree are completely free of clot before completing the anastomosis or failure is certain. An illustration of traumatic arterial thrombosis of the popliteal artery following posterior dislocation of the knee is illustrated in Fig. 15. This also demonstrates the considerable amount of secondary propagated thrombus which may occur in the distal vessel following an arterial injury. Should this event ensue it is essential to make sure that all this clot is removed using an instrument such as a Fogarty catheter prior to restoring blood flow. This is one of the most vital steps in the operation. With lateral or incomplete lesions of the arterial wall these can sometimes be repaired by sewing on a small vein patch, which can be readily fashioned from the adjacent cephalic or saphenous vein (Fig. 16). Defects of the order of 2-3 cm can usually be bridged by mobilization of the vessel and performing

FIG. 15. Traumatic thrombosis of the popliteal artery following posterior dislocation of the knee. Treated by excision and autogenous vein graft. Note the distal propagated thrombus removed with the Fogarty catheter.

an end-to-end suture; this may be facilitated by flexing neighbouring joints (Fig. 17). These techniques will permit a primary repair in 80% of cases (Morris *et al.*, 1960; Drapanas *et al.*, 1970). However, mobilization should never be carried too far and in particular major collateral vessels should never be sacrificed to enable end-to-end apposition to be obtained. In such instances, or where an end-to-end anastomosis cannot be made without undue tension on the suture line, a small segment of autogenous vein should be removed, reversed, and sewn in end-to-end (Fig. 18). These vein grafts work

extremely well and may continue to function even in the presence of mild wound infection. For this fact and their easy availability they are infinitely preferable to the use of synthetic grafts in this situation. Once blood flow has been restored in a traumatized limb it is our policy always to perform an extensive fasciotomy. This can often be done by a semi-closed technique and it is not necessary to perform extensive incisions in the distal limb in order to achieve it; all compartments however should be decompressed.

This is a point of importance because the rise of tissue pressure that can occur in a devitalized limb following restoration of arterial flow can sometimes be very considerable indeed and may in itself compromise the circulation. Similar considerations apply in the case of closed compartment injuries when a timely fasciotomy may result in an instant return of arterial blood flow (Nolan & McQuillan, 1965). It is also important if major veins have been damaged to repair these wherever possible to ensure that venous outflow from the limb will be adequate as severe venous insufficiency may also jeopardize limb survival (Treiman, Doty & Gaspar, 1966). Primary skin cover should be utilized to cover the site of arterial repair whenever possible but experience in Vietnam has conclusively demonstrated that, provided the arterial repair can be covered by muscle, delayed primary suture can be performed with safety (Cohen *et al.*, 1969; Hewitt *et al.*, 1969). If appreciable infection develops around the site of

FIG. 16. Vein patch angioplasty.

FIG. 17. End-to-end suture after excision of 2.5 cm of the external iliac artery.

FIG. 18. Repair of defect of external iliac and common femoral artery by autogenous vein graft.

any arterial anastomosis a secondary haemorrhage is almost inevitable and the only way in which this can then be handled is usually to tie off the affected artery, usually with loss of the limb. It may rarely be possible to institute an extensive by-pass graft around the infected area but this is a major procedure involving a reasonable amount of expertise and experience.

It is never necessary to put the patient on anti-coagulant therapy and it may be dangerous. If the

repair has been properly performed it will remain patent and if not no amount of anticoagulants will prevent its failure. If the peripheral pulses disappear again in the first few hours following a successful reconstruction this constitutes a strong indication for urgent re-exploration. Finally it is wise to prescribe wide-spectrum antibiotic cover, preferably commencing this prior to operation because of the risks attendant on infection.

In conclusion we would emphasize that acute

impairment of blood flow in a major artery is a surgical emergency of the first order and should take precedence over almost all others except exsanguinating haemorrhage and acute cardio-respiratory insufficiency. It follows that the initial management must be concerned with the preservation of the patient's life and the restoration of an adequate circulating blood volume. Once achieved, this should be followed by a planned programme of management in which the arterial injury must be accorded a high priority.

The traditional pessimism that tends to surround the subject of arterial injuries should be abandoned, for there is now abundant evidence that a combination of prompt diagnosis with an aggressive approach and early surgery can result in a high proportion of excellent results. The majority of the failures are due to indecision and imprecision on the part of the surgeon, rather than any inherent difficulties in surgical technique and simple arterial repair should be within the competence of most average surgeons. We would stress yet again that success is inversely proportional to the delay and that failures when they occur are usually attributable to failure to appreciate the implications of the clinical evidence. Early diagnosis followed by early intervention still remain the prime essentials for a successful outcome, and the earliest possible restoration of arterial blood flow should be the objective in every case unless the limb is irreparably damaged.

'Expectant treatment was all very well when there was no alternative, but now it is time to abandon its cherished ritual of sympathetic blocks and blasts of cold air from fans in favour of something better' (Eastcott, 1965).

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