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Air embolism in hip surgery

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Summary

The hearts of 49 patients undergoing hip-replacement surgery were auscultated and the characteristic sounds of air emboli were heard in 15 of them—30% of the total. This finding was confirmed in the later patients by the use of an ultrasonic transducer to detect air emboli.

Routine auscultation of the heart during the insertion of acrylic bone cement and prosthesis is recommended.

Key words

Embolism; air.
Surgery; orthopaedic, hip replacement.

There has been much discussion about the causes of cardiovascular collapse occasionally seen in patients undergoing total hip replacement surgery. Toxic reactions to methylmethacrylate bone cement and also fat embolism have received much attention. Air embolism has been suggested as a factor by several authors[1–3] but no study appears to have been attempted to assess its frequency. A chance observation while auscultating the heart during cement insertion led to the present study and the use of a Doppler ultrasonic flow transducer to detect air embolism and to estimate its frequency.

Methods

After informed consent, 49 consecutive patients anaesthetised by the author were studied during hip replacement. The patients were mainly elderly, in the age range 51–84 years (mean 65 years). There were 29 females and 20 males of mean weight 67 kg. Many had minor pathology in other systems, but only one or two were considered bad anaesthetic risks. Mild hypertension and ischaemic vascular disease were not uncommon findings and chronic bronchitis, diabetes and steroid-treated rheumatoid arthritis were also seen. Two patients feature twice since they had replacements of both hips and some others were having revision of an existing Charnley or Thompson prosthesis. All patients were premedicated with an opiate and an antisialogogue or with diazepam.

Anaesthetic technique

Twenty-six patients were anaesthetised with a lumbar extradural injection of bupivacaine

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followed by spontaneous breathing of a volatile agent and twenty-two were managed with a relaxant and intermittent positive pressure ventilation. One patient had a spinal anaesthetic.

The choice of anaesthetic agent was frequently influenced by the inclusion of some of these patients in other studies; for example, in some cases halothane was specifically avoided while in others halothane or buprenorphine were deliberately given.

Radial artery cannulation was performed and arterial blood pressure was recorded on a Simonsen and Weel transducer and a Devices 4-channel recorder. The electrocardiograph (ECG) was obtained from chest leads.

Surgical technique

The surgery was carried out through an anterolateral approach (except in one instance) and a Charnley prosthesis was inserted with CMW or Simplex methylmethacrylate bone cement. All the femoral shafts were vented with Redi-Vac polythene tubing during insertion.

Detection of air emboli

In the earlier patients the heart was auscultated with an oesophageal stethoscope from just before the insertion of the cement into the acetabulum until about 15 min after the insertion of the femoral cement. In the later patients an ultrasonic transducer pick-up was applied to the anterior chest wall and taped into position in the 2nd or 3rd right intercostal space beside the sternum. This was difficult to place accurately because the sonic beam is very narrow and, since the pick-up needed a heavy coating of sonic jelly, it was also hard to secure. Consequently the pick-up was inserted into a soft Portex stomach tube with a small window cut into it, producing a flexible blunt-ended tube of elliptical cross-section about 1·5 cm maximum diameter. This was then lubricated and inserted into the oesophagus. Positioning to pick up sounds from the right atrium or ventricle was then much easier and fixation was only necessary at the mouth. Monitoring was then continued for a similar period on the Devices recorder.

Dog study

Before the patient study the transducer was used on a dog to assess its sensitivity in the detection of small air emboli. A 30 kg dog, anaesthetised with nitrous oxide, oxygen and halothane, had been used for another study during which it had been found to have a cardiac output of about 2 litres/min. Small amounts of air from 0·25 to 2 ml were injected into the superior vena cava and then from 0·5 to 20 ml into the femoral vein, during continuous recording from the transducer placed over the pulmonary artery or right ventricle in the open thorax.

Results

General characteristics of air emboli. With a stethoscope, small amounts of air in the heart are heard as extraneous tinkling or 'plip-plopping' noises in time with the heart sounds. With the ultrasonic detector, air bubbles produce loud chirping sounds which appear on the trace as marked deviations from the regular pattern of normal heart blood flow.

Results in dog. In the dog study, with the probe over the pulmonary artery, 38 ml of air were injected before the flow sounds began to diminish. No 'chirps' were heard. However, when the sensor was moved to lie over the right ventricle chirps were plainly audible and visible on the trace after 2 ml increments. Smaller quantities were not used when monitoring over the right ventricle.

Results in patients. In the 18 patients in whom an oesophageal stethoscope was the only available method used for the detection of air emboli, seven cases showed positive signs. In a further 31 patients, both the stethoscope and the transducer were used simultaneously and air was detected in a further eight cases. Thus there was an overall incidence of air emboli of 30·6% in 49 patients.

When both methods were used, air was detected with a stethoscope on two occasions when the transducer failed to indicate it (Table 1). On a third occasion air was detected by the transducer (placed in the oesophagus) and not by the stethoscope on the chest wall. In a further two patients transient tinkling noises were heard but these were counted as negative. Thus in 15 patients out of a total of 49 studied definite sounds of air in the right ventricle were heard. In two of these, who both had pronounced and prolonged air sounds, there was an accompanying marked fall in blood pressure, systolic pressure falling from 140 to 70 mmHg in one and from 90 to 55 mmHg in another. Interestingly, these two patients were the only ones in whom air sounds

Table 1. Comparison of detection of air emboli by each method

	Oesophageal stethoscope alone	Oesophageal stethoscope +	Chest wall transducer	Chest wall stethoscope +	Oesophageal transducer	Total all methods
Total studied	18	17		14		49
Cases of air emboli		1	1	4	4	15
as detected by				1	0	
each method	7	1	0	0	1	

were heard after the insertion of both acetabular and femoral cement. The femoral insertion caused louder and more prolonged sounds in both of them. Usually the noises occurred from about 30 seconds after the prosthesis was hammered into the femoral canal and persisted until about 10 min later.

There appeared to be no significant correlation between incidence of detected air emboli and the age, sex or size of the patient. It might be anticipated that the half-left lateral position might give some protection against the effects of emboli. There do appear to be fewer emboli in patients having operations on the right hip, but this is not statistically significant. ($p > 0{\cdot}1$ that the incidence is the same.)

Previous exposure to bone cement. Sensitization to bone cement by previous exposure has been suggested as a cause of cardiovascular collapse. In this series, seven patients had previously had insertions of methylmethacrylate cement. Air emboli were heard in two of these and one of them had a large fall in blood pressure associated with profound changes in heart sounds. The other five had either no changes in blood pressure or a small decrease in systolic pressure of up to 20 mmHg after cement insertion.

Mode of anaesthesia. The anaesthetic used is shown in Table 2.

The numbers were fairly evenly divided between extradural anaesthesia with spontaneous breathing of a volatile agent and a relaxant technique with intermittent positive pressure ventilation.

Previous pathology. Several of the patients had diseases of other systems severe enough to give symptoms. Thirteen were hypertensive and a further five had ischaemic heart disease. There was no correlation between the incidence of air embolus and previous pathology.

Discussion

There can be little doubt among anaesthetists that joint-replacement surgery is occasionally associated with sudden and sometimes disastrous cardiovascular collapse. In hip replacement this most commonly occurs shortly after the insertion of the bone cement into the femoral canal.

Whereas there has been much discussion about the possible toxic reactions to methylmonoacrylic monomer causing cardiovascular collapse, venous air embolism has been mentioned in this discussion but appears to have received little attention. Hyland & Robbins[1] report one fatal case in which fat and air were found in the ipsilateral pelvic veins, the right side of the heart and the coronary vessels at post-mortem. This patient had marked myocardial degeneration and the cause of death was ascribed to air embolism act-

Table 2. Method of anaesthesia

Anaesthetic	No air detected	Air detected	Incidence of detection (%)
Extradural 26	·16	10	38·5
IPPV+relaxant 22	17	5	22·7
Spinal 1	1	0	0
Total 49	34	15	30·6

The incidence of air embolus with extradural or IPPV anaesthesia does not differ at the 5% level of significance.

ing on a previously defective myocardium. Ngai *et al.*[4] describe three cases in which millwheel murmurs were heard after the insertion of the femoral prosthesis.

Factors favouring air embolism

Theoretically, the insertion of methylmethacrylate cement into a reamed femoral canal would seem to provide ideal conditions for air embolism.

The sinusoids of the bone marrow must be opened by reaming. The venous sinusoids of marrow have very thin walls, in the largest sinusoids the walls are only three or four cells thick and muscular elements appear to be absent.[5] Therefore the damaged sinusoid will be unable to contract and may remain open. The sinusoids connect directly with the extra-osseous veins. The cement is then forced into the femoral canal and the prosthesis hammered in. Despite venting (with a tube at most a few millimetres in internal diameter) intramedullary pressure must rise enormously. The heat then generated by the setting cement, which possibly raises its temperature to 96°C,[6] will further raise the pressure of any air trapped in the canal by up to 20%.

Under nitrous oxide anaesthesia the size of an air bubble in the blood will also tend to increase.[7] Munsen & Merrick[8] found a threefold decrease in the median lethal dose of air injected into rabbits anaesthetised with 75% nitrous oxide.

Evidence from experimental studies

In 1973 Pelling & Butterworth[9] studied the cardiovascular responses to the forceful injection of bone cement, plasticine and paraffin wax into the medullary cavity of the femur in rabbits and cats. In six of their rabbits the blood from the femoral vein of the treated limb was conveyed by a length of polythene tubing to the contralateral femoral vein. In all of these, immediately after plasticine had been pushed into the femur, pale material (later identified as fat and marrow cells) was seen in the polythene tubing and, in two animals, air bubbles were also visible.

Sato[10] raised the femoral intramedullary pressure in cats by hammering on bone cement inserted into the medullary cavity and by introducing air into the cavity from a blood pressure cuff and manometer system, raising the pressure by 100 mmHg increments to 300 mmHg. In both groups they recorded hypotension and ECG changes. At autopsy air was found in the popliteal vein and pulmonary artery.

It is therefore hardly surprising that small air bubbles were heard in the right side of the heart in 30% of this series. These were probably only small amounts of air, of the order of a few millilitres, and 13 of these 15 patients gave no sign of any disturbance of the ECG or arterial pressure trace.

Sensitivity of sensors

Four millilitres was the smallest quantity of air heard with a precordial stethoscope by Maroon *et al.*,[11] although smaller amounts were audible with an oesophageal stethoscope. As little as 0·12 ml of intracardiac air could be heard with the transcutaneous Doppler. Edmonds-Seal *et al.*[12] found the Doppler ultrasonic flow transducer to be the most sensitive method of detecting air emboli in dogs, and found that with nitrous oxide/oxygen anaesthesia 0·025 ml of air became audible.

In the present study the ultrasonic transducer, while allowing the air artefacts to be recorded, gave very similar results to those with an oesophageal stethoscope, which was as efficient as the transducer on the chest wall and in one case more so (see Table 1). However, when the positions were reversed, the transducer in the oesophagus picked up air sounds on five out of six occasions, the same number as a precordial stethoscope.

Consequently it seems that a simple, readily accessible piece of equipment, an oesophageal stethoscope, is as efficient at detecting emboli during surgery as a costly ultrasonic transducer. It is certainly easier to use, safe with diathermy and free of the troublesome noise of artefacts that occur with the ultrasonic transducer.

Conclusion

From this small study it appears that clinically detectable air emboli occur in about 30% of patients undergoing total hip arthroplasty, usually just after the insertion of the femoral cement. In two of these patients marked falls in blood pressure coincided with pronounced heart sound changes. It would seem sensible as suggested by Hyland & Robbins in 1970[1] to use an oesophageal stethoscope and possibly a central venous pressure line in patients undergoing hip replacement. Prompt removal of nitrous oxide, positioning of the patient and aspiration of intracardiac air could save life if the possibility of air embolus is considered where there is intra-operative collapse.

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