MASSIVE INTRAOPERATIVE PULMONARY EMBOLISM IN A CHILD

N. W. GOODMAN AND M. J. FALKNER

Pulmonary embolism is rare in children. However, it can occur, as in adults, in the postoperative period—especially after operations to correct scoliosis (Behrman and Vaughan, 1983). Other general risk factors include: severe dehydration caused by diarrhoea, cyanotic heart disease, and ventriculo–atrial shunts for hydrocephalus (Buck et al., 1981). We report a case of pulmonary embolism occurring in an 11-yr-old child during an operation to insert an artificial bladder sphincter.

CASE REPORT

An 11-yr-old girl, weighing 37 kg, was admitted for the elective insertion of an artificial bladder sphincter. She had had a sacral myelomeningocele diagnosed at birth. Later she developed hydrocephalus secondary to an Arnold–Chiari malformation and a ventriculo–atrial valve was inserted initially. This was replaced with a ventriculo–peritoneal shunt 3 years before the current admission. She had developed normally and was doing well at school. She had had two orthopaedic operations and was fully ambulant. Her major disabilities were incontinence of urine and faeces.

At the time of admission, the patient was well. There were no new abnormal findings on physical examination, and the results of laboratory investigations were normal. There was no reason to request a preoperative electrocardiogram or chest radiograph. Her mother knew of no problems that had occurred during any of her previous anaesthetics. The child was taking nitrofurantoin, propanthine and codeine phosphate, and imipramine 25 mg at night. Sodium picosulphate (Picolax, Nordic) was given for bowel preparation.

SUMMARY

An 11-yr-old girl suffered a massive pulmonary embolus during an operation to insert an artificial bladder sphincter. Forty minutes after the induction of anaesthesia, the heart rate increased and she became hypotensive and deeply cyanosed. A definitive diagnosis was not made during surgery, but she improved after 45 min of resuscitation with 100% oxygen, infusion of normal saline, ephedrine (total 45 mg) and intermittent external cardiac massage. The diagnosis was proved the next day by lung perfusion scan. There were no apparent risk factors.

Temazepam was given 10 mg orally 90 min before the induction of anaesthesia, during which fentanyl 100 µg, thiopentone 150 mg, pancuronium 4 mg, 66% nitrous oxide in oxygen and 0.6% ethrane were given. The lungs were ventilated through a 7-mm tracheal tube by a Manley ventilator (tidal volume 400 ml, peak airway pressure 24 cm H₂O) using a fresh gas flow of 4.5 litre min⁻¹. Gentamicin 80 mg and metronidazole 500 mg were given at the request of the surgeon. The patient was put in the lithotomy position with a slight head-down tilt and surgery started, and monitoring (electrocardiograph (CM5 position) and an automatic arterial pressure recorder (Dinamap 845)) was established.

The procedure was uneventful for 40 min. The heart rate was about 120 beat min⁻¹ and systolic arterial pressure between 105 and 115 mm Hg. The surgeon had made a longitudinal incision in the lower abdomen and was mobilizing the bowel and bladder, having seen the ventriculo–peritoneal shunt lying free in the abdominal cavity.

The first indication of something untoward was that the heart rate increased to 135 beat min⁻¹, and the arterial pressure decreased to 95 mm Hg.
This responded initially to infusion of normal saline, but during the next 10 min the heart rate increased gradually to 180 beat min⁻¹, the systolic arterial pressure decreased to 45 mm Hg and the patient became sweaty. The surgeon was informed, the operation abandoned, and resuscitation started. There was good air entry over both sides of the chest, and at this stage she was pink and well perfused peripherally. The inspired oxygen was increased to 100% and 750 ml of normal saline was given rapidly. Despite this the systolic pressure remained at 45 mm Hg, she soon became cyanosed, and her pupils started to dilate. During the next 60 min arterial pressure was supported by injections of ephedrine 3-6 mg (to a total of 45 mg), and intermittently she was given chest compression. The impression was that these measures, particularly the compression, decreased the cyanosis temporarily and certainly decreased the pupillary dilatation. An arterial blood sample, taken during a period of deep cyanosis on 100% oxygen, showed a $P_{a\text{O}_2}$ of 4.1 kPa, a $P_{a\text{CO}_2}$ of 4.5 kPa, with a base excess of $-5$ mmol litre⁻¹. Throughout this period a vigorous heartbeat was palpable and peripheral perfusion was maintained. The cyanosis was not simply a result of low arterial pressure, for there were periods when the patient was cyanosed despite a systolic arterial pressure of greater than 70 mm Hg with a good radial pulse. Eventually, her colour began to improve and her arterial pressure stabilized. Two hours after the initial incident, she was transferred to the intensive care unit. She had already made ventilatory efforts, and had responded to her name. At that time a definitive diagnosis had not been made, and so neuromuscular function was allowed to return spontaneously. Neostigmine was not given. The trachea was extubated without difficulty and there was by then nothing abnormal on physical examination except for a persistent tachycardia of 130 beat min⁻¹. However, a 12-lead electrocardiogram showed signs of right-heart strain which resolved over the next 6 h. A tentative diagnosis of pulmonary embolism or other cause of acute pulmonary vascular obstruction was made. She breathed oxygen at 4 litre min⁻¹ overnight and remained a good colour. We did not take any further arterial samples.

The presumed diagnosis was confirmed the next day by a lung perfusion scan. There was minimal perfusion of the left lung, with some smaller defects of perfusion of the right lung. As there was no significant abnormality on the chest radiograph, the diagnosis was clearly a massive pulmonary embolus.

The patient was transferred from the intensive care unit to the care of the paediatricians, and anticoagulated. Heparin 30000 units per 24 h by infusion gave a satisfactorily prolonged partial thromboplastin time. Warfarin 5 mg was given on the 1st and 2nd days after operation and by the 3rd day her prothrombin ratio was increased such that the heparin could be stopped. There were two small episodes of melaena on the 4th day after operation without any cardiovascular disturbance. As the haemoglobin had decreased from 10.1 g dl⁻¹ on the 3rd day after operation to 8.5 g dl⁻¹, two units of blood were transfused. Her only other postoperative problem was a cough productive of clear sputum.

On the 7th day after operation, the patient was discharged home, well in herself, and taking warfarin 3 mg daily. This was later increased by her general practitioner to 4 mg. A repeat lung perfusion scan 7 weeks after the incident was normal.

It was decided to maintain anticoagulation for 6 months in total. No decision has been made yet on the possibility of further surgery.

**DISCUSSION**

Although thromboembolic phenomena are a common postoperative complication in adults, they are rare in children. Even in adults they have only rarely been diagnosed during the operation, and then there had usually been an understandable aetiology. In two patients the embolism occurred during surgery for resection of abdominal aortic aneurysm (Mangano, 1980; Hecker and Lynch, 1983): one was a patient with polycythaemia (Enright, Quartey and McQueen, 1980) and another was obese, hypertensive and had been immobilized for 1 month before presenting for surgery (Divekar, Kamdar and Pansare, 1981).

We could find no report of true thromboembolism occurring during an operation in a child who had no apparent predisposing or operative cause. There has been a report of embolism, probably of marrow or air, during the insertion of an orthopaedic nail in a child with osteogenesis imperfecta (Fosel et al., 1985) and there are reports of intraoperative tumour embolism in children (Akyon and Arslan, 1981; Dorman, Sumner and Spitz, 1985). Since they are chronically constipated, patients with spina bifida need
more rigorous bowel preparation for surgery than do normal patients. Our patient had had some diarrhoea during bowel preparation, but this was not thought to have caused significant dehydration. Clinically, she was perfectly well on the morning of operation, and the induction of anaesthesia had no untoward effect. In any case, Buck’s group concluded from their survey of postmortems that dehydration was not by itself a major factor (Buck et al., 1981). Venous thrombosis could occur in a patient immobilized secondary to spina bifida, but this girl was fully mobile. If she had still had a ventriculo-atrial valve in situ, that would have been a risk factor, but it had been replaced 3 years earlier by a ventriculo-peritoneal shunt.

There was no family history of unusual thromboembolic phenomena. It may be that she has a biochemical fibrinolytic defect, but investigation of that will have to wait until anticoagulation has been stopped.*

In retrospect, the signs noted during the operation were exactly what one would expect with a massive pulmonary embolus—with the exception, perhaps, that the event was not sudden and catastrophic, but developed over 10 min or so. In view of the patient concerned, it is not surprising that the diagnosis was not made at the time. In one of the adult patients the diagnosis was not made in the operating theatre, but only later when the event recurred on the intensive care unit (Enright, Quartey and McQueen, 1980). Earlier diagnosis would not have altered the outcome. We would still have given a vasopressor, fluid i.v. and cardiac massage; embolectomy was unrealistic.

We have no evidence as to the origin of the embolus. The evolution of the changes in the cardiovascular system could have been the result of more than one embolus, or because the initial changes were caused by a clot in the right ventricle which moved into the pulmonary arteries a few minutes later. Presumably the treatment, particularly the chest compression, then dislodged the clot, mainly into the left lung, and the patient began to improve.

She was profoundly hypoxaemic, far more so than another patient who had a similar systolic arterial pressure (Mangano, 1980). The causes of hypoxaemia after pulmonary embolism are complex. Huet’s group concluded that early hypoxaemia is caused by ventilation-perfusion imbalance, rather than by intrapulmonary shunting (Huet et al., 1985). They could not completely rule out the opening of a patent foramen ovale in their patients, and nor can we; but at no time could we hear a cardiac murmur.

“It is clear...that pulmonary embolism, although rare, should be seriously considered when unexplained hypoxia occurs during operation” (Enright, Quartey and McQueen, 1980). We would support that, although it must come at the bottom of a long list in a fit, healthy, mobile child. Nonetheless, “when you have eliminated the impossible, whatever remains, however improbable, must be the truth” (Conan Doyle).

ACKNOWLEDGEMENTS

There are a number of clinicians in Southmead Hospital whom we would like to thank for their help and advice during this case: Dr C. R. Hall (Consultant Anaesthetist), Mr Paul Abrams (Consultant Urologist), Dr M. McGraw (Consultant Paediatric Nephrologist), Dr P. Walker (Consultant Physician) and Dr A. E. Mitchelmore (Consultant Radiologist).

REFERENCES


*Note added in proof: No clotting abnormality was demonstrated. Routine tests were normal, as were protein C, anti-thrombin III, euglobin clot lysis and plasmin activator.