CURRENT SURVEY

The surgical treatment of acute massive pulmonary embolism

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The effective management of massive pulmonary embolism remains a challenge. The successful treatment rests primarily upon early operative intervention and survival without surgery is extremely low. In the series of sixty-five patients of Hampson, Milne & Small (1961) all but nine died immediately. Donaldson et al. (1963) reviewing 271 cases of massive pulmonary embolism, proved at autopsy, revealed that 30% lived for 30 min, 25% for 1 hr, 15% for 6 hr and only 9% lived for 12 hr. There is a race against time and the character of the circulatory obstruction leaves little opportunity for effective treatment. Unless, therefore, a fast decision is made, only about one in five patients survives long enough for a proper diagnosis and surgical treatment.

Although Trendelenburg first proposed pulmonary embolectomy in 1908, Kirschner in 1924 performed the first successful operative procedure. Thereafter within a 7-year period, Crafoord (1928), Meyer (1930) and Nystrom (1930) recorded eight successful cases. Steenberg et al. in 1958 reported the first successful embolectomy in America. In the last few years many more successful embolectomies have been performed at many centres (Beall & Cooley, 1965; Makey & Bliss, 1966; Cross & Mowlem, 1967; Barraclough & Braimbridge, 1967; Paneth, 1967; Sautter, 1967). Among the many factors in the recent increase in success, the most important has been the use of cardiopulmonary bypass, first employed successfully in this context by Sharp in 1962.

Clinical manifestations

The clinical picture suggests a major cardiovascular catastrophe. It consists of sudden painless dyspnoea, collapse, restlessness, apprehension, pallor, mild cyanosis and sometimes chest pain. Significant physical findings are severe hypotension, tachycardia in most cases, poor circulation, distended neck veins, gallop rhythm with premature beats, an accentuated pulmonary second sound, sometimes a pulmonary diastolic murmur and, rarely, abdominal distention and epigastric tenderness due to hepatic congestion. If pulmonary infarction precedes or accompanies the massive embolism, pleural pain, haemoptysis and a pleural friction rub may also be present (Crane, 1966).

Diagnostic studies

The chest X-ray usually remains normal but may, as suggested by Hanelin & Eyler (1951) be of occasional value. However, Taplick, Haskin & Steinberg (1964) described significant changes among two-thirds of the patients with acute embolism. Increased prominence of the main pulmonary artery or one of its primary branches, unilateral elevation of the diaphragm (Daicoff, Rams & Moulder, 1966), increased radiolucency of the lung field due to loss of the peripheral vascular markings on the side of the occlusion and cardiac enlargement due to right ventricular dilatation (Timmis, 1966) have been the significant findings. Diameters of right and left pulmonary arteries greater than 17 and 16 mm, respectively in adult males and 16 and 15 mm in adult females are definitely abnormal radiological features (Davis, 1964).

The electrocardiogram may be entirely normal but often shows non-specific changes. Israel & Goldstein (1957) reported evidence of acute cor pulmonale in 70% of their cases. Right axis deviation, prolonged P–R interval, and depressed ST segments in lead I and II and inverted T waves in II and III are strongly suggestive of major pulmonary arterial obstruction. Other changes include RBBB, an S1Q3 pattern and inversion of T waves in right precordial leads.

Pulmonary radioactive scintiscanning is utilized in diagnosis (Sabiston, 1964; Wagner, Sabiston & McAfee, 1964; Sabiston & Wagner, 1965). Macroaggregated human albumin tagged with $^{131}$I is given intravenously and is immediately followed by scanning of the chest. Perfusion defects in the pulmonary embolism are
shown by the presence of cold areas in the normal pattern of the pulmonary silhouette. Though its safe application in the ill patient and the ease and speed with which it can be carried out makes it an excellent investigation, this has its own limitation. The cold areas do not specifically mean intraluminal pulmonary arterial obstruction but can also be seen in lesions that displace pulmonary tissue (tumour, bulla, cyst), lesions featuring consolidation (pneumonia, atelectasis) and situations slowing blood-flow as in congestive cardiac failure and pulmonary hypertension (Crane, 1966). Therefore a recent chest film preferably free of opacities is vital to a lung-scan interpretation.

Pulmonary angiography is the only certain method which affords a definite diagnosis. It is safe, pulmonary arterial and right ventricular pressures may be registered at the same time, and the severity of pulmonary arterial obstruction is most accurately assessed. The positive angiogram will show a filling defect due to intraluminal thrombus or clear ‘cut-off’ due to impacted thrombus (Fig. 1). It is negative in small multiple and finely fragmented embolism. In the presence of cardiac failure and pulmonary pathology causing increased pulmonary vascular resistance, the basal segments often appear oligaemic and third- and fourth-order vessels appear pruned off or vascular filling is delayed. Therefore, while reading an angiogram non-filling or pruning of small vessels should not be given undue emphasis unless previous pulmonary and cardiac pathology have been ruled out (Crane, 1966). Though a venous angiography employing a peripheral vein may be a success, pulmonary arterial angiography gives better delineation. While catheterizing the right heart for pulmonary arterial angiography, care should be taken not to advance far into the pulmonary artery as it may dislodge and fragment the embolus. For the same reason injection of contrast medium into the outflow tract of the right ventricle is preferable than into the pulmonary artery. In the presence of a failing circulation, angiography can be performed during a peripheral partial cardio-pulmonary by-pass. A successful angiogram can even be obtained when the

![Selective pulmonary angiogram performed prior to a successful embolectomy, showing catheter in the right ventricle and clear ‘cut-off’ of the left lower lobe artery due to an impacted embolus.](http://pmj.bmj.com/)

FIG. 1.
circulation can only be maintained by external cardiac compression (Gautam, 1968). The basic indication for angiography is to establish the diagnosis in those cases, considered for surgery. Not only is the diagnosis made certain but the side of the occlusion is known and if it is unilateral, as it is in 15% (Gorham, 1961), surgery can be carried out through unilateral thoracotomy even without the help of bypass (Bradley, Bennett & Lyons, 1964; Frater et al., 1966).

Indications for embolectomy

Obviously embolectomy is reserved for one who would die without it. But to decide which will live without it is also a difficult problem. As more successful procedures have been carried out, indications for embolectomy are now being made more clear. Initially the patient is heparinized, digitalized, and is given oxygen. If the hypotension is significant, a vasopressor is used. Isoprenaline, because of its antispasmodic action on the bronchial tree, is known to give a better result. Reflex bronchospasm due to embolism is relieved by heparin which is believed to antagonize the serotonin-like action of pulmonary embolism on the bronchial tree. Heparinization also stops the distal propagation of the thrombus.

A weakening pulse, falling blood pressure despite continuous pressor support, rising respiratory rate in spite of oxygen, increasing distension of neck veins, more pronounced gallop rhythm, increased right ventricular heave and accentuation of the pulmonary second sound, definite angiographic proof of the embolism and a raised pulmonary artery pressure the mean of which is greater than 30% of the mean systolic systemic arterial pressure (Diacoff, Rams & Moulder, 1966), are the indications for the surgical approach to the problem.

Technique

The prompt establishment of a cardio-pulmonary bypass is specific temporary therapy for the essential circulatory derangement of massive pulmonary embolism (Gibbon, 1937). It reduces the acute right heart strain and restores a vital flow of oxygenated blood to the coronary, cerebral and visceral circulations. If the patient's condition does not permit delay and severe right ventricular failure is soon evident, peripheral partial bypass is instituted under local anaesthesia. It may be established between jugular vein and iliac artery (Crane, 1966) or femoral artery and vein. Cross, Jones & Marolner (1964) and Cooley & Beall (1962) have reported an impressive rise in peripheral arterial pressure following the establishment of peripheral bypass in massive pulmonary embolism. Patients may be put on peripheral bypass in the ward using a portable pump oxygenator (Paneth, 1964; Beall & Cooley, 1965). The peripheral bypass also averts severe circulatory collapse and hypoxic myocardial damage (Vosschulte, 1958) secondary to induction of anaesthesia prior to the establishment of total bypass for pulmonary embolectomy (Sharp, 1962).

Sudden death from pulmonary embolism should be treated by external cardiac compression, for in some cases, the compression will break up the thrombus and move it on and so allow some pulmonary blood-flow. If the circulation is restored and the patient responds, then this is continued until the establishment of supportive peripheral bypass.

General anaesthesia is administered and the chest is opened by transverse or median sternotomy. If the peripheral bypass has not been established beforehand simultaneous exposure of the femoral artery is performed. Before cannulation, heparin (300 units/kg body weight) is administered. The superior and inferior venae cavae and femoral artery are cannulated and a total bypass begun (Fig. 2). Though it is quite possible to perform the operation using dextrose solution to prime the pump oxygenator (Dewall & Cooley, 1968).
& Lillehei, 1964; Perkins, Rolfs & McBride, 1964; Daicoff et al., 1966), blood replacement usually becomes necessary as the rapidly made incisions in a hypotensive, heparinized and vasoconstricted patient start to bleed as the circulatory state improves under the bypass.

The main pulmonary artery is opened longitudinally and emboli are removed from the main and right and left pulmonary arteries by using right-angled Dejardin common-bile-duct forceps or some other suitable instruments. It is important that efforts are made to remove the clots intact including their long peripheral tails.

The other measures assisting complete clearance of the pulmonary arterial tree are the use of a suction catheter down both pulmonary arteries combined with irrigation and as suggested by Cooley, Beall & Alexander (1961) bilateral intrapleural pulmonary massage from the periphery to the centre. Sometimes the occlusion of subsegmental tributaries can be cleared by combining the above procedures with retrograde flushing of the pulmonary arterial tree by injecting saline in temporarily clamped pulmonary veins (Timmis, 1966). Successful embolectomy should be followed by brisk back-bleeding of bright red blood. Before closing the pulmonary arteriotomy, the right ventricular cavity and the chordae tendineae therein are scrutinized for any trapped emboli and the abdomen compressed to retrieve any late loose clots. The bypass is discontinued, cannulae removed and protamine given.

The high incidence of recurrence following pulmonary embolectomy makes inferior vena cava plication (Fig. 3) or ligation below the renal veins mandatory as a part of the same operative procedure (Sharp, 1962; Spencer et al., 1962; Krause, Cranley & Hallaba, 1963; Stoney, Jacobs & Collins, 1963). If the patient’s condition permits it is preferable to precede embolectomy by plication (Stoney et al., 1963; Bradley et al., 1964). However, it should always be performed prior to the decannulations, for if recurrent embolization occurs while plicating the vena cava, clot can then be removed without any difficulty. In females, the ovarian veins may also be ligated (Timmis, 1966).

Though it is admitted that the use of cardio-pulmonary bypass offers the best chance of survival to a patient undergoing pulmonary embolectomy (Cooley et al., 1961; Paneth, 1967; Sauter, 1967), it is by no means the only available method. Without cardio-pulmonary bypass, successful embolectomies by the classical Trendelenburg procedure (Kirschner, 1924; Crafoord, 1928; Meyer, 1930; Steenberg et al., 1958) and under hypothermia (Allison, Dunhill & Marshall, 1960) have been performed. Unilateral pulmonary embolectomy can also safely be performed without the use of cardio-pulmonary bypass (Bradley et al., 1964; Frater et al., 1966). Pulmonary embolectomy using normal-thermic venous inflow occlusion is a reliable technique of great value in centres where facilities for providing bypass are not available. Vosschulte, Slitter & Eisenreich (1965) and Clarke (1968) were successful in four out of seven embolectomies using this technique.

Fig. 3. Spencer’s method of plicating the inferior vena cava.

The ideal treatment of acute massive embolism is to remove the obstruction as quickly as possible. The mortality of the operation is over 50% (Cross & Mowlem, 1967) and the precise indications for operation are difficult to define. Conservative treatment offers little help in acute massive embolism. However, the recent introduction of thrombolytic therapy in acute pulmonary embolism has proved of great value (Hirsh et al., 1967). The mortality and morbidity from carefully controlled streptokinase treatment should prove to be considerably lower than that from embolectomy. Moreover, thrombolytic therapy may dissolve surgically inaccessible small emboli which often accompany the large embolus and also the peripheral thrombus
from which the embolus is derived thus obviating the need for vena caval plication or ligation. On the other hand thrombolytic therapy may not produce its effect quickly enough in some patients who are dying from massive embolism. In these patients, embolectomy may be lifesaving. The place of thrombolytic therapy in the management has yet to be established.

Post-operative care

The post-operative care is that of an ill post-perfusion cardiac patient. Problems requiring specific attention are the consequence of pre-operative poor tissue perfusion due to low arterial and high venous pressure. These may be cerebral oedema, renal failure, hepatic insufficiency and cardiac failure. Even after a successful embolectomy as judged by an immediate post-operative angigram, there remains some respiratory insufficiency suggested by a low arterial Po2 in spite of high concentration of oxygen administration (Daicoff et al., 1966). This may be due to veno-arterial shunting, hyperventilation and faulty diffusion. As it may last for a few days, a tracheostomy and intermittent positive pressure ventilation may be indicated. To decrease the risk of thrombosis at the embolectomy site and to prevent further embolization, the patient is anticoagulated for about 6 months.

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References

Current survey


