Anaesthetic technique for transvenous pulmonary embolectomy

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Transvenous pulmonary catheter embolectomy is being used as a method of treatment for patients suffering from massive pulmonary embolism. The anaesthetic management of these patients can be complex. Presented is a case of transvenous pulmonary embolectomy in a patient who also had an intravascular volume deficit secondary to haemorrhage and possible reperfusion oedema. A discussion of appropriate monitoring during procedure follows.

Anaesthetists are often involved in the care of individuals suffering from unexpected sudden cardiac arrest. The rate of successful resuscitation of this group of patients is less than 50 per cent, with less than 15 per cent eventually being discharged from hospital.¹ Up to 25 per cent of patients with sudden death have suffered massive pulmonary embolism (PE).² This suggests a large population of patients with a treatable entity. Treatment has consisted of either medical³⁻⁵ or surgical therapy.^{6,7} More recently, catheter embolectomy has been described.^{8,9} The following is a case report of transvenous catheter pulmonary embolectomy performed after a massive pulmonary embolus. The case demonstrates the role the anaesthetist plays in the

management of these patients, as well as some important considerations for the conduct of anaesthesia.

Case report

A 66-year-old white male was admitted with a one week history of swelling and tenderness in the right leg. He had a history of non-insulin dependent diabetes mellitus and a left hemisphere cerebral vascular accident. His medications included tolazamide, hydrochlorothiazide, probanthine, and diazepam.

The physical examination was normal except for the presence of two ulcers on the right foot, a swollen right leg and right inguinal adenopathy. The arterial pulses (dorsalis pedis and popliteal) were decreased, and there was pitting oedema. Neurologic findings included aphasia, positive Broca's sign, right hemiparesis and positive right Babinski's sign. Laboratory values were normal except for a fasting blood glucose of 273 mg·dl⁻¹.

The patient was started on intravenous cephoxitin. A venogram demonstrated extensive acute venous thrombosis, involving the right popliteal, femoral and iliac veins. He was placed on streptokinase and was thrombolytic based on a prothrombin time greater than 1.2. (Thrombin time not done.) On the second day of admission when the patient was being moved for a bed change, he became unresponsive, with a short respiratory arrest followed by spontaneous restoration of breathing and an erratic pulse. The patient was tachypnoeic, tachycardic and cyanotic. Blood pressure was 70/40 mmHg and arterial blood gases (ABG) (FiO₂ 1.0) were: pH 7.41, PaO₂ 53, PaCO₂ 34, O₂Sat 88 per cent (total CO₂ 22, BE -1.9). The patient was intubated. Based on the clinical findings and past history, an acute PE was suspected. The patient was transferred to the angiography suite for

Key words

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an emergency pulmonary angiogram. Upon arrival a right radial artery catheter was placed (BP systolic was approximately 90 torr) along with an 18 gauge left peripheral intravenous catheter. ECG electrodes were placed to monitor lead II and a precordial stethoscope was placed. The patient was sedated with 5 mg of diazepam, 8 mg morphine and paralyzed with 3 mg of pancuronium intravenously. Mechanical ventilation was begun with a tidal volume of 800 cc, respiratory rate of 12 per minute and FiO_2 of 1.0. A percutaneous venous access sheath was placed in the left femoral vein after cutdown and angiography proceeded. Pulmonary angiography revealed a massive pulmonary embolism sparing only a portion of the right upper lobe pulmonary artery.

At the start of the procedure the patient's blood pressure was 100 torr systolic. The pulmonary artery pressure was 60/20. Following the start of the procedure the patient's blood pressure dropped to 80 torr systolic with no change in heart rate. Crystalloid was administered rapidly with little response and a dopamine infusion was started at a rate of $5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. Pulmonary artery pressures were still elevated (42/18). Fifteen minutes later, while embolectomy was being performed on the right and then the left inferior pulmonary arteries, the patient had an episode of ventricular tachycardia (presumed secondary to ventricular irritation from the embolectomy catheter). Sodium bicarbonate (89.2 mEq) was administered and cardioversion was performed with 300 joules. Sinus rhythm returned. Arterial blood gases at this time were: pH 7.20, PaO_2 447, PaCO_2 43, BE -8.0. Another 44.6 mEq of bicarbonate was administered.

During the procedure there were several more episodes of ventricular tachycardia and the patient was treated in the same fashion except that a lidocaine bolus, followed by an infusion, was administered. There were also several episodes of bradycardia (heart rate 30-40) associated with a drop in blood pressure to 60 torr systolic. These were unresponsive to two doses of atropine 1 mg and continued dopamine infusion. An epinephrine infusion was therefore started, with little response and subsequently an isoproterenol drip was begun. At this time the emboli had been removed through the suction apparatus. A pacemaker was inserted through the left antecubital fossa with capture and restoration of the heart rate to normal (70-80). The

blood pressure stabilized at approximately 100 torr systolic, immediately following removal of fresh embolus from both inferior pulmonary arteries. No obvious blood loss was appreciated. However, as a result of inability to maintain blood pressure and without the benefit of a pulmonary artery catheter at this time, 500 ml of five per cent albumin was rapidly infused with an increase in BP to approximately 110 torr.

It was evident as the drapes were being changed for insertion of a Greenfield filter, that there was a large pool of blood beneath the patient's lower extremities (volume estimate 1000 ml). Therefore 500 ml of packed red blood cells along with 500 ml of normal saline was infused. Blood pressure now stabilized at approximately 120 torr systolic, with sinus rhythm. Following placement of a pulmonary artery catheter, a Greenfield filter was inserted via the left femoral vein cutdown and the venotomy was closed. Pulmonary artery pressure was approximately 25/15. During the episodes of ventricular tachycardia and bradycardia with hypotension, only oxygen and pancuronium were administered to the patient. The patient was transported to the intensive care unit. Total fluids administered were: 2000 ml normal saline, 1000 ml D₅W, 500 ml five per cent albumin and 500 ml packed red blood cells.

The patient was extubated on the second post-operative day. He had a perioperative anterolateral myocardial infarction, but was discharged from the hospital following convalescence, and remained without long-term sequelae at one year.

Discussion

Approximately 630,000 hospitalized patients in the United States suffer a pulmonary embolus each year.¹⁰ Moreover, there are approximately five deaths per 1000 hospital admissions attributable to pulmonary embolism. A pulmonary embolus can be found in about 141,000 patients who die each year in the U.S.¹¹ Greenfield classified patients suffering from pulmonary embolism into two groups, based on physiologic parameters (Table). The first consists of Class I, II and III patients who can be managed medically. The second group includes Classes IV and V who have either a massive pulmonary embolus or a pulmonary embolus associated with pulmonary or myocardial disease¹² and demonstrate a very high morbidity and mortality

TABLE Classification of pulmonary thromboembolism

Class	Symptoms	Blood gas levels (mmHg)	Pulmonary artery occlusion pressure (mmHg)	Haemodynamics
I	None	Normal	<20	Normal
II	Anxiety, hyperventilation	PaO ₂ < 80 PaCO ₂ < 35	20–30	Tachycardia
III	Dyspnoea, collapse	PaO ₂ < 65 PaCO ₂ < 30	30–50	CVP elevated, $\overline{PA}$ > 20 mmHg
IV	Shock, dyspnoea	PaO ₂ < 50 PaCO ₂ < 30	>50	CVP elevated, $\overline{PA}$ > 25 mmHg
V	Dyspnoea, Syncope	PaO ₂ < 50 mmHg PaCO ₂ 30–40 mmHg	>50	BP < 100 mmHg PA 40 mmHg, CVP elevated, CO low, no shock

Abbreviations: PA, pulmonary artery; CVP, central venous pressure; $\overline{PA}$, mean pulmonary artery; BP, blood pressure; CO cardiac output.

From Stewart JP, Greenfield LJ. Transvenous vena caval filtration and pulmonary embolectomy. *Surgical Clinics of North America* 1982; 62: 411–30. With permission of the publisher.

(50 per cent dead in 30 minutes, 70 per cent in one hour, 85 per cent by six hours)^{13,14}

Several methods of treatment for acute PE have been described, including heparin therapy,^{10,15} streptokinase and surgery (open pulmonary embolectomy).^{6,7} In Class IV and V patients medical therapy has little to offer because of the associated high early mortality. Those patients who are treated in the acute situation with open embolectomy continue to demonstrate a significant morbidity and mortality (approaching 50 per cent).¹¹ As a result, the technique of transvenous pulmonary embolectomy has been advocated by Stewart and Greenfield.⁹

Indications for transvenous pulmonary embolectomy are similar to those for open pulmonary embolectomy, i.e., Classes IV and V patients with a strong clinical suspicion for pulmonary embolism. These patients, if identified at an early stage, could be saved by rapid pulmonary embolectomy.

The technique of transvenous pulmonary embolectomy has been described by Stewart and Greenfield.⁹ The catheter is steerable, measuring 100 cm in length with a suction cup at the end. It is inserted through the femoral venotomy to the right atrium and ventricle to the pulmonary outflow tracts. After arteriographic localization of the embolus, the distal cup is placed in close proximity to the embolus, and suction is applied. The embolus is then extracted via the femoral venotomy along with the catheter. Frequently, multiple attempts are

necessary to remove enough clot to achieve haemodynamic stability, as evidenced by recovery from shock and a significant improvement in the PaO₂. Complications of the procedure include ventricular arrhythmia, inability to extract the embolus, perforation of the pulmonary artery⁹ and fatal haemorrhage¹¹ following reperfusion of the lungs.

At the completion of the pulmonary embolectomy, a Greenfield filter is inserted through the femoral venotomy. According to Greenfield,⁹ indications for filter insertion include: recurrent pulmonary embolism despite adequate anticoagulation, pulmonary embolism with contraindication to anticoagulation; complications of anticoagulation; the Class V patient; following pulmonary embolectomy and septic pulmonary embolism. The filter is inserted via the femoral or jugular route over a guidewire using fluoroscopy. It is positioned in the inferior vena cava at the level of the third lumbar vertebra. Complications of Greenfield filters include: recurrent embolism; retroperitoneal haemorrhage, recurrent thrombophlebitis, peripheral oedema and filter migration.

The filter is inserted to protect against further embolization in an already haemodynamically compromised patient. Systemic anticoagulation should be continued following the conclusion of the procedure, as the filter protects against further embolization but does not treat the initiating problem, i.e., deep venous thrombosis.

Appropriate monitoring should include arterial

catheter placement for the second to second observation of blood pressure and to obtain ABG's as indicated. A large bore peripheral or central IV is indicated for rapid infusion of volume and/or vasoactive drugs. A pulmonary artery catheter should also be placed as early as possible both to determine pulmonary artery pressures and aid in evaluation of haemodynamic status. In the above described case, an 18 gauge catheter was the only one which could be placed peripherally and therefore the side arm of the catheter introducer sheath at the femoral cutdown site was utilized as our central venous access. Pulmonary artery pressure monitoring is desirable and the values are obtained from the angiographic catheter. After these catheters were removed a pulmonary artery catheter was placed through the internal jugular vein (since the insertion of a Greenfield filter via the femoral vein was indicated immediately after a pulmonary embolotomy). Monitoring should also include an ECG which should be able to give a lead II reading. In more than one third of patients with pulmonary embolism the ECG is normal, while other patients will show various transient abnormalities of the ST segment and T-wave in the precordial leads.¹⁶ However, tachycardia both supraventricular and ventricular, as well as bradycardia often result from pulmonary embolus manipulation and therefore a lead II or transoesophageal lead is best elected.

Anaesthetic management requires a motionless patient. Muscle paralysis with controlled ventilation was employed. If used alone, awareness might result, and therefore some form of sedation or general anaesthesia without cardiovascular depression is desirable. We opted for small doses of diazepam and a narcotic, which has been documented to be useful in patients with a compromised myocardium.¹⁷ The diazepam was utilized for its amnesic properties and its cardiovascular stability. Narcotic was utilized for suppression of pain associated with the procedure.

Pulmonary embolotomy can be associated with a significant loss of blood. The case described demonstrates that large amounts of blood loss may not be readily evident because of inaccessibility to the patient's lower extremity. These patients will generally be on an anticoagulant or thrombolytic agent and therefore may bleed profusely from the cutdown site. Bleeding secondary to heparin can be treated with protamine and that secondary to strep-

tokinase may respond to treatment with epsilon aminocaproic acid.

There appear to be two main periods when large volume infusions are necessary. The first is at the time the embolus is removed, with reperfusion and its associated complications. The second is when the Greenfield filter is being inserted, due to the surgical incision and bleeding in an anticoagulated patient. In our patient, there appears to have been a significant decrease in intravascular volume after embolus removal, requiring a rapid infusion of 500 ml five per cent albumin. (In two subsequent patients we have also found this to be the case.) Therefore one must especially consider volume infusion as these critical points are approached during the procedure. Early placement of a pulmonary artery catheter after embolus retrieval is highly desirable and advocated.

Conclusion

The anaesthetist must be prepared to provide careful monitoring of cardiovascular and respiratory parameters, sedation and perhaps paralysis, fluid and blood administration and further resuscitation efforts. All this may be necessary in a remote location. Therefore advance preparation is essential, as well as an understanding of the haemodynamic changes associated with acute pulmonary embolism.

References

- 1 *Bedell SE, Delbanco TL, Cook EF, Epstein FH.* Survival after cardiopulmonary resuscitation in the hospital. *N Engl J Med* 1983; 309: 569-76.
- 2 *Morrell MT, Dunnell MS.* The post-mortem incidence of pulmonary embolism in a hospital population. *Br J Surg* 1968; 55: 347-52.
- 3 *Hirsh J, McDonald IG, Hale GA, O'Sullivan EF, Jelinek SM.* Comparison of the effects of streptokinase and heparin on the early rate of resolution of major pulmonary embolism. *Can Med Assoc J* 1971; 104: 488-91.
- 4 *Tibbitt DA, Davies JA, Anderson JN et al.* Comparison by controlled clinical trial of streptokinase and heparin in treatment of life-threatening pulmonary embolism. *Br Med J* 1974; 1: 343-7.
- 5 *Miller GAH, Hall RJC, Pareth M.* Pulmonary embolotomy, heparin, and streptokinase: Their place in the treatment of acute massive pulmonary embolism. *Am Heart J* 1977; 93: 568-74.

- 6 Sharp EH. Pulmonary embolectomy: successful removal of a massive pulmonary embolus with the support of cardiopulmonary bypass – case report. *Ann Surg* 1962; 156: 1–4.
- 7 Glassford DM, Alford WC, Burrus GR, Stoney WS, Thomas CS. Pulmonary embolectomy. *Ann Thorac Surg* 1981; 32: 28–32.
- 8 Greenfield LJ, Kimmell GO, McCurdy WC. Transvenous removal of pulmonary emboli by vacuum-cup catheter technique. *J Surg Res* 1969; 9: 347–52.
- 9 Stewart JR, Greenfield LJ. Transvenous vena caval filtration and pulmonary embolectomy. *Surg Clin N Am* 1982; 62: 411–30.
- 10 Dalen JE, Alpert JS. Natural history of pulmonary embolism probe. *Cardiovasc Dis* 1975; 17: 259–70.
- 11 Mattox KL, Feldman RW, Beall AC, DeBakey ME. Pulmonary embolectomy for acute massive pulmonary embolism. *Ann Surg* 1982; 195: 726–31.
- 12 Greenfield LJ. Pulmonary embolism: diagnosis and management. *Curr Probl Surg* 1976; 13: 1–52.
- 13 Gorham LW. A study of pulmonary embolism. *Arch Intern Med* 1961; 108: 76–90.
- 14 Soloff LA, Rodman T. Acute pulmonary embolism. *Am Heart J* 1967; 74: 829–47.
- 15 McDonald IG, Hirsh J, Hale GS. Early nature of resolution of major pulmonary embolism. A study of angiographic and hemodynamic changes occurring during the first 24–48 hours. *Br Heart J* 1971; 33: 432–7.
- 16 Bell, William R. Pulmonary embolism. The principles and practice of medicine. Eds. Harvey AM, Richards JJ, Mekusick VA, Owen AN, Ross RS, New York: Appleton-Century Croft. 1880: 277.
- 17 Kaplan JA, Dunbar RW. Anesthesia for noncardiac patients with cardiac disease. In: Kaplan JA ed. *Cardiac anesthesia*. New York: Grune and Stratton, 1979: 385.

Résumé

L'embolectomie pulmonaire par cathéter transveineux a été décrite comme méthode de traitement pour l'embolie pulmonaire massive. La conduite anesthésique de ces patients peut être complexe. On présente ici le cas d'une embolectomie pulmonaire par cathéter transveineux chez un patient présentant aussi une hypovolémie secondaire à une hémorragie et aussi possiblement un oedème de reperfusion. Cette présentation est suivie d'une discussion des moyens appropriés de surveillance physiologique lors de cette procédure.