Med. J. Malaysia Vol. XXXII No. 2, December, 1977

Acute arterial embolism

by H. S. Saw
M.B.B.S., F.R.A.C.S.
Department of Surgery,
University Hospital,
Kuala Lumpur.

Summary
ACUTE PERIPHERAL ARTERIAL EMBOLISM is commonly encountered today. The significance of considering this entity as a medical and surgical emergency is stressed and its management outlined.

Introduction

Of recent occurrence, is an increase in the number of referrals of patients with acute arterial embolism. Unfortunately, these patients are seen at such a late stage that nothing short of an amputation of the affected extremity, is the only definitive therapy that can be entertained. In the light of this experience, it is felt that a brief note about the management of arterial embolism is perhaps both indicated and timely.

The purpose of this communication is to present a simple method of dealing with the problem of an acute peripheral embolus, with the hope that embolectomy will be in the armamentarium of the General Surgeon.

Clinical course

Regardless of the origin of an embolus and the underlying disease process, it is the location of the embolus and its subsequent propagation that determines the viability of the limb. Following occlusion, a softer coagulum (thrombus) forms in areas of decreased flow. Linton (1941) has emphasized that this propagation of thrombus distal to the embolus is of major importance to the outcome of the disease. It would not be difficult to appreciate, therefore, that time factor is an important consideration in management.

Preoperative assessment

The sudden onset of symptoms following embolisation is dramatic and its recognition and diagnosis should present no major problem. Atrial fibrillation is frequently present and is perhaps responsible for the source of the embolus in a vast majority of cases. Mural thrombi in a patient with acute myocardial infarction is not an uncommon source of an embolus. In the absence of atrial fibrillation and a history of myocardial infarction, one should consider primary arterial thrombosis as the more probable cause of the symptoms.

The time of onset of symptoms is noted since this may well determine the final prognosis. Ideally, treatment should be instituted within 6 hours of the embolic incident.

In the physical examination of the patient, the colour and temperature of the extremity are recorded. Peripheral pulses must be meticulously palpated for and their presence or absence accurately documented. There is almost never any necessity to confirm the diagnosis by preoperative angiography.

These patients invariably have concomitant heart disease. Hence, careful evaluation of cardiac function must proceed simultaneously with initial assessment and medical treatment.

Initial medical treatment

Morphine, Digoxin, diuretics and antiarrhythmic drugs are vital in the initial medical management of acute vascular occlusion.

Heparin is given immediately upon diagnosis in order to prevent repeated embolisation and to minimize distal propagation of the thrombus. The vasodilatation and anti-inflammatory properties of heparin are also beneficial to the patient. The initial dose is 5000 units intravenously. Thereafter, the same dose should be given 4 hourly to maintain a clotting time of between 2–3 times that of a control. Blood is taken $3\frac{1}{2}$ hours after a dose and the clotting time determined; if the clotting time is 2–3 times the control level, another 5000 units of heparin is given; if the clotting time exceeds 3 times the control, heparin is withheld till the next clotting time determination. Should the clotting time be less than twice that of the control, $3\frac{1}{2}$ hours after the initial dose, 7500 units may be given as the next dose instead of 5000 units (Fig. 1).

ANTICOAGULATION CHART

DATE	TIME	TIME INTERVAL (HOURS)	CLOTTING TIME RATIO (PATIENT/CONTROL)	HEPARIN DOSE (UNITS)	NURSES'S INITIALS
12/8/76	0900 AM	0	1·0	5000	
	1230	3½	1·5	–	
	1300	½	–	7500	
	1630	3½	2·1	–	
	1700	½	–	5000	
	2030	3½	2·5	–	
	2100	½	–	5000	
13/8/76	0030	3½	3·5	–	
	0100	½	–	OMIT	
	0330	3½	2·9	–	
	0400	½	–	5000	
	0730	3½	2·9	–	
	0800	½	–	5000	
	1130	3½			
	1200	½			

Figure 1

A sample anticoagulation chart

Operative management

Since Fogarty's (1963) original description of the technique of embolectomy using a balloon catheter, the mortality and morbidity associated with this condition has markedly decreased. It is important to note that the main aim in surgical intervention is to save the affected limb, hence restoration of the circulation to its immediate pre-embolic state should be the goal; lengthly reconstructive surgery should not be attempted. The surgical instruments commonly used in performing an embolectomy are as shown in Figure 2.

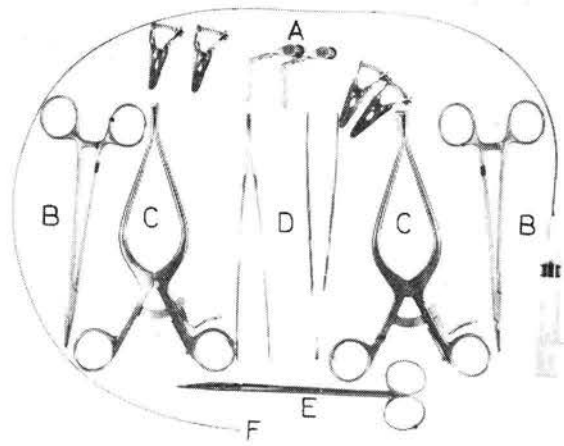

Figure 2
Basic Instruments for embolectomy.
A – variously angled vascular clamps; B – fine-tipped needle holders; C – Weitlaner's Self-retaining retractors; D – Carbide-tipped vascular dissecting forceps; E – sharp-pointed angle-on-flap scissors; F – Fogarty embolectomy catheters (size 3 + 4 Fr).

Under local anaesthesia and morphine sedation, the classical incision to expose the proximal femoral artery is made (Inset, Fig. 3). The common femoral, superficial femoral and profunda femoris arteries are circumferentially mobilized and tapes passed around them individually.

Prior to clamping the vessels, 5000 units of heparin is given intravenously. The common femoral and profunda femoris arteries are then clamped. A longitudinal arteriotomy is made and a suitably sized Fogarty catheter passed as far down as possible into the superficial femoral artery (Fig. 3, A). So as to reduce blood loss, the unclamped vessel may be angulated by traction on the encircling tapes during passage of the catheter. The balloon is inflated and the catheter gradually withdrawn. This process should be repeated until no further clots are obtained or until back-bleeding occurs. The superficial femoral artery is then clamped and the catheter passed down the profunda femoris artery (Fig. 3, B). Finally, the catheter is passed proximally into the aorta (Fig. 3, C). If there is suspicion that the embolus has propagated proximally to be sufficiently close to the aortic bifurcation, the contralateral common femoral artery should be exposed and clamped before passage of the Fogarty catheter proximally into the aorta, lest a piece of the thrombus be shot off into this "good" artery during the process. Repeated passes must be made until one is confident that all obstructing thrombi have been removed. After this 25–50 ml of heparinized saline (5000 units

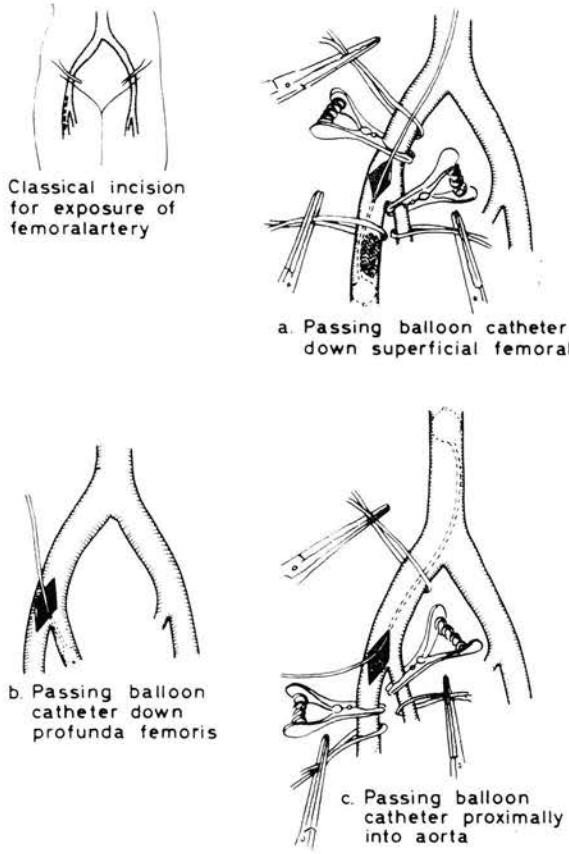

Figure 3

Technique of Embolectomy

heparin in 250 ml normal saline) should be injected into the distal vessels.

Primary closure of the arteriotomy (Fig. 4, A) is then attempted using 4-0 Prolene, Ethiflex or Tevdek. If the vessel is atherosclerotic and the arteriotomy inadvertently made jagged in the process of the embolectomy, primary closure without compromise to the lumen of the artery may not be possible. Under such circumstances, a short segment of saphenous vein may be excised via the same incision and used as a vein patch for the closure of the arteriotomy (Fig. 4, B).

A 21 gauge needle may be placed in the artery just distal to the most proximal clamp to serve as an air vent as the distal clamps are released first.

Since the patient has been anticoagulated, meticulous haemostasis is imperative before closing the various layers of the incision. It is advisable to

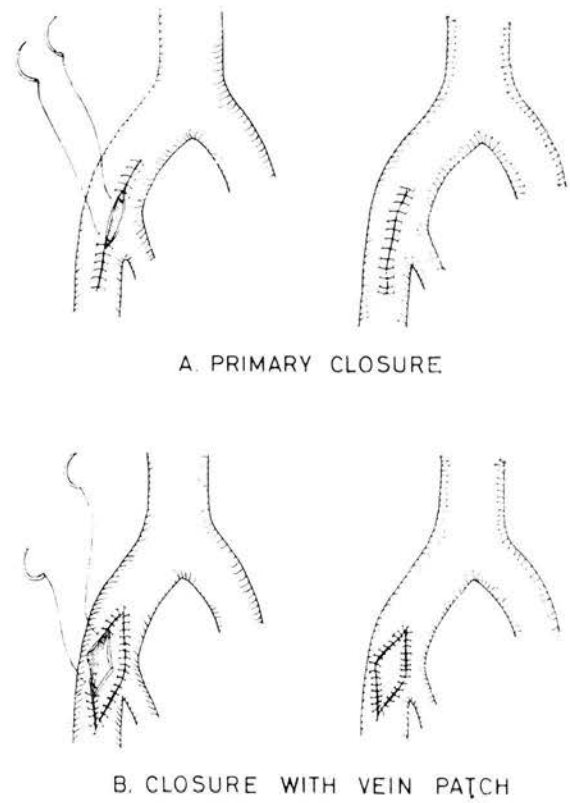

Figure 4

Closure of Arteriotomy

insert a Radivac drain in the area of dissection so as to prevent haematoma formation.

Post-operative management

During the post-operative period, heparin administration is continued according to the plan outlined earlier. The moment the patient is well enough to tolerate oral medications, he should be started on Warfarin and maintained on it for about 3–6 months; heparin being taken off the moment adequate anti-coagulation is obtained with Warfarin.

In the meantime, all therapy for his underlying cardiac and/or other medical condition should continue uninterrupted.

Discussion

Emboli, as a major cause of acute arterial occlusion resulting in gangrene has been recognized since time immemorial. Soon after embolectomy was popularised, it was observed that operations performed within 4–6 hours of the lodgement of the embolus were largely successful whereas those

carried out subsequently had a progressively higher failure rate. Based on this observation it was proposed that patients who had emboli lodged more than 12 hours previously should not be operated upon. This concept has now proved to be wrong: with proper management, viability can be preserved in well over 90% of patients so long as surgery is conducted before the muscles become necrotic. However, in delayed cases, achieving cure taxes the resources, skills and ingenuity of the vascular surgeon to the utmost for thrombi may have propagated very distally and complete removal require multiple incisions and perhaps serial operative arteriograms.

The operative procedure must be planned with regard to the influence of anaesthesia and of the operation, on the heart since patients with arterial emboli usually have serious cardiac disease. This is especially so, if the embolus had originated from a mural thrombus consequent upon a myocardial infarction. Because embolectomy can be done safely and with minimal trauma under local anaesthesia, few patients should be denied the benefit of such an operation. Imminent death and definite gangrene constitute about the only reasons not to operate. Procrastination of the surgical procedure in the vain hope of getting the patient's heart into better shape, can only invite gangrene which, of necessity, requires a major amputation – a much greater surgical stress than simple embolectomy.

Post-operatively, adequacy of the peripheral circulation must be ascertained. A palpable pulse is the most useful clinical sign and this should be identified immediately after operation. The anticoagulant effect of the heparin given preoperatively and intra-operatively should not be reversed with Protamine. In fact, anticoagulant therapy should continue into the post-operative period. The importance of prompt and continuous anticoagulation cannot be over-emphasized for the arterial embolus is only a symptom of serious heart disease. Unless the underlying source of the embolus is simultaneously treated, the patient runs a risk of recurrence of emboli, each with a 25–30% likelihood of lodging in the brain.

The prognosis of these patients depends on the ability to prevent further emboli. If anticoagulation is inadequate, recurrence is inevitable and this can only terminate in fatal or crippling cerebral thrombosis. Attention to prevent recurrent embolization can be relaxed after the predisposing cardiac condition has been taken care of e.g. a mitral valvotomy has been performed for mitral stenosis, atrial fibrillation has reverted to sinus rhythm or a myocardial infarction has healed. In many large series, the hospital mortality is about 30%; usually from the underlying heart condition. Our experience, however, does not bear this out as many of our patients arrive in extremis, sometimes several days after the onset of symptoms.

References

1. Fogarty, T.J., Cranley, J.J., Krause, R.J., Strasser, E.S and Hafner, C.D. (1963). A method for extraction of arterial emboli and thrombi. *Surg. Gynae. & Obst.* **116:** 241.

2. Linton, R.R. (1941). Peripheral arterial embolism – a discussion of the post-embolic vascular changes and their relation to the restoration of circulation in peripheral embolism. *New England J. Med.* **224:** 189.