AIR EMBOLISM DURING NEUROSURGERY IN THE SITTING POSITION

Two Case Reports

BY

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SUMMARY

Two patients undergoing posterior fossa exploration in the sitting position for removal of foramen magnum meningiomata suffered circulatory arrest from air embolism. Both were successfully resuscitated. The mechanism of air embolism is discussed with a review of relevant literature. The disadvantages of spontaneous ventilation are emphasized and it is recommended that controlled positive-pressure ventilation should be employed. The means of maintaining a safe venous pressure and other precautions to reduce the incidence of air embolism are discussed. The monitoring techniques available and the treatment of this accident are evaluated.

The sitting position for posterior fossa exploration, by virtue of good surgical access and excellent operating conditions, has become standard in many centres throughout the world. However the disadvantages of circulatory instability and the risk of air embolism still prevent its general acceptance. Neurosurgical units also differ one from another in the use of controlled or spontaneous ventilation when the sitting position is chosen (Galloon, 1959; Hunter, 1962; Marrubini, 1965; Michenfelder et al., 1966). This paper is concerned with the general management of anaesthesia in the sitting position with special reference to air embolism following the experience of two patients who suffered this complication.

The incidence of air embolism during neurosurgery is difficult to assess. Ericsson, Gottlieb and Sweet (1964) reviewed ninety-three cases from the available literature and reported a further seven. Hunter (1962) described one case in a series of twenty posterior fossa operations in the sitting position with spontaneous ventilation, while in seventeen trigeminal nerve sections the two patients in whom controlled ventilation was used showed signs of air embolism. Emery (1962) cited two cases with spontaneous respiration; one patient was in the sitting position, the other being prone. Marshall (1965) reported five cases in a series of thirty-four patients all of whom breathed spontaneously. Michenfelder and associates (1966) reported eleven incidents of air embolism in 418 posterior fossa operations in the sitting position with controlled ventilation. Since then Michenfelder has described one more (1968). The case reported by McComish and Thompson (1968) was breathing spontaneously in the sitting position.

During the past three years in this unit forty-three posterior fossa operations have been performed in the sitting position with controlled ventilation. The two cases of air embolism which occurred early in this series had several interesting points in common and differed in a number of ways from previously reported cases. These patients were relatively young and in good general health. They had extensive meningiomata in the region of the foramen magnum, a condition which carries a hopeless prognosis without radical operation. It was considered that the sitting position would offer the significantly better access and operating conditions required. Following the experience of these cases the anaesthetic technique has been progressively modified to include more comprehensive monitoring and preventive measures.


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A 40-year-old housewife presented in June 1967 with a 3-month history of tingling and stiffness in both arms and unsteadiness of gait. Examination revealed a spastic tetraparesis with a sensory loss affecting the arms and trunk. On admission to hospital for investigation she weighed 60 kg. The haemoglobin estimation and white cell count were normal. The cerebrospinal fluid contained 330 mg/100 ml protein but was otherwise normal. Chest, skull and cervical spine radiographs were normal but a myelogram revealed a complete blockage at the foramen magnum with displacement of the spinal cord to the right. A diagnosis of a foramen magnum meningioma was made and the patient presented for surgery on July 17, 1967. Premedication consisted of diamorphine 5 mg and haloperidol 5 mg injected intramuscularly. Anaesthesia was induced with thiopentone 300 mg, tubocurarine 30 mg, and phenoperidine 45 mg given. An intravenous infusion was set up and electrocardiographic monitoring begun. The patient was then positioned on the operating table sitting with the head well flexed. Positive-positive ventilation (+2 to +18 cm H2O) with 70 per cent nitrous oxide and 30 per cent oxygen was maintained using a Blease Pulmo-flator with a non-rebreathing system. The systolic blood pressure was steady at 90 mm Hg, the pulse rate was 80 beats/min and the electrocardiogram normal.

Constant monitoring of the heart sounds was maintained throughout the early part of the operation. During the clearing of the arches of the axis and the atlas the surgeon noticed some large veins in the intralaminar space and one of these was opened. Immediately following this a loud splashing sound and occupying cardiac systole was heard, though no additional sounds were noticed in diastole. Bilateral jugular compression was applied and the surgeon sealed the affected veins with the head well flexed. Positive-negative ventilation (+2 to +18 cm H2O) was used to reduce the intrathoracic pressure and to assist the positive pressure ventilation. The pulse and the electrocardiogram remained unchanged for another 30 seconds before the onset of ventricular fibrillation. External cardiac massage was commenced immediately and produced a palpable pulse at the wrist. The nitrous oxide was discontinued and ventilation continued with pure oxygen. The wound was covered with sterile towels and the patient was then gently lowered from the sitting position. By the time this was achieved the ventricular fibrillation had reverted spontaneously to sinus rhythm and the systolic blood pressure was 120 mm Hg. After a few minutes the patient was again placed in the sitting position and the operation recommenced.

Anaesthesia was continued initially with trichloroethylene and oxygen but halothane was later substituted and the blood pressure was maintained at 100 mm Hg without difficulty. The inflation pressure was increased to +20/+7 cm H2O. The pulse rate, blood pressure and electrocardiogram remained stable throughout the rest of the operation.

A meningioma extending from the lower anterior border of the foramen magnum along the inferior clivus as far as the junction of the vertebral arteries was totally removed during the operation which lasted over 8 hours. The patient awoke on return to the ward. The following day her main complaint was of severe retrosternal soreness which was thought to be related to the prolonged use of dry gases with a high oxygen concentration. The patient had a transient mild meningitis but otherwise made a good recovery. When last seen some 2 years postoperatively, she was well with negligible neurological defects.

A 30-year-old man presented with a 3-month history of progressive tetraparesis mainly on the right side with paresthesia of both arms. Clinical and radiological examination indicated that there was a tumour anterior to the upper cervical cord and medulla. No abnormalities were detected in the cardiovascular and respiratory systems. Hamoglobin estimation and white cell count were normal. On July 9, 1968, a transclival exploration of the tumour was attempted but eventually abandoned because of inadequate access. A deterioration in the patient's condition necessitated exploration by posterior craniotomy 2 days later. Premedication consisted of diamorphine 10 mg i.m. Anaesthesia was induced with thiopentone 500 mg and intubation carried out after suxamethonium had been given and the larynx sprayed with lignocaine solution. An oesophageal stethoscope was introduced and electrocardiographic monitoring commenced. The patient was allowed to breathe halothane, nitrous oxide and oxygen spontaneously until fixed in the sitting position with the head well flexed.

The halothane was then discontinued and tubocurarine 45 mg given. Positive-negative ventilation was commenced with a Blease Pulmo-flator using a circle system and soda-lime absorber. Phenoperidine was given intermittently. The pulse rate, blood pressure and electrocardiogram were stable.

During the stripping of the muscles and fascia from the upper cervical laminae a splashing sound during systole was heard through the oesophageal stethoscope. Bilateral jugular compression was applied while the surgeon occluded a large open vein. The heart sounds returned to normal with a stable pulse rate, blood pressure and electrocardiogram. Some 30 seconds after disappearance of the abnormal sound, ventricular ectopic beats were noted on the electrocardiographic trace. Ventricular tachycardia and then fibrillation rapidly supervened. External cardiac massage was commenced immediately. The wound was covered with sterile towels and the patient gently lowered to the left lateral head-down position. Some 90 seconds later ventricular complexes reappeared and some beats were palpable at the wrist. Reversion to sinus rhythm soon occurred and a systolic blood pressure of 130 mm Hg maintained.

The patient was again fixed in the sitting position, the systolic blood pressure falling to a steady 100 mm Hg. A tachycardia of 120 beats/min persisted, however. Anaesthesia was maintained for the next 5 hours. Anaesthesia was then lightened and tubocurarine 45 mg employed.

An extensive clivus meningioma was totally removed. At the end of the operation a tracheostomy was performed as the surgeon thought that bilateral twelfth nerve palsy might be present. The patient awoke at the end of the procedure. The postoperative course was complicated by a low-grade meningitis and a right hemiparesis from both of which he recovered. He subsequently made an excellent neurological recovery apart from residual paralysis of the right trapezius muscle and has fully resumed his professional career.
DISCUSSION

Aetiology.

An interesting feature of major air embolism is its rarity when the circumstances in which it could occur, i.e. an open vein and low venous pressure, must be relatively common. In the present series the venous pressure has rarely approached the level of wound height above the second intercostal space. Factors other than hydrostatic pressure in the venous system are probably important. The flow in the jugular veins may be capable of producing a venturi effect which entrains air into tributary veins held open in bone or soft tissues. The anatomical configuration of the veins and local factors influencing blood flow may thus be as important as the gross measurable pressures in affected cases. It has been shown experimentally (Coles, Richardson and Hall, 1937) that it is during the inspiratory phase of spontaneous ventilation that air is prone to enter open veins. The characteristic deep gasping inspirations produced by air embolism would themselves seem likely to cause even greater amounts of air to be sucked into the circulation.

Prevention of air embolism.

The maintenance of a venous pressure sufficient to discourage the entry of air into open veins, while not at the same time causing cerebral oedema and troublesome bleeding, is the peculiar problem of using the sitting position.

TABLE I

Preventive measures.

<table>
<thead>
<tr>
<th>Observation by the surgeon</th>
<th>Maintenance of venous pressure</th>
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<tr>
<td>Avoidance of negative phase of ventilation</td>
<td>(a) loaded expiratory valve (spontaneous)</td>
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<tr>
<td>(b) positive-positive ventilation (controlled)</td>
<td>2. Use of plasma expanders (including Macrodex, Mannitol, etc.)</td>
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<tr>
<td>3. Use of anti-gravity suit</td>
<td>4. Avoidance of vasodilator drugs (including volatile agents such as halothane)</td>
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Local venous congestion

1. Intermittent jugular compression
2. Inflatable or adjustable collar
3. Balloon catheter in the superior vena cava

Avoidance of high nitrous oxide concentrations

Early surgical identification of open veins is of prime importance. Intermittent production of venous congestion by jugular compression is widely practised but the frequency and effectiveness of the compression is inevitably variable. The tightening of a soft rubber collar has been tried but it may be difficult to fit when the neck is thin or the wound extends to the upper thoracic region.

In theory the raising of the mean intrathoracic pressure by employing a loaded expiratory valve (Hewer and Logue, 1962) during spontaneous ventilation or by controlled positive-positive ventilation should be advantageous. On clinical grounds we consider it inadvisable to allow spontaneous ventilation for the prolonged periods necessary for such operations because of the strong risk of respiratory acidosis. A substantial rise in the central venous pressure can be produced by use of a G-suit (Gardner and Dohn, 1956; Hewer and Logue, 1962) or infusion of plasma expanders. Mannitol produces a transient rise while mannitol-fructose solutions cause a more prolonged high level of venous pressure (Barker, 1969). In combination with raising intrathoracic pressure by ventilation techniques, such measures could produce safe levels of venous pressure and at the same time minimize the risk of circulatory overload. The object is to provide optimum operating conditions by adjusting the venous pressure to slightly less than that which causes troublesome oozing.

Fine control of the venous pressure at wound level might be obtained by using a triple lumen balloon catheter in the superior vena cava, with monitoring of the pressures above and below the balloon. The small degree of obstruction required to produce the desired rise in venous pressure above the balloon is unlikely to cause an appreciable fall in venous return or cardiac output.

Detection of air embolism.

A definite diagnosis can be made when the murmurs produced by the air in the heart can be heard. The sounds have been variously described, ranging from the "drum sounds" to the classical "mill-wheel" murmur. This latter sound, extending throughout systole and diastole, is thought to represent a relatively large amount of air and carries a bad prognosis. For the purpose of auscultation an oesophageal stethoscope is easily introduced after induction of anaesthesia. Its position should be adjusted after the patient is
sat up. Constant listening is necessary during the dissection of the muscles and fascia and bone work, when air embolism is most likely to occur. This arduous task would be greatly facilitated by electrical amplification, but so far we have not succeeded in finding apparatus that is sufficiently selective in amplifying the low-pitched heart sounds. The diagnosis in both our cases was made by the sudden occurrence of murmurs heard in the oesophageal stethoscope, the abnormal sound being confined to systole.

Small amounts of air, however, may produce no detectable change in the heart sounds while still producing electrocardiographic abnormalities (Michenfelder, 1968). Continuous electrocardiographic monitoring is therefore necessary not only to indicate trauma to vital centres but also as a possible means of detecting air embolism (Whitby, 1963). Changes in the pulse rate, blood pressure and venous pressure are most pronounced for major embolism but are less reliable signs for smaller amounts which nevertheless may be significant. Variations in the level of anaesthesia and surgical trauma may also give rise to changes not immediately distinguishable from those produced by air. In both cases reported here the pulse, blood pressure and electrocardiogram remained normal until the sudden onset of ventricular fibrillation some 30 seconds after the air had first been detected. Neither cardiovascular monitoring nor the oesophageal stethoscope should be relied upon as the sole means for detecting air embolism.

### TABLE II
Available monitoring techniques.

| 1. Observation by the surgeon |
| 2. Oesophageal stethoscope |
| 3. Blood pressure (preferably direct arterial manometry) |
| 4. Pulse rate and character |
| 5. Continuous electrocardiography |
| 6. Central venous pressure |
| 7. Jugular venous pressure |
| 8. Doppler ultrasonic device |
| 9. Rapid carbon dioxide analyzer |
| 10. Pattern of spontaneous ventilation (if used) |

The value of observing the pattern of spontaneous ventilation for irregularities which may indicate air embolism or surgical trauma to the vital centres is limited. Variations may be due to unrelated causes, while in some circumstances trauma may cause electrocardiographic changes in the absence of respiratory effects. Furthermore, it will be necessary to assist or control ventilation if spontaneous respiration becomes inadequate because of surgical interference with the cranial nerves or brain stem causing apnoea or respiratory irregularities. The sign of spontaneous respiration is then lost at a critical stage of surgical manipulation when it would be of most value. If spontaneous ventilation had been used in our cases it is unlikely that the diagnosis would have been made earlier with respiratory monitoring. In the case reported by McComish and Thompson (1968) there was a significant observer delay while the changes in the breathing pattern reached a magnitude sufficient to cause concern, and the deepening breaths during this period may have encouraged more air to enter.

The pulmonary arteriolar obstruction caused by bubbles of air will change the ventilation perfusion ratio, as shown by the sudden diminution in the carbon dioxide content of the expired gas mixture on a rapid carbon dioxide analyzer (Betnhune and Brechner, 1968). A criticism of any undue reliance on such a method is that it will only detect air at a later stage in its progress through the circulation. The Doppler ultrasonic flow detector (Edmonds-Seal and Maroon, 1969) was found to be an extremely sensitive instrument in detecting intravascular air, though one drawback may be that it is too sensitive. Edmonds-Seal, Prys-Roberts and Adams (1970) report that air enters the circulation frequently during this type of operation but that the effects are often small or insignificant.

These techniques, particularly the latter, would be of great value for routine use if it could be shown that in conditions likely to produce major air embolism small vanguard bubbles can be detected early enough for effective prophylactic measures to be instituted.

**Effect of embolised air.**

Experimental work has shown that the morbidity produced by air embolism is not so much related to the actual volume as the rate of entry. Richardson, Coles and Hall (1937) suggested that the species LD₅₀ be expressed as ml/kg/min. Durant, Long and Oppenheimer (1947) showed that a dog might tolerate a litre of air injected...
over a period of 50-100 minutes but that 100 ml
given quickly could rapidly prove fatal. In clinical
practice the volume of air has been estimated in
some cases (Stallworth, Martin and Postlethwait,
1950; Hewer and Logue, 1962; Michenfelder,
1968) but there is little worthwhile information on
the rate of entry. This rate may be affected by
the venous pressure, the cross-sectional area of
the opening in the vein, the size of the affected
vein and, possibly, a number of other unknown
factors. In the two cases reported here the pattern
of events was remarkably similar; the same group
of pathologically dilated veins was affected and
the period between opening the vein and hearing
the murmur was very short. Furthermore the
veins were sealed quickly and it therefore seems
that a large volume of air entered in a short time.
The volume was not sufficient to produce cardio-
vascular disturbances while it remained in the
right side of the heart as shown by the time
interval before the onset of ventricular fibrillation
and the fact that in the second case the heart
sounds had returned to normal before circulatory
arrest occurred.

Large volumes of air may completely obstruct
the pulmonary outflow and cause cardiac arrest
while lesser amounts produce a fall in cardiac
output, hypotension and arrhythmias. Small vol-
umes which reach the left side of the heart are
able of producing arterial air embolism. The
deep gasping respirations which have character-
ized some reported cases may be caused by
reflexes arising from the pulmonary vascular bed,
inadequate blood supply to the brain or arterial
embolism specifically affecting the medulla.

Experimentally as little as 0.05 ml of air injec-
ted into the coronary arteries of the dogs has
induced ventricular fibrillation (Durant, 1935).
The relatively long delay before the sudden onset
and relatively easy reversion of the ventricular
fibrillation in our cases would be consistent with
coronary embolism being the cause of cardiac
arrest rather than pulmonary outflow obstruction
(Wolfe and Robertson, 1935).

Nitrous oxide/oxygen anaesthesia can increase
the volume of embolized air as it equilibrates with
blood (Nunn, 1959; Munson and Merrick, 1966).
This is dependent upon the differential solubili-
ties of nitrous oxide. The decrease in the LD₅₀
volume of air by a factor of 3.4 in rabbits anaes-
thetized with nitrous oxide/oxygen mixtures
instead of oxygen, corresponded well with the
theoretical factor. Munson and Merrick (1966)
considered that equilibration could take place in
the venous system of the rabbit. Whether this
can also occur in man and whether a similar vol-
ume change could take place in air bubbles per-
fusing the alveolar capillaries of patients ventila-
ted with 70 per cent nitrous oxide are interesting
possibilities. If this were so it could account for
an insignificant volume of air in the right side of
the heart reaching the left where it is potentially
lethal.

Aspects of treatment.
As soon as the diagnosis of air embolism is
made, steps should be taken to prevent further
air entry and to define the source. In the two
cases reported here jugular compression demon-
strated the open veins which were then sealed by
diathermy coagulation. If the open vein cannot
be immediately seen the wound should be packed
and flooded with saline. All anaesthetic agents
should be discontinued and the ventilation main-
tained with pure oxygen.

| Table III |
| Possible treatment of air embolism. |

Prevention of further air entry
1. Bilateral jugular compression
2. Sealing of open vein
3. Flooding wound with saline
4. Inflation of balloon catheter
5. Inflation of antigravity suit

Removal of air
1. Aspiration through right atrial catheter
2. Inflation of antigravity suit
3. Direct percutaneous aspiration of air from right
   ventricle

Resuscitation
1. Ventilation with pure oxygen
2. External cardiac compression and other general
   resuscitative measures
3. Change of posture

Various methods have been suggested to
attempt to remove the air that has entered.
Direct percutaneous aspiration of air from the
chambers of the heart has been successful on
occasions (Stallworth, Martin and Postlethwait,
1950). Hewer and Logue (1962) report a case
where inflation of the antigravity suit actively
expelled air from the open veins. Martin
(Michenfelder, 1968) quotes a recommendation
by Senn in 1885 for the use of a catheter passed
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...down a jugular vein to aspirate embolized air from the heart. Michenfelder and associates (1966) advise the use of a catheter introduced through an arm vein and positioned so that its tip lies in the right atrium. These workers have shown the value of this for aspirating the air from the venous system in a number of cases. We now insert an 8FG infant feeding catheter into either basilic vein at the elbow after the technique described by Strunin (1969). The attachment of a three-way stopcock to the catheter permits its use for intra-venous infusion, venous pressure monitoring and aspiration should this become necessary. Ericsson, Gottlieb and Sweet (1964) recommended the use of closed cardiac massage for circulatory arrest due to air embolism and demonstrated its value in six cases. Cardiac massage was begun while our patients were still in the upright position and resulted in a rapid spontaneous reversion of the ventricular fibrillation.

On the basis of experiments in dogs (Durant, Long and Oppenheimer, 1947), it has long been recommended that patients affected by air embolism should be placed in the left lateral decubitus position to shift the obstructing bubble of air from the pulmonary outflow tract. The ventricular action then can churn the air into a foam which can be expelled, albeit with some difficulty, into the pulmonary artery. However, a study of the anatomy of man shows that it is the right lateral head-down position that places the pulmonary outflow tract inferior to the cavity of the ventricle and the foam formation might theoretically be facilitated in the same way as by the left lateral position in dogs. The early adoption of the left lateral position will, however, have the advantage of delaying the passage of air through the tricuspid valve into the right ventricle. It is likely that this manoeuvre would allow more time for the air that remains in the great veins and right atrium to be aspirated through the indwelling catheter. In theory, therefore, the change of posture is of variable value in different circumstances. A rapid change of posture is very difficult to achieve in patients necessarily firmly secured in the sitting position. Furthermore, some effort must be made to prevent contamination of the wound. The delay in our cases was such that the circulation had already been re-established before the patients could be lowered from the sitting position. The adoption of the decubitus position may in practice have its main beneficial effect in increasing the venous return and thereby helping to remove the air from the heart and great vessels. External cardiac compression, in addition to its main objective of maintaining the circulation, will also help in this process.

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REFERENCES


DEPARTMENT OF ANAESTHETICS, ST THOMAS'S HOSPITAL

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