The efficacy of Doppler monitoring for the detection of venous air embolism

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Venous air embolism can usually be detected by the use of a precordial Doppler ultrasound monitor at an air infusion rate as low as 0.015 ml/kg/min, and consistently at a rate of 0.021 ml/kg/min. This is in contrast to previously reported thresholds wherein the first physiological change, a gasp, occurs at 0.36 ml/kg/min, electrocardiographic changes first take place at 0.60 ml/kg/min, drop in blood pressure at 0.69 ml/kg/min, increased central venous pressure at 0.40 ml/kg/min, and end-tidal CO₂ decreases at 0.42 ml/kg/min. The first change in heart sounds monitored through an esophageal stethoscope is not detectable until an air infusion rate of 1.70 ml/kg/min, and the classical mill-wheel murmur does not occur until 1.96 ml/kg/min. This demonstrates that Doppler ultrasound can detect venous air embolism before the earliest physiological changes, in contrast to most other methods which do not detect venous air embolism until after cardiopulmonary changes have become well established.

Key Words • venous air embolism • Doppler ultrasound monitor • neuroanesthesia • air embolism

One of the most common and potentially fatal complications of operations on the cervical spine or posterior fossa with the patient in the sitting position is venous air embolism. Pressure within the veins of the head and upper portion of the neck is less than atmospheric, so that air may be sucked in when these veins are incised. Usually the air enters the vein gradually, and it is only after several minutes that a potentially fatal chain of events is initiated. The earlier the air embolism is detected, preferably before any physiological alteration begins, the greater the possibility of avoiding a fatal outcome by occluding the open vein, increasing venous pressure, or aspirating air from the atrium or pulmonary artery.

A recent report clearly demonstrates that the usual methods of monitoring, such as esophageal or precordial stethoscope, electrocardiogram (EKG), blood pressure, or central venous pressure measurement cannot detect the presence of air within the venous system until physiological deterioration has become well established. In particular, changes in heart sounds do not occur except with rates of infusion of air that are often fatal.

It has been suggested that precordial monitoring by Doppler ultrasound is the most sensitive method to detect the presence of venous air embolism. To test experimentally whether air embolism can be detected by Doppler ultrasound monitoring before it becomes apparent by other methods of monitoring or before physiological changes occur, a study was performed to determine the minimum rate of infusion that could be consistently detected by Doppler monitoring. This infusion rate was then compared in identical experimental circumstances to the rates of air infusion that produce the various pathophysiological changes associated with venous air embolism.

Materials and Methods

Six mongrel dogs weighing between 16.4 and 28.8 kg (mean 20.4 kg) were anesthetized with a combination of morphine sulfate (2 mg/kg) and alpha-chloro-lose (60 mg/kg). Arterial pressure was monitored through a catheter introduced via the femoral artery to the abdominal aorta just above the renal arteries. Central venous pressure was monitored through a catheter introduced via a subclavian vein. It was filled with saline and used as an EKG electrode while it was advanced until electrocardiographic changes indicated that its tip was positioned just within the atrium. The
lead I EKG was monitored. All recordings were performed on a Beckman Dynograph.*

A Doppler transducer was taped to the chest at the point of maximum cardiac impulse, with ultrasound gel to assure coupling of the transducer to the chest wall. Audible monitoring was carried out with a Metrix Echo-Tone Doppler ultrasound air embolism detector.† Air was infused via a catheter in the left external jugular vein at rates controlled by a Harvard infusion pump.‡ Infusions and recordings were performed in a manner identical to that in a previously reported study¹ to assure comparison to thresholds of infusion rates for various physiological changes.

Infusions were begun for 1 minute at a rate of 0.001 ml/kg/min. Each successive infusion was increased in a stepwise fashion until detected by the Doppler monitor. Note was made of the minimum infusion that could just be detected by the first change in sound of the Doppler monitor and of the rate at which the change in sound could be distinctly detected even by a casual listener to the monitor. Air was infused at progressively higher rates for periods of 3 minutes each, until the thresholds could be determined for changes in each of the physiological parameters measured. At least 30 minutes were allowed between each infusion above 0.01 ml/kg/min. No further infusions were performed in an animal if the central venous blood pressure or EKG did not return to control levels by the end of the 30-minute interval.

**Results**

The first detectable change in Doppler sounds occurred at a mean rate of 0.015 ml/kg/min (range, 0.010 to 0.026 ml/kg/min). Unmistakable changes could be detected at a mean rate of infusions of 0.021 ml/kg/min, and in every case at a rate less than 0.037 ml/kg/min (range, 0.015 to 0.037 ml/kg/min) (Fig. 1).

These rates were compared with the first change in any other parameter measured, a gasp, which occurred at a mean threshold rate of 0.36 ml/kg/min. The first change in the EKG, a peaking of the P wave, was seen at a rate of 0.60 ml/kg/min, and ST-segment depression occurred at 1.22 ml/kg/min. Central venous pressure increased progressively at rates greater than 0.40 ml/kg/min, but blood pressure did not fall significantly until 0.69 ml/kg/min. The physiological changes were at thresholds identical to those found in a prior study.¹

**Discussion**

The first change in Doppler sounds takes place in this model at a rate of 0.015 ml/kg/min, whereas the first change in any measured physiological parameter does not occur until air is infused at 24 times that rate. When air is injected at a rate even less than 6% of that required for any detectable physiological change, the Doppler sounds are so characteristic as to be unmistakable, demonstrating the efficacy of Doppler monitoring for the detection of venous air embolism. Thus, Doppler monitoring represents the most practical method by which air embolism can be detected before physiological changes take place, when it can be dealt with most safely and effectively.²,⁶,¹¹,¹²,¹⁴,¹⁶,¹⁸,²⁰,²² This affords one the opportunity to abort the infusion of air and detect the open vein by compressing the jugular veins, flooding the wound with saline, or to institute treatment measures such as aspiration of air through a previously placed catheter in the atrium²,¹¹,¹³,¹⁴,¹⁶,¹⁸,²⁴ or a Swan-Ganz catheter in the pulmonary artery,²⁰ lowering the patient's head,⁶,¹⁸ and attenuating any increase in the volume of infused air by discontinuing nitrous oxide administration.¹⁹

The argument has been raised that Doppler monitoring is “too sensitive” in that it detects even minute, clinically unimportant, venous air emboli. Certainly, both the surgeon and anesthesiologist should be aware of even these minute emboli, since they could very abruptly lead to the infusion of a large fatal bolus of air. Recognition that there is an open vein affords the surgeon the opportunity to find and occlude the offending vessel while it is still clinically unimportant.

Another complaint about the use of Doppler monitoring is the distressing noise that occurs when an electrocautery is used. This can be avoided by the use of a Doppler air embolism detector incorporating interference sensing and rejection circuits to silence the audio output during electrocautery.⁸ Although this disables the unit so that monitoring does not occur during the actual application of electrocautery, the opportunity to monitor the patient at all other times represents a significant advantage. Certainly, the EKG monitor is likewise disabled during use of electrocautery, but that is not presented as a counter-argument.

The pathophysiology of an air embolism caused by a slow infusion of air is different from that following a sudden bolus injection. In the former, which is much more common clinically, shock occurs secondary to a sympatholytic reflex initiated by a shower of small air bubbles to the lung. After the injection of a bolus of air, however, cardiovascular collapse follows the development of an air lock in the heart.¹

The physiological changes monitored occurred at essentially the same thresholds as they had in the comparison study.¹ In that report, a peculiar gasp was described as being the earliest change on slow infusion

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*Beckman Type R Dynograph manufactured by Beckman Instruments, Inc., Schiller Park, Illinois.
†Metrix Echo-Tone Doppler ultrasound air embolism detector manufactured by Metrix, Inc., Denver, Colorado.
‡Harvard infusion pump manufactured by Harvard Apparatus Co., South Natick, Maine.

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²Roche Embosonde air emboli detector manufactured by Roche Medical Electronics, Inc., Cranbury, New Jersey.
Doppler monitoring of venous air embolism

The earliest cardiovascular physiological changes began at mean infusion rates of 0.40 to 0.42 ml/kg/min. There was a steady increase in central venous pressure as rates of air infusion increased above that point.

Pulmonary artery pressure, however, followed a different pattern. It began to increase at an infusion rate of only 0.42 ml/kg/min, and rose quickly to 300% of baseline at 0.46 ml/kg/min, at which it reached a plateau and did not rise further. This abrupt pulmonary artery pressure change can also be used as an indicator of venous air embolism. It is theorized that the initial increase in pulmonary artery pressure is a reflection of constriction of the vessels within the pulmonary vascular tree, since it occurs with much less air than could produce a diffuse mechanical obstruction. The plateau very likely represents the opening of shunts in the lung, a concept consistent with the decrease in end-tidal CO₂, as has been reported.

The first change in cardiac function was an increase in rate at 0.42 ml/kg/min. However, the first detectable change in the pattern of the EKG was a peaking of the P-wave at a threshold of 0.60 ml/kg/min. The ST-segment depression that is usually described did not occur until 1.22 ml/kg/min. Thus, a change in heart rate is the most sensitive cardiac index of venous air embolism, and the first change in waveform to be monitored is the form of the P-wave.

Blood pressure changes were not seen until a threshold dose of 0.69 ml/kg/min. The blood pressure decreased very gradually to 40% below control levels, up to an infusion rate of 1.70 ml/kg/min. Thereafter, the fall in blood pressure became increasingly dramatic, and, at a rate of 2.0 ml/kg/min, it fell to 25% of control values, which was usually fatal.

In the comparison study, cardiac output was measured with an electromagnetic flowmeter and peripheral resistance was calculated on-line by means of an analog computer. Both parameters changed in a reciprocal fashion at a rate of 0.52 ml/kg/min. As the peripheral resistance decreased, a compensatory increase in cardiac output maintained the blood pressure. It was only after this compensatory mechanism was exceeded, at infusion rates greater than 1.70 ml/kg/min, that the blood pressure fell abruptly and the animal went into shock.

The total inadequacy of monitoring for venous air embolism with precordial or esophageal stethoscope was demonstrated. Even when monitored by an experienced anesthesiologist anticipating a venous air embolism, the first change in heart sound was not heard until an infusion rate of 1.70 ml/kg/min, and...
this was a "drum-like" sound. The characteristic "mill-wheel" murmur was not heard until 1.96 ml/kg/min, long after all physiological parameters had changed and the animal was well on the way to potentially fatal irreversible cardiovascular collapse.

Figure 1 compares the threshold of the rates at which the first changes occur in the various functions that have been monitored. Note particularly that venous air embolism can be detected by Doppler ultrasound at an infusion rate far below that at which the earliest physiological changes occur, providing a significant safety factor. On the other hand, the first changes in heart sounds, even the "drum-like" sounds when monitored under ideal circumstances, do not occur except with rates far in excess of those that cause significant changes in the cardiopulmonary status of the animal.

The clinical effectiveness of early treatment of venous air embolism was likewise demonstrated experimentally, using a previously placed atrial catheter. In 10 dogs, air was infused at the rate of 2.0 ml/kg/min, a rate which would ordinarily be fatal. When deterioration in vital signs began, after a total infusion of a mean of 120 ml, air was aspirated from the atrial catheter and an average of 60 ml was recovered. This brought the EKG and blood pressure changes promptly back to normal and each of the animals in the treated group survived, whereas the control animals died.

A study by English, et al., involved the intravenous injection of bolus air embolism of varying sizes in an attempt to detect the air embolism by 20 different techniques. On injection of the smallest bolus, 0.1 ml/kg, there was a suggestion of change in the pulmonary artery pressure and the precordial Doppler ultrasound. Air embolism was definitely detected at 0.25 ml/kg, and that was with precordial Doppler ultrasound. The next most sensitive techniques were an increase in pulmonary artery pressure and a decrease in end-tidal CO₂, documenting that Doppler ultrasound monitoring is more sensitive than either of these other two favored techniques.

References

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