THE MANAGEMENT OF PERIPHERAL ARTERIAL EMBOLI

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ONE of the main difficulties in assessing the results of any surgical procedure is the provision of a directly comparable series of patients on whom it has not been employed. Nowhere is this more evident than in trying to determine the value of embolectomy in the management of peripheral arterial emboli. The problem of comparing the results of conservative and operative management is considerable, due to the fact that conservative treatment is employed in poor risk patients in whom surgery of any kind is inadvisable and also in those patients whose limbs are recovering spontaneously when they are first seen.

Peripheral arterial embolism is a grave condition. This is evidenced by the fact that in most published series of cases the mortality rate is in excess of 40 per cent whether embolectomy has been employed or not. It has even been suggested that in some series where surgical intervention was extensively employed, the mortality rate was higher than if conservative management alone was used. However, we feel this does not necessarily mean that surgery should not be advised. Although the overall results are poor in terms of mortality and loss of limb attributable to the natural history of the disease, a worthwhile result can be achieved in a minority of patients whose limb would not otherwise be restored to normal function. The problem of selection of those patients who are likely to benefit from embolectomy is difficult if not impossible. It is therefore inevitable that the procedure will be employed in a number of instances where the eventual outcome is fatal.

Historical Review

The pathology of embolism was first described by Virchow in 1854. It was not until half a century later, however, that surgical relief of the condition was attempted.

The first successful embolectomy in this country was performed by Jefferson (1925),

who also achieved the first successful aortic embolectomy in England, as reported by Griffiths (1938). Since then there have been periodic changes of opinion with regard to the advisability of performing embolectomy for acute ischaemia in the lower limb. This difference of approach has been due to several factors. Firstly, these patients are often very ill and thus poor surgical risks, even for what may be a relatively minor procedure. Many of them also have severe cardiac disease. Lastly, and perhaps the most relevant factor of all, emboli are often multiple. In spite of this, Shaw (1956) and Shumacker and Jacobson (1957) considered a more aggressive approach was indicated on the grounds that although some limbs might recover spontaneously, there was a considerable incidence of disability due to minor but nevertheless chronic ischaemia. Others disagreed, including Metcalfe (1960) who found a 35 per cent mortality associated with conservative management alone.

Surgeons would now agree that somewhere between these two extremes lies the most desirable course. The problem of accurate selection, however, remains.

Pathology

Whereas the most common clinical problem associated with arterial embolism is concerned with occlusion of the vessels of the lower limb, the cerebral and visceral vessels and those of the upper extremity may also be affected. In these latter situations therapeutic considerations will be modified by several additional factors. First, the location may be inaccessible as in the case of the cerebral vessels. Secondly, there may be no collateral circulation as in the case of the abdominal viscera, and irreversible changes may rapidly supervene. On the other hand, embolic occlusion of the vessels of the upper extremity is attended with a high rate of spontaneous recovery due to the ready response of the collateral circulation.

The source of arterial emboli is usually the left side of the heart, although the causative condition is not always apparent or demonstrable. Many of the patients seen have some cardiac irregularity such as atrial fibrillation. As one would expect the commonest associated condition is mitral valve disease. Next in frequency is arteriosclerotic heart disease, particularly in those patients who show clinical or other evidence of myocardial ischaemia. Occasionally the source of emboli is not clear, and instances have been reported of emboli arising from the wall of the aorta itself, especially in the presence of an aortic aneurysm or gross atherosclerosis. In approximately 10 per cent of cases, no cause can be found. Some of these, however, may well originate as a mural thrombus secondary to a "silent" myocardial infarct.

While most embolic occlusions are clinically manifest as instances of acute or subacute ischaemia, symptomless emboli do occur. The mechanism here is difficult to explain. It may be that because of narrowing of the vessel by pre-existent disease the collateral circulation has already been partially developed.

Emboli tend to become lodged or impacted at arterial bifurcations. This is due to the fact that when an artery divides its branches are correspondingly reduced in size. The common sites of impaction then in the lower extremity are the bifurcations of the aorta, the common femoral and popliteal arteries; of these the common femoral is the most frequently seen. In the case of the upper limb the distal brachial artery will be the usual location, although an embolus may also become lodged more proximally. An embolus usually produces complete occlusion of an artery or arteries or at least does so promptly when aided by spasm and the formation of a secondary clot. Occasionally, however, a partial occlusion is found which will allow the passage of some blood distally. As well as occluding a main artery and its bifurcation, several smaller but nevertheless important vessels which might form collaterals may also be occluded, where they arise close to a bifurcation. It must also be remembered that multiple small emboli in the presence of severe spasm may simulate a complete more proximal occlusion.

Following the occlusion, complete cessation of blood flow occurs distally and this favours thrombosis. Some clotting may also occur proximally but this is of less importance from a technical point of view. Spasm affecting the

main vessel and vessels forming potential collaterals may also be present and is presumably mediated by local nervous reflexes. Thus a clinical picture of acute ischaemia may be produced. The onset of irreversible tissue changes will depend on the readiness or otherwise of the collaterals to develop. The onset of distal thrombosis and the rate at which it progresses may also limit the extent to which the collateral circulation can develop. However, no definite time interval can be placed on this sequence of events, one can only attempt to assess it clinically.

One further important point should be mentioned, namely the presence of pre-existing arterial disease. Whereas in normal vessels emboli tend to become impacted at bifurcations in the case of vessels previously narrowed by arterial disease this is not necessarily so. In such cases the location of the embolus may be a point of narrowing in the artery, at for example, an atheromatous plaque. This feature is of considerable importance, for it may mean that as some collateral circulation is already present, the impact of sudden occlusion may not be so dramatic.

When a considerable volume of ischaemic tissue has been present for some time, restoration of blood flow to the part may liberate the many harmful products of anaerobic metabolism. This, in turn, may give rise to kidney damage and a condition simulating the crush syndrome. Other metabolic and homeostatic problems may also be called into play in certain circumstances. It is possible that such factors may play a part in the alleged increased mortality following embolectomy.

Diagnosis

In most cases an accurate diagnosis of embolic occlusion of a major limb vessel can be made on the mode of onset and the clinical examination. The presence of a pulseless, painful, cold and anaesthetic limb in which the skin is often white or mottled in colour is unmistakable. Frequently motor activity is absent or at least reduced. Whereas most of these physical signs are usually present in any one case, pain is not necessarily so. In fact, some patients may assert that they have no pain, but that the limb feels "dead" or limp. The absence of pulsation, coldness and sensory loss of stocking distribution are the most reliable signs. Occasionally sensory loss is equivocal. Finally, the presence of an irregular pulse with or without other evidence of heart disease will be strong confirmatory evidence.

Whereas the diagnosis of embolism is relatively easy, recognition of the site of the occlusion may not be so simple. Accuracy of diagnosis can be influenced by the bodily configuration of the patient. When the subject is obese the peripheral pulses may be difficult to feel even in the most favourable circumstances. It is therefore essential to have some other means of assessment. This is afforded by the level of sensory loss and the presence of a marked temperature gradient. Reliable confirmation of the absence of adequate arterial flow will be obtained by employing an oscillometer.

When the aortic bifurcation is occluded by an embolus, signs will be present in both legs. The femoral pulses will be absent and sensory and temperature loss will be evident as high as the groins. There is usually complete loss of motor power at least as high as the knee. The proximal level of the occlusion may be confirmed by palpating the abdomen above the umbilicus.

In the case of a common femoral embolus it is important to realise from the outset that femoral pulsation may still be present. This is particularly the case if the profunda is given off some inches below the inguinal ligament. One is therefore able to palpate the artery above the embolus. In some patients the artery at the site of impaction is tender and we consider this a most reliable indication of the location of the embolus. Other signs include sensory loss to the level of the knee or mid-calf, together with a sudden temperature drop at the same level. Incidentally, in assessing temperature gradients one should compare the limb with the opposite side as sudden gradients do occur at this level in the normal. Ankle and popliteal pulses will be absent. Popliteal embolus, on the other hand, produces ischaemic changes in the foot or lower leg, and motor activity is rarely impaired.

In the upper limb the diagnosis is easier as the pulses are more readily palpable, and providing the occlusion is not too far proximal the site of occlusion can be detected by the use of the oscillometer.

It is important from the outset to know the duration of symptoms as accurately as possible. On this time interval and the findings at the first examination, a baseline is formed on which one can note further deterioration or improvement. This information is vital from the point of view of treatment.

Various other signs may be present depend-

ing on the duration of symptoms. Firstly, there may be signs of improvement even when this can only be deduced from the history. On the other hand there may be continued deterioration. Severe tissue damage may have occurred in the digits in the form of intense discolouration. Frank gangrene takes several days to develop. There may also be muscle tenderness. While this is strongly indicative of muscle necrosis it does not necessarily mean that irreversible changes have taken place. We have seen complete functional recovery in two limbs in which muscle tenderness was initially present.

The differential diagnosis is firstly from an acute arterial thrombosis. A more gradual onset, together with a previous history of insufficiency in the limb, is more indicative of a thrombosis in the absence of any obvious source of embolus. However, emboli may sometimes be derived from a "silent" source. When in doubt it is safer to err on the side of an embolic episode and to submit it to the last method of diagnosis, namely exploration, provided surgery is indicated. Occasionally a localised dissection of a vessel can occur and by narrowing its lumen induce a thrombosis simulating an embolus. Ilio-femoral venous thrombosis frequently seems to cause confusion in diagnosis. Here, however, the onset is gradual and is measured in days rather than hours. Although the limb feels heavy and weak due to the associated swelling (which is rarely seen in arterial disease) there is no loss of sensation or motor activity. Though the pulses are difficult if not impossible to feel, oscillometry will reveal adequate arterial circulation.

Emboli are multiple in 40 per cent of cases. Therefore no clinical assessment is complete without a diligent search for the presence of other emboli. It is of particular importance to examine the abdomen carefully and also to test the urine. It is also not unknown for an embolus to break up at its site of impaction into several fragments which are carried distally. Therefore, if there appears to be an embolic occlusion of the vessels of one leg the other leg should also be carefully examined.

Treatment

It has been intimated that as arterial embolism is a serious condition, there are good grounds for recommending conservative management. This is due to several factors. First, emboli are often multiple and may involve

vital centres where they are inaccessible to treatment. Secondly, the patient may be gravely ill from associated cardiac disease. Lastly, embolic occlusions in the lower limb have a fair chance of spontaneous recovery. However, in spite of these factors we feel that operative treatment has an important part to play. We base this view on the presence of a small number of patients who by operation are restored to complete function, for whom the withholding of surgery would be attended with the risk of chronic limb ischaemia or even amputation.

Our indications for surgical treatment then are as follows: Any patient with clinical evidence of embolic occlusion of a major limb vessel should be considered if there is not marked evidence of early spontaneous recovery. In some sites the incidence of adequate spontaneous recovery is high. Such is the case in the upper limb where we have not seen a patient requiring amputation or suffering from post-ischaemic symptoms. A comparable situation exists for popliteal emboli where although the incidence of post-embolic symptoms is higher, they may not be of great significance to the patient. When an embolus lodges in the superficial femoral artery, an adequate collateral circulation develops for reasons already discussed. Thus the main clinical problem is one of management of proximal main vessel occlusion in the lower limb, that is, the aortic, iliac and common femoral bifurcations. In these sites the likelihood of spontaneous recovery is slight and even if amputation can be avoided the patient may experience severe chronic ischaemia, such as severe claudication or even occasional mild rest pain. If embolectomy can restore normal circulation in these cases the advantages are obvious.

Although emboli are frequently multiple, this cannot be forecast and so should not constitute an objection to surgery. The main contraindications, in our opinion, are as follows: An extremely ill patient who would not tolerate an operation in any form; marked signs of clinical improvement within eight hours of the incident, and the presence of definite and irreversible tissue damage.

When mitral stenosis is associated with a peripheral embolus, the possibility of correcting the cardiac cause of the embolus must be considered. Opinion is divided as to whether the cardiac operation should be undertaken at the same time as the embolectomy, or delayed until the earliest favourable occasion.

Aortic Embolectomy

We feel confident the most satisfactory technique is a direct approach. While retrograde evacuation of the clot via the femoral arteries can be achieved, some clot may remain undetected at the bifurcation and give rise to further trouble. Therefore, unless the patient is very ill, we prefer a laparotomy. Both techniques are relatively straightforward. Any abdominal incision will do that provides adequate exposure of the aortic bifurcation. Having done so and confirmed the diagnosis the aorta and both common iliac vessels are defined and slings passed around them. Care should be taken not to disturb the clot. The aorta is then clamped proximally and a longitudinal incision is made in the wall of the aorta just above its bifurcation. It is then a simple matter to remove the clot. Brisk back bleeding from both common iliac arteries is very reassuring at this point and when one is sure that all the clot has been washed out the bleeding is controlled by tightening the slings. Clamps are then applied. At this point, dilute heparin solution is injected into the aorta and also distal to the iliac clamps. It is useful at this stage to confirm that at least part of the back bleeding is from the external iliac arteries, and not from the internal vessels alone. If not, then both common femoral vessels are exposed through separate groin incisions, and the arteries carefully inspected. If clot is palpable, then further arteriotomies must be made and the clot evacuated. Having confirmed that no further distal clot is present as judged by back bleeding the arteriotomies are sewn up with fine continuous silk and the clamps removed. Oozing from the arterial wounds is controlled by pressure with a gauze swab. Observation of the legs, which should be left uncovered throughout the procedure, will reveal if adequate flow has been restored. There should be signs of immediate improvement in colour and temperature. Provided that there has not been undue delay in performing the operation, a reactive hyperaemia will be apparent. Although restoration of ankle pulses is gratifying at this point, it is in fact unusual and does not necessarily mean that organic obstruction is still present. If available, an arteriogram is useful in doubtful cases, whereby any residual clot or mere spasm distally can be detected.

Performing an aortic embolectomy via the common femoral arteries while less satisfactory, has its advantages in the very ill patient provided it is successful. It may be performed

under local anaesthesia. The embolus can be removed either by suction, using a catheter, or alternatively by employing a wire loop or spiral such as is used for disobliteration. If anything less than full flow is obtained when the clot is removed then the aortic bifurcation should be exposed to rule out the presence of any residual clot. This latter factor may well be responsible for the persistence of marked spasm in some cases.

Femoral Embolectomy

Regarding the technique of common femoral embolectomy there is little to be added to the well known general principles. Removal of the distal clot is again the main consideration. We have found a moderately firm polythene catheter very useful for this purpose. This is advanced down the superficial femoral artery and when in position strong suction is applied. It is then slowly withdrawn. If retrograde flow is not satisfactory after removal of the embolus and its secondary clot, then exploration of the popliteal artery is imperative. This is most readily done by a longitudinal incision one hand's breadth behind the medial border of the patella. It is helpful to have the patient's pelvis tilted to the same side and the hip and knee partially flexed. The popliteal space is entered in front of and deep to the sartorius muscle. Further downward exposure can be readily attained by dividing the muscles attached to the medial aspect of the upper end of the tibia and the medial head of gastrocnemius. Provided these muscles are carefully repaired later we have not seen any disability. The popliteal artery can then be exposed down to its bifurcation with ease. From this vantage point the anterior and posterior tibial arteries can be explored with the catheter as previously described and any further clot removed. We do not routinely anticoagulate these patients, and the heparin administered locally is small in amount.

Conservative Treatment

The details of conservative management are widely known. Of the many techniques directed at saving the limb, keeping it cool in relationship to the rest of the body and slight dependency, are the most important. The place of anticoagulants is rather more obscure and general agreement has not yet been reached. Their theoretical value directed to limiting the extent of distal clot formation is not entirely convincing. Low molecular weight dextrans may have some future in this field. Whether anticoagulants prevent the occurrence of further immediate emboli is questionable. Energetic measures to correct any heart failure or other associated disease should be promptly undertaken. We would emphasise that prevention of hypotension is of the greatest importance, not only in conservative management but also during and after surgery.

While the use of sympathetic nerve block in the acute phase of ischaemia is uncertain, it should be employed in cases following surgery when severe spasm is persistent. Its more widespread employment as part of routine conservative treatment has the usual theoretical disadvantages. However, it is of the greatest help in the treatment of post-embolic ischaemia, when it may be carried out as a paravertebral block with Phenol, or lumbar sympathectomy.

Conclusion

Conservative management must remain the standard method of treatment in the majority of patients who suffer an embolic episode. It can be seen that the opportunities for achieving lasting benefits from embolectomy are confined to a small proportion of patients, whose existence, nevertheless, makes the procedure worthwhile. A more profitable approach would be the effective prevention of recurrent embolic episodes, and much work remains to be done in this field. We also await with interest any forthcoming information on the value of low molecular weight dextrans in the treatment of these cases.

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