Venous Air Embolism as a Complication of the Sitting Position in Surgery

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Summary

The sitting position is the patient position mostly favoured for neurosurgical exploration of the posterior cranial fossa and cervical spine. It allows excellent physical access to the operative site and reduced bleeding – due to improved venous drainage by the physical force of gravity.

Two specific position-related problems are of interest to the anaesthetist

1. Postural haemodynamic disturbance
2. Venous air embolism

Introduction

Although aspiration of air into the venous system during surgery is possible whenever the operation site is above the right atrial level and low central venous pressure exists, this complication is rather rare.[1]

Two factors may predispose to the occurence of venous air embolism during posterior fossa surgery in the sitting position.

i. the high level of the operation site above the heart
ii. the nature of dura sinuses and skull veins whose walls attached to adjacent structures prevent their collapse.

Venous air embolism has been known since the 19th century but was mentioned as a very rare complication. Because at that time only clinically evident abnormalities such as arrhythmias and hypotension were noted it was described as being a very dangerous but rare complication.

Nowadays, Doppler ultrasonic devices enable the anaesthetist to detect airbubbles in the right atrium; bubbles as small as 0.5 ml., a size that is not significant for pulmonary embolism, are detected by the Doppler. More serious embolisation can be detected by capnography – sudden drop in end tidal pCO_2 as evidence of fall in pulmonary flow and by monitoring pulmonary vascular resistancce that rises with diminished blood flow to the lungs.[2]

The sensitivity of three current methods of venous air embolism detection was compared in the prospective study of Bedford[3] on a group of 100 sitting position operative procedures on head and neck. Doppler detection of air bubble was positive in 80 cases, pulmonary artery pressure elevation in 36 patients, end tidal pCO_2 decrease in 30 cases and yet no patient had hypotension or arrhythmias.

From a catheter in the right atrium or pulmonary artery small volumes of air 2 to 20 ml were aspirated quite commonly in the monitored cases.

Now we know that in most cases air aspirated into the veins above the heart passes on and collects in the superior vena cava-right atrium junction, it floats there causing turbulent blood flow and is slowly removed from there to the pulmonary circulation partly dissolving in the blood and partly as small bubbles ending up by blocking the small pulmonary arteries. It seldom happens that large volumes of collected air pass rapidly forward causing massive pulmonary embolism, pulmonary vasoconstriction, diminishing the right heart output, followed by low left heart preload and failure of the left ventricle performance.

Despite the tachycardia, compensatory hypotension follows and influenced by pulmonary shunts a further decrease in oxygen saturation occurs.

The development of these events depends on:

1. volume and rate of air aspiration
2. increase in volume of air bubble relation to N_2O inhalation
3. rate of clearance from superior vena cava – right atrial junction.

From studies performed on animals the possibility of estimating LD 50 of the air in ml/kg of body weight may be calculated in man.[4]

In one study utilising Doppler detection of venous air embolism the LD 50 was calculated to be 30mls air per kg body weight. As clinical symptoms developed in only 69% of children and in 36% of adults from Doppler positive groups it follows that clinical detection does not correlate with physical symptoms, even though smaller volumes of air were sufficient for symptoms to develop in children.[5] The rate of aspiration is of greater value

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than the volume aspirated. Slow I.V. injections of 1000ml of the air during 50-100 minutes were well tolerated by dogs, but rapid injections of 100ml were always fatal.

Since there is the big difference in blood/gas distribution coefficient for N_2O (0.46) and for N_2 (0.013) it is much easier for nitrous oxide to pass from air-buble to blood than for Nitrogen to do the same. During anaesthesia with 50% N_2O in Oxygen we can expect 100% enlargement of air-bubble in blood and with 70% inspired N_2O even 340% enlargement.

Management

The time taken for the air bubble to clear is well correlated with the cardiac index and the anaesthetist has to maintain a good cardiac output. The treatment of detected venous air embolism consists of the elimination of N_2O from inspiratory gas and actively sucking air and foam from a right atrium through the cardiac catheter. Immediate treatment avoids haemodynamic deterioration although pulmonary artery pressure remains elevated for minutes or even hours.

Some anaesthetists used to apply PEEP in order to prevent venous air embolism. Virtually, only high PEEP that is over +15 kPa is effective, but it eliminates one of the advantages of the sitting position by causing increased oozing of blood from the operation area. PEEP can reverse right to left atrial pressure gradient and produce the most serious venous air embolism complication – paradoxical air embolism i.e. the passing of bubbles through the patent foramen ovale to the left heart and systemic circulation and to the brain or other organs.

Assuming that a patent foramen ovale occurs with an incidence of 26 - 30% of population[6] the risk of paradoxical embolism must always be considered.[7]

Conclusion

It seems reasonable to introduce echocardiographic examination of the patient before performing the sitting position surgery and in case right-left heart patency is proven to choose a modified lateral sitting position.[8]

It is essential to have an indwelling cardiac catheter (right atrial one) during the performance of surgery in the sitting position.

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