Pulmonary Embolism

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There has been no change in the position in this country since Short wrote in 1952, "A careful watch in all departments of a general hospital combined with the free use of radiography showed thrombo-embolic disease of the lung to be the commonest of all serious acute pulmonary diseases." A high incidence among patients with fractures, heart disease, cancer, recent operations, and disease or trauma of the leg veins is recognized by all. To these must now be added the occasional case associated with oral contraceptives. Of great interest and possible importance is the reportedly low incidence of the disease in hospital patients in African countries. If it proves to be the case that there are some aetiological factors shared by venous thrombosis and occlusive arterial disease, then studies of regimens which protect against pulmonary embolism may provide some useful clues in other fields. Dietary factors should certainly be examined.

In the legs, the important complication of venous thrombosis is persisting swelling with the risk of troublesome ulceration of the skin. In the lung, pulmonary infarction causes fever and dyspnoea, and additional infection with abscess formation may threaten life. The principal risk lies in the sudden massive obstruction of pulmonary blood flow with hypotension, arterial hypoxia, and cardiac arrhythmia. Persisting pulmonary arterial obstruction, caused by repeated embolism, remains a rare disease, and its rarity seems likely to be due as much to the usually self-limiting nature of the disease as to good medical treatment of diagnosed cases.

Diagnosis is often most difficult in patients with established heart disease, for the common clinical features of pulmonary embolism may equally be due to worsening heart failure. Thus, sudden breathlessness, haemoptysis, raised venous pressure, basal râles, and arrhythmias are common to both conditions. Furthermore, a pleural effusion may defeat radiological distinction, and previous electrocardiographic abnormalities make the electrocardiographic diagnosis difficult or impossible. In

patients with left ventricular failure, therefore, the cardiologist will frequently be in the position of being unable to decide if pulmonary embolism has occurred. Under these circumstances it will usually be the case that anticoagulant treatment is called for on the double score of treatment of a probable attack and prevention of further episodes. In the routine examination of patients with myocardial infarction, frequent percussion and auscultation of the lung bases should not be neglected, and loss of resonance, or râles at one base, are common warnings of a pulmonary infarct which may well be symptomless. Unexpected low grade fever should also evoke the same suspicion.

Bedside radiography is invaluable to confirm the presence of a pulmonary lesion when physical signs are equivocal. At other times the distinction between acute left ventricular failure and pulmonary embolism will be clarified.

In patients with previously normal electrocardiograms the presence of the $S_1 Q_3 T_3$ pattern with clockwise rotation and inverted T waves in V2-3 provides the single, most conclusive evidence of massive pulmonary embolism. There will be no such help if the embolism is less massive or if previous abnormalities obscure the evidence. Atrial fibrillation or flutter are common accompaniments of both embolism and underlying disease.

Laboratory tests of serum levels of bilirubin, lactate dehydrogenase, or other enzymes are not to be relied on to clinch the differential diagnosis between pulmonary embolism and infarction on the one hand, or worsening heart failure or pulmonary infection on the other.

Pulmonary function tests may provide corroborative evidence. The characteristic abnormality is an increase in ventilated, unperfused lung (physiological dead-space), with a low expired CO_2 concentration and a high dead space to total ventilation ratio (V_D/V_T). The latter measurement, which can easily be estimated from the expired air and arterial blood CO_2 tensions, needs careful interpretation in the presence of a rapid, shallow respiratory pattern. These changes may, however, be

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masked by pre-existing ventilation-perfusion abnormalities, and, in addition, a massive embolus nearly blocking both pulmonary arteries may produce a relatively uniform diminution in perfusion, which is not detectable by this technique.

It is now easy and safe to record pulmonary artery pressure in ill patients by using a flow-guided fine polyethylene catheter. But such measurements are of limited value in assessing this clinical problem: first because pulmonary artery pressure may be grossly raised in left heart disease, and secondly because acute obstruction of the pulmonary tree may not be accompanied by very high levels of pulmonary artery pressure and death may occur without very severe pulmonary hypertension. Of interest is the fact that the modest pulmonary artery pressure rise may return to normal in a day or two, long before severe electrocardiographic changes have begun to resolve.

Specialized investigation can play an important part in assessing the difficult case. Pulmonary angiography gives the clearest evidence of localized obstruction to major vessels in patients found to have unexplained pulmonary hypertension. It is always worth while, for a few cases have been recorded when major clots have remained in the pulmonary artery for months, and surgical removal may then be attempted.

Radioisotope scanning with aggregated albumin is safe and helpful. It does not distinguish well between infarction and collapse but it may prove to be the most acceptable method to record the return of pulmonary blood flow as the clot is lysed, and it therefore provides a useful tool to assess the rival claims of different types of antithrombotic medication.

Simple prophylactic measures are difficult to evaluate. Earlier mobilization of patients with heart failure or myocardial infarction, which is now generally practised, is to be welcomed. So far as preventing pulmonary embolism is concerned, it is important that early mobilization should not consist of long periods of sitting in a chair. A short walk and then lying on the bed should be the rule. Raising the foot of the bed on 6-inch blocks has been shown to speed calf vein blood flow and should be instituted wherever suitable. Most patients sleep quite comfortably in this way.

Pride of place in prophylaxis, however, must go to medicaments which specifically inhibit clot formation. Anticoagulants should be used in a prophylactic way in the treatment of severe heart disease which will involve a prolonged period of immobilization. This is particularly apt in the situation discussed earlier when the distinction between worsening left heart failure and additional pulmonary em-

bolism cannot be made. There are a number of other circumstances, particularly a previous history of post-operative thromboembolism in a patient undergoing further surgery, in which prophylactic anticoagulant treatment may be wise. The recently reported antithrombotic action of infusions of dextran may be very helpful in surgical wards where the risk of bleeding is important and control of oral anticoagulant dosage may be a problem (Ahlberg *et al.*, 1968; Johnsson, Byggleman, and Eliasson, 1968).

Treatment of the overt attack should consist of heparinization together with other measures to support the circulation if life is threatened. Oxygen therapy is indicated, for arterial hypoxia, due to disturbances of ventilation-perfusion ratios, may be severe. Digitalization is correct, particularly in the presence of arrhythmia, and pressor amines may be indicated when severe hypotension threatens life. Electrocardiographic monitoring is desirable. Heparin is best administered by continuous intravenous infusion. In men 40,000 units and in women 28,000 units in 24 hours is usually effective and can safely be employed without laboratory control, but it is wise to check that the whole blood clotting time lies in the range 15–20 minutes. Continuous heparinization should be continued until the threat to life has receded. Oral anticoagulants are then begun in the knowledge that as long as one week may ensue before an adequate dose range is found. Warfarin is preferred to phenindione because there is a small risk of severe skin reactions and hepatic or renal damage when the latter is used. The control of oral anticoagulant therapy demands a measure of therapeutic skill and patience, which is often lacking, and failures of treatment are all too often due to clumsy use of the drug. The duration of treatment needs equally careful appraisal. A single episode of pulmonary infarction complicating a period of immobility may reasonably be treated for as short a period as three weeks, provided the patient is fully mobile by that time. Even if there have been no clinical signs in the legs, recurrence is possible after that time, and a second, longer period of treatment will then be necessary. In the presence of overt disease in the leg veins anticoagulant treatment is best continued for 2 or 3 months for recurrence is much more likely. When pulmonary embolism is complicating severe heart disease a long period of treatment is also necessary, as full mobility will be longer delayed. In a small number of otherwise healthy people treatment may have to be continued for many months or years or even for life, owing to further episodes occurring whenever treatment is discontinued. In all these circumstances the level of treatment should be high with the one-stage Quick prothrombin time at two-and-a-

half times control, or thrombotest between 5 per cent and 10 per cent. Occasional haematuria and epistaxes are inevitable in a group of patients at this level of treatment but they call for only temporary relaxation.

Heparinization discourages the addition of further thrombus to that already in the pulmonary artery, but there is no evidence that it has any thrombolytic action. The claims of other regimens which may promote clot lysis are, therefore, to be considered. Preparations of urokinase and streptokinase clearly enhance fibrinolytic activity. Until recently their usefulness was limited by the risk of producing fever and hypotension and it is clearly inadvisable for clinicians to forsake well-tried methods of treatment until the results of streptokinase treatment are more fully documented and dosages and methods of laboratory control clearly laid down. In the same way, it is clear that the new therapeutic agent "Arvin" rapidly produces a state of relative incoagulability without bleeding, and it may in future find widespread use.

Physical removal of large clots by surgical means has now been successfully achieved in many centres. Whether or not to recommend pulmonary embolectomy continues to be one of the physician's most difficult decisions. In the first place, the risk to life is relatively short lived, and even if the patient appears within a few heart beats of death, recovery usually takes place within a matter of days and seri-

ous, permanent sequelae are rare. It is clear, therefore, that pulmonary embolectomy should very rarely be undertaken except by cardiac surgeons with a fully experienced team. Where such help is available the decision needs to be made in a matter of hours. Continuous assessment of venous and arterial pressures and the electrocardiogram allow a well-based judgement as to whether or not clinical improvement is taking place. If, after a small number of hours, there is no improvement in the state of cardiogenic shock then embolectomy may be the correct procedure. There is still the hazard of an important operative mortality, and the likelihood that some clot may be very firmly adherent to peripheral vessels and may defy the attempts of repeated massage and suction to remove it.

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