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Pulmonary embolectomy re-evaluated

David B Clarke FRCS
Consultant Cardiothoracic Surgeon, Queen Elizabeth Hospital, Birmingham

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Summary

Forty-two patients who had sustained massive pulmonary embolism were treated by emergency pulmonary embolectomy using normothermic venous inflow occlusion circulatory arrest. Of 26 patients who had not had cardiac arrest before surgery 25 survived the operation, but 7 later died from various causes. Only 1 of 16 patients who had sustained cardiac arrest survived. In all, 19 patients (45.2%) left hospital alive. This simple and widely applicable technique has enabled an emergency pulmonary embolectomy service to be offered to all the hospitals in a metropolitan area.

Introduction

Pulmonary embolism was first described in detail by Virchow in 1850, 50 years after the death of John Hunter. The lessons of scientific observation and critical evaluation of results taught to us by Hunter have much to do with the development of the basic surgical principles which run as a clearly discernible thread through the complexities of modern surgical practice, and yet in the management of massive pulmonary embolism these general principles would seem to be denied. It is generally accepted that the relief of obstruction is a fundamental role of surgery. Paradoxically this is not held to be true when the pulmonary arteries are critically obstructed.

Pulmonary embolectomy is performed rarely and when it is performed it is frequently attempted too late. It is my intention to examine the reasons which have led us to adopt an attitude so at variance with our approach to acute obstruction in other situations, to present our experience with pulmonary embolectomy, and to ask whether the role of this operation should not, in the light of this experience, be re-evaluated.

The first pulmonary embolectomy was performed by Friedrich Trendelenberg in 1908. In three operations attempted, one patient died on the operating table and two survived to return to the ward but later died, one from right ventricular failure and the other from bleeding from the internal mammary artery *(1)*. It was not until 1924 that his pupil Kirchner first succeeded in saving a patient's life. In succeeding years a scattering of successes were achieved by Mayer in Berlin, by Crafoord and Nystrom in Sweden, and by Ivor Lewis in Britain, but these were sparse reward for the many operations attempted.

In 1958 Steenberg performed the first successful pulmonary embolectomy in the United States. At this time there were only 12 successful cases in the world literature. In 1960 Allison of Oxford performed pulmonary embolectomy under hypothermia induced by surface cooling, and in the same year Ivor Lewis described the technique of venous inflow occlusion *(2)*, a technique which we have adopted. The following year Sharp *(3)* performed the first pulmonary embolectomy using cardiopulmonary bypass and this success was repeated later in the same year by Cooley.

The introduction of enzyme therapy designed to dissolve the embolus without recourse to surgery diverted attention away from pulmonary embolectomy, but a few small series continued to be reported, such as that of Vossschulte in 1965 *(4)*. In 1967 Cross and Mowlem *(5)* published a survey of 137 pulmonary embolectomies which had been performed in 28 centres in the United States and Canada. The total experience was shared by 40 surgeons. Cardiopulmonary bypass, with its 43% success rate, was obviously far more successful than the older techniques, which carried a 90% mortality.

Today the initial management of the patient with a massive pulmonary embolus is usually with intravenous heparin. Streptokinase and urokinase, which are capable of dissolving thrombus, are claimed to be more effective. Controversy still surrounds the role of pulmonary embolectomy. Pulmonary embolism is not a minor problem. It is the commonest reason for death from pulmonary causes in hospital and is an important cause of death in surgical practice.

It is not easy to state with any degree of precision the number of patients who are likely to benefit from surgical removal of their pulmonary emboli.

Figure 1 is derived from reports *(6,7)* an-

Hunterian Lecture delivered on 27th February 1980

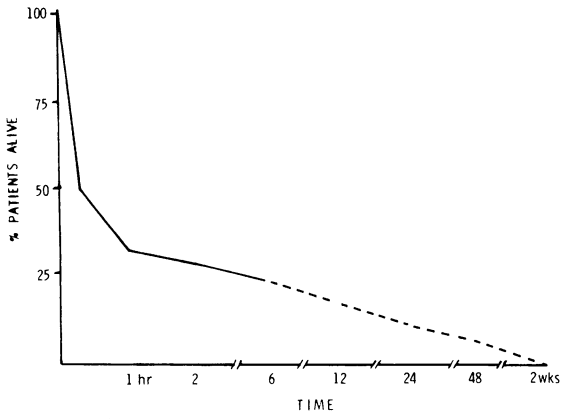

FIG. 1 *Time of death after massive pulmonary embolism.*

alysing the times at which patients sustaining a massive embolism died. It is apparent that although half die within 20 min, one-third are still alive at 1 h and thereafter a significant number are still alive up to 12 h later. Survival to 48 h is no guarantee that the patient will eventually leave hospital alive, and death from pulmonary embolism can occur up to 2 weeks after the initial episode. To make any impression on this mortality treatment, be it surgical or medical, must succeed in relieving the obstructed pulmonary artery within the first hour following the onset of symptoms, but there is still much to be gained by effective treatment later than this. It has been our conviction that surgical relief of the obstruction performed at the earliest possible opportunity is the most effective means at our disposal for reducing the mortality of massive pulmonary embolism, and to this end we have offered a pulmonary embolectomy service in Birmingham for the past 20 years *(8,9)*. I must acknowledge my indebtedness to my colleague Leon Abrams; the results to be presented represent our combined experience.

Diagnosis

The diagnosis of pulmonary embolism has largely been made on clinical findings supplemented by the chest radiograph and the electrocardiogram (ECG). The sudden onset of acute breathlessness, sometimes with a crushing pain in the chest and cyanosis, is characteristic. In about 75% of our patients a third heart sound has been audible over the right ventricle. The association of cyanosis with hypotension has been shown to be indicative of a poor prognosis and patients have been accepted for surgery when the systolic blood pressure was reduced to 100 mm Hg (13.3 kPa) or less. The ECG will often reveal the classical pattern of a deep S wave in Lead I and a Q wave in Lead III. Although the features of pulmonary embolism may be recognised on the plain chest radiograph, these are often open to debate and the principal value of the chest radiograph is to exclude other causes of breathlessness, such as a tension pneumothorax.

Most of our patients have been subjected to operation without further investigation. Pulmonary angiography has seldom been employed to confirm the diagnosis. This is because the pulmonary embolectomy service has been offered to all hospitals in the Birmingham area, and in many of these it has not been possible to obtain pulmonary angiograms. Even if this investigation is available it may take an hour or more to obtain the angiogram and such delay is not acceptable when death may occur at any moment. Furthermore, the patient's condition will often deteriorate following the injection of contrast medium into the pulmonary artery. However, there have been occasions in the less sick patients when an element of doubt exists, and particularly when the history suggests that there may have been several episodes of pulmonary embolism, on which pulmonary angiography has been performed and the information thus provided has been valuable.

Pulmonary embolectomy

Venous inflow occlusion pulmonary embolectomy is a simple operation requiring few specialist instruments. All that is required to supplement the general surgical set to be found in any operating theatre is the means to divide the sternum longitudinally with a Gigli saw and three vascular clamps. Usually the theatre personnel will not have encountered this procedure before and indeed may have never seen the heart exposed at an operation. A few minutes taken in explaining quietly to the nursing personnel and the anaesthetist precisely what has to be done is vital. The anaesthetist must be aware that the time of greatest danger is during the induction of anaesthesia because vasodilatation in a patient dependent on increased vascular tone to maintain the circulation may lead to cardiac arrest. The patient must be ventilated with oxygen throughout and inotropic drugs must be immediately available.

TECHNIQUE

The sternum is exposed through a midline incision and a long pair of artery forceps is used to pass the Gigli saw behind the sternum, which is then divided. There is little bleeding and time is not spent in achieving haemostasis. The pericardium

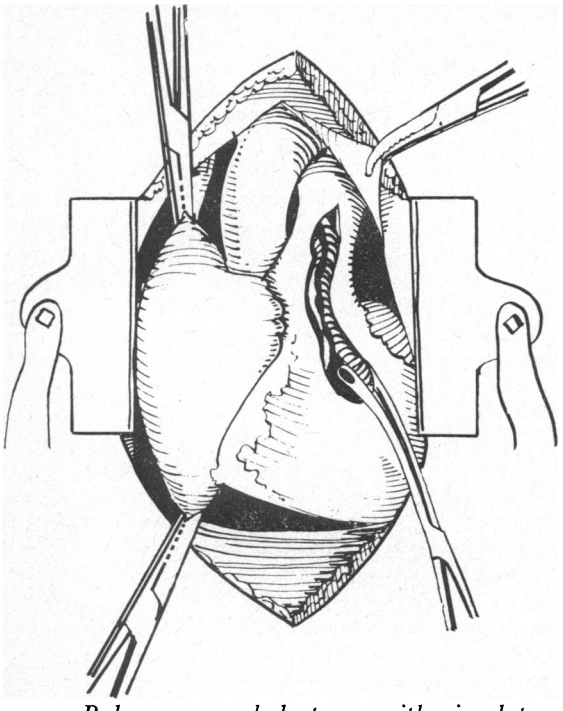

FIG. 2 *Pulmonary embolectomy with circulatory arrest produced by clamping superior and inferior venae cavae.*

is opened longitudinally and the distended and labouring heart exposed. The assistant holds the pericardium away from the pulmonary artery with a pair of forceps. The superior and inferior venae cavae are identified and occluded with vascular clamps. No venous blood can now enter the heart and the circulation will rapidly come to a standstill (Fig. 2).

The brain can tolerate circulatory arrest for 4 min at normal body temperature, but it is unwise to interrupt the circulation in a hypoxic patient for longer than 3 min. As soon as the surgeon signals that he has clamped the venae cavae the anaesthetist starts a stopwatch. The surgeon now has 3 min to explore the pulmonary artery, remove the embolus, and restore the circulation.

An incision 2 cm long is made in the main pulmonary artery and des Jardin common-duct forceps are used to explore the main pulmonary trunk and its right and left branches. Magill forceps, which are to be found on most anaesthetic trolleys, serve the purpose equally well. There is ample time to remove major emboli. A sucker may also be passed into the right and left pulmonary arteries and smaller fragments of clot removed. If the anaesthetist inflates the lungs firmly other fragments may be washed within reach of the grasping forceps. Little blood is lost once the contents of the right ventricle have been sucked out and it seldom exceeds 500 ml (Fig. 3).

When the time-keeper signals that $2\frac{1}{2}$ min have elapsed the lips of the incision in the pulmonary artery are picked up by the surgeon and his assistant and an angled vascular clamp is applied tangentially to the vessel wall. The clamps are removed from the venae cavae and the circulation restored. Usually the heart will have remained beating throughout and the restoration of the circulation is commonly followed by a short period of hypertension. If it is considered that more clot has to be removed the procedure is repeated after waiting a quarter of an hour. The incision in the pulmonary artery is then repaired with a continuous atraumatic suture.

It was our practice at one time to supplement this procedure by narrowing the inferior vena cava to prevent further pulmonary embolism. This was accomplished through an incision in the right flank, but it was noticed that the patient's condition often deteriorated quite markedly because of reduction of the venous return to the right atrium. The efficacy of caval ligation has subsequently been questioned and we no longer perform this operation.

The chest is closed with drains to the pericardium and the retrosternal space. Whenever possible the patient is returned to an intensive care unit, where blood loss and cardiac performance can be closely monitored for the next 24 h. Anticoagulation is started as soon as bleeding has ceased and the chest tubes have been removed.

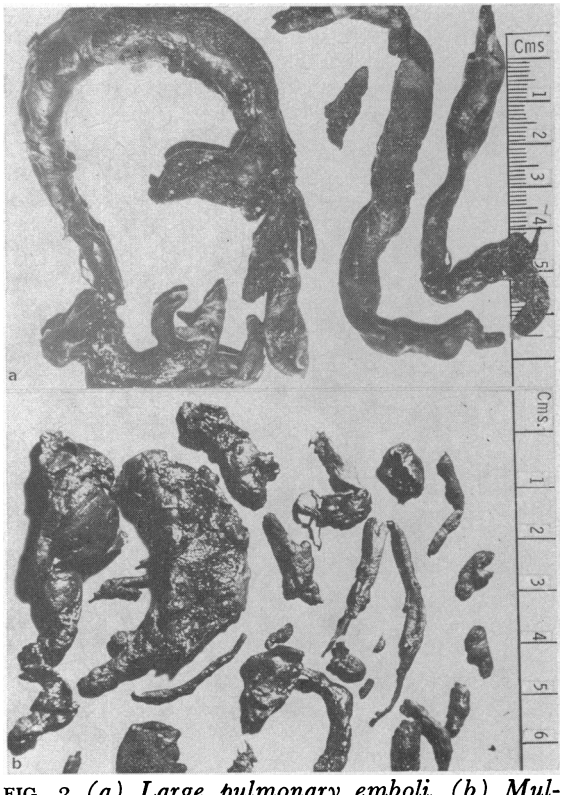

FIG. 3 *(a) Large pulmonary emboli. (b) Multiple pulmonary emboli removed during $2\frac{1}{2}$ min of circulatory arrest.*

This operation is simple to perform, does not require specially trained personnel or complicated equipment, and is extremely quick. We have succeeded regularly in removing clots from the pulmonary artery and restoring the circulation within 10 min of making the skin incision.

Results

Fifty-one pulmonary embolectomies have been performed during the past 20 years. Forty-two of these were emergency operations using venous inflow occlusion. Three were performed on cardiopulmonary bypass in less urgent cases. On 6 occasions the diagnosis of pulmonary embolism was made in error; in all these cases the patients were extremely ill, in 4 the blood pressure was unrecordable (in 2 it was less than 50 mm Hg (6.7 kPa)), and 3 out of the 6 patients had sustained an episode of cardiac arrest. It was appreciated that although the diagnosis might be in some doubt pulmonary embolism was the most likely explanation and that urgent surgery was justifiable as death would have been inevitable without it. The correct diagnosis was myocardial infarction in 3 patients. In one of these, a young woman of 26, there was previously unsuspected aortic stenosis. One patient had terminal right ventricular failure due to an unrecognised atrial septal defect, one had occlusion of a prosthetic heart valve, and one had biliary peritonitis. This last patient survived exploration of the pulmonary artery, but the others died.

The 42 patients who underwent emergency pulmonary embolectomy for proven massive pulmonary embolism will now be considered. Twenty-three of these patients died after surgery, a mortality of 54.8%. Eleven of these deaths occurred either on the operating table or within the first 24 h after surgery and 12 occurred up to 5 weeks later, 6 being related to pulmonary embolism and 6 from other causes. The total mortality attributable to pulmonary embolism was 40.5%. Nineteen patients left hospital, an overall success rate of 45.2%. It is impossible to say what this represents in terms of saving life as one is not to know how many of these patients would have survived without surgery. However, all were cyanosed, dyspnoeic, and shocked, circumstances known to be associated with poor survival.

Of these patients 18 were male and 24 were female. They ranged in age from a 16-year-old girl, who suffered massive pulmonary embolism shortly after a caesarean section, to a 70-year-old man. The results were influenced adversely by age: the success rate was only 18% in those over 60 years, while it was 50% in the younger group. The oldest survivor was 68.

The single most important factor determining the success or failure of the operation was an episode of cardiac arrest before surgical removal of the emboli. Sixteen of the 42 patients sustained an episode of arrest either in the ward, on the way to the operating theatre, or on the operating table. Ten of these patients died on the operating table, 5 survived to reach the ward but subsequently died (2 from causes unrelated to pulmonary embolism), and one survived. The mortality in this group was 93.75%.

In contrast to this there were 26 patients who reached the operating theatre without an episode of arrest and 25 of these survived surgery (96%). Seven of these died later. The overall mortality in this group was 30.8%. However, 4 of the late deaths were not attributable to pulmonary embolism but were from a variety of other causes connected with the patient's admission to hospital. If these are excluded 22 patients out of the 26 were successfully treated for pulmonary embolism, a success rate of 84.6%. Five patients underwent pulmonary embolectomy while cardiac massage was actually being performed. In 3 this was performed through a left thoracotomy; all were unsuccessful. In 2 the embolus was removed through a median sternotomy; one died subsequently from cerebral damage and one survived pulmonary embolectomy performed in the ward without an anaesthetic.

Of 6 patients who survived embolectomy but subsequently died from causes not directly related to pulmonary embolism 2 were in the group which sustained an episode of cardiac arrest before surgery and 4 reached the operating theatre without such an event. Two died from pulmonary infection, 2 from septicaemia associated with peritonitis, 1 had pre-existing renal failure, and in 1 severe haemorrhage from the large bowel, which necessitated a total colectomy, was eventually responsible for death.

Late deaths related to pulmonary embolism were due to a further massive embolism in 1 patient, to intractable right ventricular failure in 2 (both of these had episodes of cardiac arrest before operation), and to cerebral causes in 3, all of whom had episodes of arrest requiring cardiac massage, and brain damage was almost certainly sustained at this time.

In all patients the blood pressure was less than 100 mm Hg, but a successful outcome was achieved even though the blood pressure at the time of coming to the operating theatre was only 50 mm Hg. The interval between the onset of symptoms and operation did not influence the results as much as might have been expected,

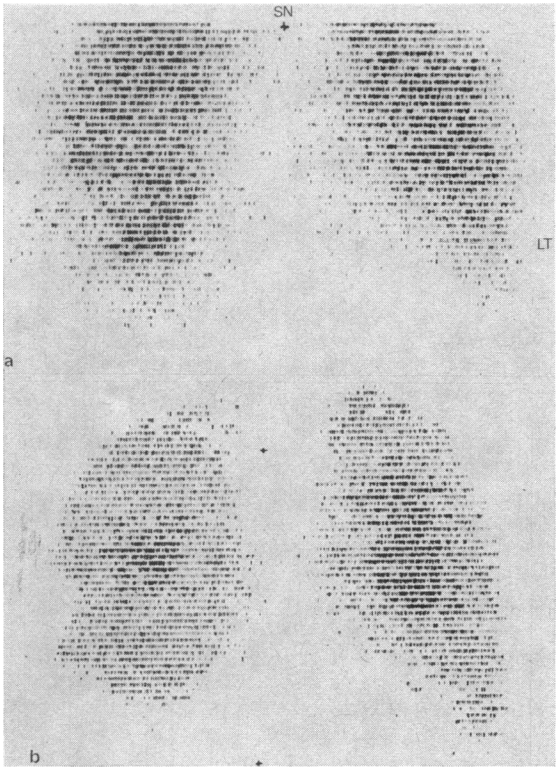

FIG. 4 *(a) Pulmonary isotope scan one week after pulmonary embolectomy; perfusion of the lower lobes is impaired. (b) Scan one month later showing improved pulmonary perfusion as residual emboli are absorbed.*

and the results obtained 1 h after the embolism were as good as those obtained 6 h or more later. Lack of success in patients operated on within 30 min of the embolism is attributable to the fact that most of these required cardiac massage before coming to theatre.

A peripatetic service has been offered to the Birmingham area. Half of the operations have been performed in the Queen Elizabeth Hospital and half in other hospitals where thoracic surgery is not usually performed. It is of interest that the success achieved in the latter hospitals, working with staff quite unfamiliar with the procedure, has been somewhat better than the results obtained in the thoracic surgical unit. This is partly because some of the latter patients were in the group who suffered cardiac arrest shortly after the embolism but also because our early experience of this operation was within our own unit and it was not until later that the service was taken to other centres.

If pulmonary embolectomy is to be available to a patient in any hospital in the British Isles, then a simple procedure of the type described would be appropriate. Many surgeons today have spent part of their training in a cardiothoracic surgical unit, where they have acquired confidence in handling the heart and the great vessels. Inflow occlusion pulmonary embolectomy is within the competence of most of them.

It is not claimed that this operation will remove every embolus from the pulmonary vascular bed, but in time most small residual emboli will become absorbed, as can be demonstrated by serial pulmonary isotope scans (Fig. 4). It would be dangerous to extrapolate from this and to argue that all pulmonary emboli are capable of resolution in time; such is not the case. In a study of 68 patients surviving massive embolism and followed up for a mean of 17.5 months 15% were found to be still severely disabled by breathlessness and 10 had had recurrent embolism. Of these severely disabled patients 80% had right ventricular hypertrophy and 20% had pulmonary hypertension. One-third of them subsequently died in cardiac failure *(10)*.

In contrast to this, patients surviving pulmonary embolectomy have had a remarkably smooth and short convalescence and they have been able to return to full physical activity within a short time. The only postoperative complaints have been of swelling of the leg in those patients who had undergone narrowing of the inferior vena cava.

Experimental work

The crucial role of cardiac arrest in determining whether the patient lives or dies has presented us with a question which to date remains unanswered. The clinical pattern is remarkably consistent. Even though emboli are successfully removed and the circulation restored, the overdistended right ventricle beats feebly or not at all despite the use of manual compression of the heart and the liberal use of inotropic drugs.

A small series of animal experiments was performed to test the hypothesis that right ventricular failure was due to distraction of the interleaved actin and myosin molecules in the cardiac muscle cell. Electron microscopy failed to demonstrate alterations in the striation pattern, which would have been expected if this mechanism was responsible.

The case against pulmonary embolectomy

There are several cogent arguments against pulmonary embolectomy. The mortality is considered to be unacceptably high, but this must be balanced against a reported mortality in untreated patients which may be as high as 75%.

In this series when surgery was performed before an episode of cardiac arrest had occurred 84.6% of patients were treated successfully for massive pulmonary embolism.

Inevitably the operation will acquire a bad reputation if surgery is delayed until the chances of success are remote. It would be more logical to offer pulmonary embolectomy to the less seriously ill patient in the anticipation that he would have a shorter and less complicated convalescence and that the risk of death from pulmonary embolism would be averted.

It has been said that if the patient survives to reach the operating theatre he would have lived without surgery. This may be true of the patient operated upon 24 h after pulmonary embolism, but it is not necessarily so of the patient operated upon 1–2 h after the catastrophe.

It has also been suggested that most patients who sustain massive pulmonary embolism have an underlying fatal disease and it is therefore not justifiable to treat them *(11)*. This has not been the case in this series. Only 4 of the 42 patients we have treated were suffering from malignant disease. All of these had been treated surgically with a reasonable expectation of worthwhile survival. Others had been treated for such minor conditions as varicose veins and hernia. Many had had surgery of the lower limb or pelvis.

The case for embolectomy on cardiopulmonary bypass

Most cardiothoracic surgeons favour the use of cardiopulmonary bypass for pulmonary embolectomy. We have elected to use a less popular technique which in Cross and Mowlem's survey *(5)* had been attended by a mortality of 87%.

Although the results we have achieved compare favourably with the 115 bypass pulmonary embolectomies reported by Cross and Mowlem, the series reported by Heimbacher *(12)*, Beall *(13)*, and Paneth have achieved better results. One of the largest reported series is that of Miller, Hall, and Paneth *(14)* from the Brompton Hospital. They too have done least well with those patients who sustained an episode of cardiac arrest but have achieved 50% survival in this group. This is attributable to the ability of cardiopulmonary bypass to provide support for the heart and enable it to recover. In 23 patients who were shocked 64% survival was achieved and in this group their results are similar to those obtained using inflow occlusion.

A protocol for patients sustaining massive pulmonary embolism has been devised in which treatment initially is with inotropic drugs and enzymes and embolectomy is reserved for those in whom enzyme treatment is shown to be ineffective or is contraindicated. The results obtained in patients treated with enzymes alone and in those treated by surgery are similar. This period of trial and appraisal has meant that patients are accepted for surgery rather later and further delay is occasioned by the time taken to assemble the necessary personnel and equipment to perform an open heart operation.

The mean time between the embolism and surgery in the Brompton Hospital series was 11.9 h. In the Birmingham series the mean time to surgery was 5.2 h, but many operations were performed within the first 2 h after the embolism, at which time the patient is at greatest risk of dying (Fig. 5).

The case for thrombolytic enzyme therapy

Extensive and detailed surveys have been carried out on the effectiveness of the enzymes streptokinase and urokinase *(15)*. It has been shown that these enzymes increase the rate of dissolution of thrombi in patients when studied after 24 h. However, some hours must elapse before effective resolution is achieved, and enzymes are not effective if the thrombus is older than 5 days. There are many contraindications and some complications. As yet there is no clear indication that the mortality of massive embolism is reduced.

The term 'massive pulmonary embolism' is often used rather loosely, but if we are to compare the results of surgery with those obtained by other means it is necessary to be rather more precise. In the streptokinase–urokinase

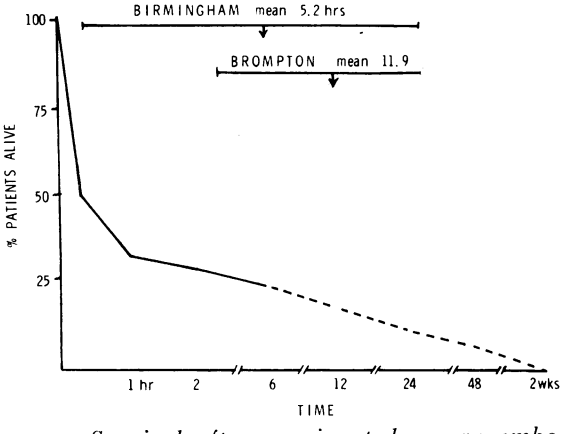

FIG. 5 *Survival after massive pulmonary embolism. The times at which pulmonary embolectomy was performed in the Brompton Hospital and Birmingham series is shown.*

trial performed in the United States *(15)* there were 167 patients who were stated to have suffered massive pulmonary embolism, but this was defined as occlusion of two or more lobar arteries and only 7% of the patients studied were shocked. In the Birmingham series all of the patients were shocked and it is known that for the cardiac output to be impaired two-thirds of the pulmonary vascular bed must be obstructed by emboli. We are comparing entirely different groups of patients and such comparisons are not valid.

The value of enzyme therapy has yet to be defined, but even its protagonists admit that there is a place for pulmonary embolectomy and list the necessary criteria which must be present before proceeding to operation. These include failure to resuscitate the patient, persistent hypotension, certain angiographic findings, evidence of a low cardiac output and hypoxia, evidence of right ventricular failure, and contraindications to thrombolytic enzyme therapy. This describes with accuracy the type of patient that we have accepted for surgery.

Conclusion

In conclusion I would reaffirm our belief that pulmonary embolectomy has a place in the management of massive pulmonary embolism. It is also our belief that the technique of inflow occlusion has many advantages. It can be performed rapidly and its simplicity should bring it within the competence of any surgeon with a basic training in thoracic surgery. It can be made available to the patient who is unfortunate enough to have a pulmonary embolism in a hospital without facilities for open heart surgery and the results obtained compare favourably with those obtained using more complicated techniques. Recovery from the emergency is short and the late complications are avoided.

In 1912 Friedrich Trendelenberg wrote: 'All new and difficult problems require much work, favourable conditions, and unfaltering confidence and perseverance. This holds good also for the operation for the pulmonary embolism. Twelve times we have done it at the clinic, my assistants oftener than myself, and not once with success, and yet I would continue trying'. Surely we can match the determination and vision of this with the resources available to us now.

I hope that our experience has convinced you that pulmonary embolectomy deserves to be reinstated in the surgical repertoire. We believe so and, like Trendelenberg, we shall continue trying.

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