Retained intracardiac air

Transesophageal echocardiography for definition of incidence and monitoring removal by improved techniques

Retained intracardiac air is a continuing hazard for cardiopulmonary bypass. M-mode transesophageal echocardiography of the left atrium, left ventricle, and aorta is a highly sensitive method for detecting retained intracardiac air bubbles. In 15 patients having valve operations and 18 having coronary bypass, M-mode transesophageal echocardiography was used to record air bubbles during and for 15 minutes after bypass. Routine air clearing methods were used: needle aspiration of the ascending aorta (combined coronary and valve operations) and left atrial, left ventricular, and aortic aspiration after careful passive chamber filling (valve operations). Air was detected in 12 of 15 (79%) patients having valve operations and two of 18 (11%) patients having coronary bypass. One with air in the aorta had visible right coronary air embolism. Three patients with positive echograms had transient central nervous system disturbances.

In a further 11 patients having valve operations, an ascending aorta-venous shunt was created before bypass was discontinued, but air continued to be present in the left atrium. Finally, in seven patients, we added the following maneuvers to our routine: positive chamber filling with echocardiographic demonstration of left atrial stretching, vigorous chamber ballottement, specific echo-directed chamber aspiration, and maintenance of cardiopulmonary bypass until transesophageal echocardiography showed no retained air. Although small amounts of atrial air could still be detected for a minute or two in some patients, this technique appears finally to have eliminated significant retained air and its consequences. A sensitive technique for intracardiac air detection reveals retained air surprisingly often after cardiopulmonary bypass. There are both possible and probable adverse consequences of this air. After valve operations, it is most difficult to eliminate air from the left atrium. There are three essential elements of air removal: First is mobilization of the air; positive chamber filling, stretching of the atrial wall, and ballottement are critical. Second is removal of mobilized air; continuous ascending aorta-venous shunting and nonsuction venting of the left atrium are very important. Third is proof of elimination of air before cardiopulmonary bypass is terminated; transesophageal echocardiography is vital for this.

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We have previously reported our experience with transesophageal echocardiography for detecting entrapped air in the left side of the heart after cardiopulmonary bypass (CPB). In this study we evaluate the various air-clearing methods and try to find the most effective technique for all elimination after CPB.

Methods of echocardiography

Transesophageal M-mode echocardiography was performed with a commercially available Picker Echo View System 80 C (Picker Corp., Cleveland, Ohio) with a 3.5 MHz nonfocused transducer attached to a bronchoscope. The echocardiogram was recorded at a paper speed of 50 mm/sec.

The transducer was introduced into the esophagus after induction of anesthesia and positioned so that a
Fig. 1. Transesophageal M-mode echocardiogram taken during cardiopulmonary bypass. Air is seen as swirling lines in the aorta. LA, Left atrium. Ao, Aorta. RVOT, Right ventricular outflow tract. EKG, Electrocardiogram.

view of the left atrium, aorta, and right ventricular outflow tract was obtained (Fig. 1). The echogram was carefully observed for the appearance of air during CPB and for 60 minutes after termination of CPB. On the M-mode strip chart, air is seen as short, randomly diverted lines. Findings of air in the left atrium on the echogram were arbitrarily graded on a scale of 0 to 3: grade 0 indicated no air; grade 1, trace; grade 2, moderate; and grade 3, dense (Fig. 2).

Because of the importance of the initial 5 minutes after CPB, the presence and amount of air was recorded from 0 to 1 minute during termination of bypass, at 1 to 5 minutes, and at 10 minute intervals thereafter. The presence of air was graded by the maximum finding in each observation period.

Patients and methods of CPB and air removal

With informed consent, 52 adult patients undergoing CPB were studied. Of these, 20 patients underwent coronary bypass grafting (Group A), five atrial septal defect closure (Group B), and 27 mitral, aortic, or combined valve operations (Group C). Group C was divided into three subgroups: C1 (10 patients), C2 (10 patients), and C3 (seven patients).

Anesthesia was induced with fentanyl and maintained with isoflurane or halothane in oxygen. Pancuronium was used as a muscle relaxant. Hypothermic or normothermic CPB was used with hemodilution, a membrane oxygenator, and an arterial filter. Right and left atrial and arterial pressures and cardiac outputs were also measured routinely.

Group A. A single two-stage atrial cannula was used for CPB. Cardiac standstill was obtained by aortic cross-clamping and injection of cardioplegic solution into the aortic root. The aortic root cannula was maintained on slight suction between doses of cardioplegic solution. No other form of venting was used. After cardioplegic arrest of the heart, distal anastomoses were performed first. The cross-clamp was then released at a low perfusion pressure with the ascending aortic vent open and the root of the aorta pinched off. Proximal anastomoses were made with partial clamping of the aorta. At their completion, the grafts were temporarily occluded and aspirated with a needle to remove all retained air.

Group B. During total CPB, the atrial septal defect was closed with direct sutures or with a pericardial patch with the operating table tilted to the left side and the left ventricle and atrium filled with blood. The aorta was not cross-clamped and the heart was neither arrested nor fibrillated. Intracardiac venting was not used. After closure of the defect, needle aspiration of the interatrial septum, the left ventricular apex, and the ascending aorta was performed repeatedly.

Group C1 (10 patients). Of the 10 patients in this group, one had coronary bypass in addition to an aortic valve operation, one had coronary bypass in addition to a mitral valve operation, and one had repeat mitral valve replacement.

The intracardiac procedure was performed with the use of aortic cross-clamping and cardioplegic arrest. The mitral valve was approached through the atrial septum. To eliminate air, a venting tube 18 Fr. in diameter was placed through the left atrium into the left ventricle at the beginning of aortic valve replacement operations and after completion of the valve operation in patients having mitral valve repair or replacement. As the closure of the atriotomy or aortotomy was nearing completion, suction on the vent was discontinued and the heart was allowed to fill passively with the vent open to gravity. The last stitches in the suture line were placed as the lungs were inflated and blood was overflowing between the edges of the incision. The aorta was unclamped at a low perfusion pressure with the aortic root pinched off and the cardioplegic cannula on suction. When cardioplegic solution had been given by coronary catheters, a separate aortic root cannula was inserted specifically for air removal. This cannula was connected by tubing to the vena caval line. The resulting conduit produced a continuous siphonage from the ascending aorta during bypass and an aorta–right atrial shunt once the natural circulation had resumed 2,3 (Fig. 3). In the case of combined coronary bypass and valve operations, proximal anastomoses were done after the chamber was
Fig. 2. The three grades of observed air. The opening and closing of the aortic valve is clearly seen in the frame depicting Grade 1 air.

closed. In this series, the air-evacuation line in the ascending aorta was then removed so that the proximal anastomoses could be performed.

Our standard long-established technique for vent removal was used in all cases: As soon as the heart is beating satisfactorily at normothermia, the vent is taken off suction and put on “overflow,” that is, the system is open to atmospheric pressure at the suction reservoir, which is at or slightly below patient level. The patient is placed in a head-down position, which places the tip of the vent in the apex of the ventricle at the highest point of the heart. Soon after this, with continued evidence of good left ventricular function, the vent is clamped and the venous return to the patient is reduced so that the left atrial pressure rises. Blood is allowed to pool around the right superior pulmonary vein and the clamped vent is pulled only when the left atrial pressure reaches 5 mm Hg. This ensures that as the vent is pulled out and side holes emerge on the outside, blood flows out of these holes under pressure precluding the entrance of air.

In this group of patients, if the amount of air in the left atrium was grade 3 as the patient was being weaned from CPB, extracorporeal circulation was resumed and a 19 gauge needle was used to aspirate air from the left ventricular apex and left atrium through the most accessible route or routes. In the two patients with combined coronary and valve operations, needle aspiration of the ascending aorta was also performed. In the absence of further demonstrable air by needle aspiration, CPB was discontinued.

**Group C2 (10 patients).** Two of these patients had additional coronary operations, and one had repeat mitral valve replacement. Because it had become apparent from experience with the Group C1 patients that if the left atrium was collapsed, the presence or absence of air in this chamber could not be determined with certainty, a different procedure was adopted in these patients. With the vent still in the left ventricle, the left atrial pressure was raised by reducing the venous return to the pump oxygenator. If air was seen in the now distended left atrium, ballottement of the atrial dome and appendage and the ventricle was performed, with the vent and the ascending aortic air-evacuation line open. Both of these were watched for the passage of air bubbles. When no more bubbles were seen in the air-evacuation line and vent, and the left atrium and aorta were clear of air echocardiographically, the vent removal procedure was followed (Fig. 4). Because it was often difficult to maintain a 5 mm Hg left atrial pressure without losing the level in the oxygenator, the procedure would be repeated as necessary, before the vent was finally removed and bypass discontinued.

**Group C3 (seven patients).** A further seven patients (one with additional coronary artery disease and two with repeat aortic and mitral valve replacements) were treated according to a different protocol. In these patients, the ventricle was filled actively rather than passively. The line formerly used to pump in the blood cardioplegic fluid was temporarily connected to the vent and used to pump blood into the ventricle to the accompaniment of atrial and ventricular ballottement and ascending aorta–venous line shunting via the previously described air-evacuation line (Fig. 5). Once no more air could be seen passing through the air-evacuation line, the pumping of blood through the ventricle was stopped, the vent returned to mild suction,
Fig. 3. The ascending aorta–venous shunt (air-evacuation line). A, Patient on cardiopulmonary bypass. B, Venous line clamped. Patient off bypass.

Fig. 4. Vent removal procedure. In B the vent is removed with the atrium full at a mean left atrial pressure of at least 5 mm Hg.

Additional procedures for recognition of residual air after CPB

The major measure for the recognition of residual retained air after the termination of CPB was transesophageal echocardiography. However, with the air-evacuation line allowing blood to shunt from the ascending aorta to the right atrium, if air emerged from the aorta it would be trapped at the apex of the curved venous drainage cannula. This system cannot be assumed to catch all the air passing through the ascending aorta, since we have seen air bubbles appear in the aortic cannula despite the presence of a proximally placed functioning air-evacuation line. However, the method is crudely quantitative. If no air is seen, it suggests that not much was present in the heart; if 1 or 2 ml is seen, it raises the alarming possibility that had all this air gone into a portion of myocardium or cerebrum, significant local damage could have been produced. The presence or absence of a collection of trapped air bubbles in the venous drainage cannula was, therefore, recorded in all cases.

Results

Group A (n = 20). Air was detected in six patients (30%). In three patients, grade 1 or 2 was seen in the left atrium and aorta for 1 to 5 minutes after termination of CPB. The source of air entry was not clear. Five to 20 minutes after the appearance of the air, each of these patients had a sudden fall in systolic blood pressure to 60 mm Hg associated with a raised left atrial pressure (15...
to 25 mm Hg mean). Two patients were managed with resumption of CPB and one without. All three recovered without further trouble. In another three patients, air was seen in the aorta when the partial aortic clamp was released after the proximal anastomoses had been constructed. There was no noticeable consequence of this. All 20 Group A patients recovered without complications. No air-evacuation lines were used, but in two of the three patients who had hypotensive episodes, air bubbles were seen in the aortic perfusion cannula.

**Group B (n = 5).** Air was detected in four patients (80%). In two patients, air remained in the left atrium up to 60 minutes after termination of CPB. The findings are shown in Fig. 6.

All patients of this group made uneventful recoveries and none had any complication. Of interest is the observation that with the right atrium open during CPB, the aortic valve was seen to open. One patient in this group provided another interesting finding; although grade 1 air was observed after termination of CPB, grade 2 air suddenly appeared as the surgeon moved the heart to check hemostasis. This air had been in the heart for more than 10 minutes. In this patient, bubbles of air appeared in the aortic cannula at about the same time. No air-evacuation line was used.

**Group C1 (n = 10).** Air was present during the first minute after completion of CPB in 100% of patients and during the next 4 minutes in 90% (Fig. 7). Generally, large quantities of air were present for 20 minutes or more despite further efforts at air elimination by needle aspiration and the resumption of CPB. After bypass, air left the aorta via the air-evacuation line and settled at the apex of the venous cannula in 100% of the patients.

Two of the patients died. Both had prolonged grade 3 air in the left atrium and ascending aorta. One 75-year-old man had an aortic valve replacement. The appearance of air in the ascending aorta forced a temporary resumption of CPB. He died abruptly in the intensive care unit 15 minutes after the operation. The chest was opened and the heart was in systolic arrest. A 69-year-old man underwent mitral valve repair and placement of quadruple coronary artery grafts. After he was weaned from CPB, small bubbles suddenly appeared in the coronary grafts and the patient became hemodynamically unstable. CPB had to be resumed and maintained for some time before stable hemodynamics could be restored. In addition, a hematologic disorder developed for which he had to return three times to the operating room over several days. At autopsy 8 days postoperatively, there was a massive recent myocardial infarction of uncertain age. It is of interest that during a 2 year period straddling the period of this study, the mortality for isolated aortic valve replacement was 1.5% and for combined valvular and coronary operations,
10.5%. Three other patients in this group had transient neurologic disturbances after the operation. As a result of this experience, we realized that air could not easily be seen in the collapsed left atrium. M-mode echocardiographic observations were valid, therefore, only with the atrium distended to a mean left atrial pressure of at least 5 mm Hg. We also became dissatisfied with needle aspiration of the left atrium as a consistently effective process for eliminating echocardiographically visible air bubbles. For these reasons we adopted, for Group C2, the combination of intermittent distention and collapse, vigorous ballottement for all chambers, careful observation of the air-evacuation line and left ventricular vent, and distention of the atrium for echocardiographic conclusions.

Group C2 (n = 10). By contrast with Group C1, no air was seen during the first minute in 40% of patients in Group C2, and it had cleared completely in 90% by the end of 5 minutes (Fig. 8). During bypass, when the left atrium was distended for the first time, grade 3 air was seen in virtually every patient. After the procedures described had been repeated up to four times, the air was no longer visible. Even so, in some patients, small amounts of air bubbles became visible on the echocardiogram during the first minutes after CPB only to clear quickly thereafter. In this group of patients, the air-evacuation system collected only one or two visible "microbubbles" in three patients: In the rest, air was strikingly absent. There were no central nervous system or cardiac complications in these patients.

Group C3 (n = 7). The results in this group were essentially the same as in the previous group. Very small amounts of air were seen after bypass was terminated, and it cleared very quickly. In one patient, however, grade 3 air appeared in the first minute and, although it diminished rapidly and progressively, it was not completely clear until between 15 and 20 minutes after the end of bypass. Significant amounts of air were captured in the air-evacuation system. This patient had had a mitral valve repair and was one of the two patients in this group with an apical left ventricular vent. It was apparent that air, probably trapped somewhere in the left atrium, had been missed by both the detection and the removal systems. Fortunately, there were no apparent consequences.

Discussion

All cardiac surgeons know that they have inadvertently left air in the heart at the end of CPB on occasion. The possibility that harm may have resulted from this must be admitted. Our previous experience with transesophageal echocardiography as a detection method has shown large amounts of bubbles persisting for surprisingly long periods after the completion of bypass. Extensive studies in both animals and human beings have documented this in the past. Radiographic methods, direct internal cardiac inspection, and ultrasonic and echocardiographic methods have all been used. The simplest to use and interpret are M-mode and two-dimensional echocardiography. For either form of echocardiography the transesophageal probe has a distinct advantage, in that observations can be made continuously and without need for the surgeon to hold a sterile probe in the operative field. Two-dimensional echocardiography has an advantage in that it scans a section of a whole chamber and visualization of the left ventricular cavity is more consistently of good quality. We suspect that for the bubbles in the blood, the two systems are likely to be equally sensitive. The capacity for continuous chart recording makes M-mode echocardiography useful for research purposes, but its most distinct advantage is cost.

We have used a discarded M-mode machine to look at chamber dimensions and motions, as well as air. In today's cost-conscious atmosphere, it would be hard to find the money for a two-dimensional echocardiographic machine for exclusive intraoperative use. Given a method for the detection of air, three related questions naturally arise.

1. What is the significance of echocardiographically detectable air?
2. Should attempts be made to remove it?
3. What methods work best?

What is the significance of echocardiographically detectable air? The bolus injection of air, 0.0001 ml/kg, into the left ventricle is detectable by echocardiography. The smallest bubble size detectable has been variously estimated at 2 to 125 μm. The larger of these may coalesce and be too large to traverse capillary
It has been pointed out that large showers of bubbles may be echocardiographically detectable for long periods without gross neurologic or cardiac change occurring. We can certainly confirm this, but we were also impressed by two observations from our patients: first, complications that could be related to air embolism occurred when air bubbles were seen echocardiographically and did not occur in the absence of bubbles. Second, grossly visible air, seen after CPB in coronary grafts, the arterial cannula, or in our air-evacuation system, correlated very closely with the presence of echocardiographically visible air bubbles during the same period. Past experimental work has shown that small amounts of coronary air embolism can produce acute changes in myocardial blood flow, acute depression of myocardial function, histologic changes of acute myocardial necrosis, and later chronic myocardial infarction. The amount of air that can cause this acute and long-term damage is as little as 0.1 to 0.2 ml (less than 0.01 ml/kg) introduced directly into a canine coronary artery.

Cerebral consequences of echocardiographically visible intracardiac air are obviously difficult to prove. Certainly more sensitive tests than the occurrence of gross neurologic injury are necessary when investigating the neurologic consequences of cardiac operations and CPB. Although improvements in this area are widely acknowledged, subtle changes are still occurring. Aber and associates, using adenosine kinase in the cerebrospinal fluid as a marker of ischemic brain cell injury, have concluded that subclinical brain injury is often seen, that focal brain injury caused by "semimicroemboli" is the likely cause, and that the evidence points to the operative field rather than the extracorporeal equipment as the source. From all of this we can only conclude that echocardiographically detectable air may presage the possibility of obstructive air embolism in the cerebral or coronary circulation with either gross or subtle consequences.

Should attempts be made to remove echocardiographically visible air? Despite a lack of quantitative proof that a particular echocardiographic appearance carries a connotation of a specific volume of retained air or a specific risk of coronary or cerebral air embolism, it seems fair to state that echocardiographically visible air bubbles may signify a risk of air embolism and that they, therefore, cannot be disregarded.

What methods of air removal work best? It is apparent from the experience we and others have described that the long-established usual methods of air removal may be carefully used and yet fail to rid the heart of all intracardiac air before the end of bypass. By adding and emphasizing a few simple and by no means new maneuvers, we were able to achieve this goal. The first of these is vigorous ballottement of the atrium, ventricle, and aorta while the heart is being filled before unclamping. Observation of the air-evacuation line during this maneuver is the key to determining that air has been removed before unclamping. The second maneuver is alternately raising and lowering the left atrial pressure to expand and contract the left atrium. This may or may not help to loosen air bubbles adherent to the atrial wall, but expanding atrium makes it possible to detect air bubbles echocardiographically that cannot be seen with the atrium collapsed. A third procedure is to keep the air-evacuation line and the vent in place until no more air is seen coming from them and no more air bubbles are seen on the echocardiogram. A major ingredient in these additions to the standard procedures is time. On occasion, as much as 5 to 15 minutes may be required to achieve an air-free heart.

Finally, there are several further questions:

1. Is there any advantage to pumping perfusate into the heart via the vent, as was done in Group C3?
2. Should a distinction be made between apical and transvalvular left ventricular vents?
3. Can the vent system itself be a source of air?
4. Can the heart be cleared of air without the use of echocardiographic detection methods?

Positive versus passive ventricular filling. Pumping hemodiluted blood from the oxygenator through the vent with the ascending aortic air-evacuation line open and during ballottement of the heart is a good way to clear air from the ventricle and aorta. It is useful when the ventricle is not filling well because the right side of the heart is open or because the pulmonary venous return is, for the time being, low. This method is not necessary if the heart is filling well without assistance so long as the air-evacuation line is open and ballottement of the heart is being performed. Another method, especially useful when the left atrium is open during a mitral procedure, the aortic valve has become incompetent allowing air into the ascending aorta, and a repeat dose of cardioplegic solution is necessary, is to pour cold electrolyte solution into the atrium during ballottement of the ventricle and ascending aorta and siphonage through the air-evacuation line. These methods are essentially the same: The essence is to loosen and displace bubbles and pull them out of the aorta.

Apical ventricular versus transatrial ventricular vent. The advantage of the vent that is inserted through the right superior pulmonary vein is that it can be removed in stages with a pause in the left atrium for a
combination of ballotttement and siphonage. When an apical ventricular vent is being used, it is advisable to give special separate attention to the left atrium. An appropriate form for this is an air-evacuation line on siphonage.

The vent system as a source of air. With a vent on suction and the heart empty, there is no question that air can be drawn into the heart around the entry site of the vent. To avoid this, knowledge of the left atrial pressure, the ability to take the vent off suction and allow it to drain by gravity, leaving the entry site under a fluid pool, and removing the vent only with a raised left atrial pressure, are all important.

Clearing the heart of air bubbles without echocardiography. Echocardiography has been for us a most useful tool for defining a problem and for monitoring its management. It provided, for example, a surprising insight into a possible problem with our technique for closure of atrial septal defects which, for over 20 years, has been used without mortality and without evident neurologic injury. During the evolution of a routine for clearing air after left-sided valve operations, it was a vital tool. However, although it would be ideal to have echocardiography available in every case, it would seem reasonable that the adoption of the techniques learned through its use will result in virtually the same degree of air clearance. Useful as the echocardiogram is for air detection, if the air-evacuation line and the vent are very carefully observed until no more air is seen coming through them from within the heart, it seems safe to assume that there is no residual air in the heart. Certainly when this is done, it is most unlikely that the air-evacuation line will harvest any more air after bypass.

REFERENCES
22 Savageau JA, Stanton BA, Jenkins CD, Frater RWM:

Discussion

DR. HANS G. BORST
Hannover, Federal Republic of Germany

I would like to comment briefly on the use of the thermocamera for studying the evolution of cerebral and coronary air embolization and its possible reversal by certain therapeutic interventions. After injection of a standard aliquot of air into the carotid artery of the cat, the area of brain surface cooling rapidly increases. Within the first minutes, arterial pressure rises while the area involved tends to diminish. Subsequently, however, arterial blood pressure decreases successively and the perfusion defect widens as the temperature on the brain surface falls until a large irreversible defect is established. Administering methoxamine 2 minutes after air embolization produces a sharp sustained rise in arterial blood pressure. As a result the air is cleared from the brain within minutes. Retrograde brain perfusion, as often has been claimed, does not effectively diminish postembolic brain ischemia. We have done a similar study on the coronary circulation in dogs. Again, only raising the pressure was effective in totally eliminating air, whereas dipyridamole and cardioplegia were not.

For clinical purposes, these findings indicate that if air embolism is evident or suspected, the arterial pressure should be raised substantially, either mechanically or pharmacologically. In cerebral air embolism, we suggest briefly directing the arterial perfusion cannula into the innominate artery. In case of coronary embolization we turn the aortic perfusion cannula down to the aortic base and constrict the vessel downstream. In coronary embolization we turn the aortic perfusion cannula down to the aortic base and constrict the vessel downstream. Visible intracoronary air immediately disappears and normal contractile power is reestablished promptly.

DR. BENSON B. ROE
San Francisco, Calif.

The insidious persistence of intracardiac air is one of the most neglected and most hazardous aspects of cardiac surgery.

About 20 years ago I was persuaded to put Doppler probes on the ascending aorta in a series of patients. We recorded air bubbles for 15 to 20 minutes after the end of bypass, even though vigorous air evacuation measures had been instituted. Our conclusion from this experience is that God is good to cardiac surgeons and that patients tolerate much more air embolism than the textbooks would allow us to believe.

In addition to the measures enumerated by the authors, there are others that could be added. In particular, I would like to emphasize the importance of putting the head down as the circulation is being reestablished. One must assume that air is trapped in the heart and some of it will not be removed. In addition, the left atrial appendage should be inverted because it is an air trap at the high point. Of course, the air lies in little crevices in the trabeculae so I make a point of shaking the whole table vigorously.

DR. WATTS R. WEBB
New Orleans, La.

Our interest in an embolism goes back well over 20 years. The cardioscope introduced into the heart, which we used many years ago, demonstrated that despite all the vigorous mechanical manipulations, such as Dr. Frater has described, a large number of bubbles remained trapped in the trabeculae and the papillary muscles of the heart. In addition, we have always believed it was futile to expect the little aortic vent to be adequate in removing all the bubbles that swirl past in systole. Movies of the intracerebral circulation show that it takes some 20 to 30 minutes for a small slug of air within the 200 to 300 μm range to be absorbed from an arteriole. On the other hand, carbon dioxide, being 22 times more soluble than air, is almost instantly removed.

Accordingly, we have used a technique, not original with us, of placing a prosthetic vascular graft around the pericardial well. Through the innumerable interstices of the graft, 12 to 15 L of carbon dioxide flows into the pericardial well per minute. If one is careful not to leave the cardiotomy sucker in place constantly, one will achieve an atmosphere of 90% to 95% carbon dioxide within the well. In addition to this technique, we use all the mechanical measures we can to make sure that no gas of any nature is left within the heart. Nonetheless, gas is always there, because we never take out an aortic vent without seeing some bubbles. However, it has been a long time since we have seen anything that suggests an intracerebral air embolus. Similarly, we no longer see the frequent intensive care unit syndrome, which I'm sure was related in large measure to innumerable very tiny gas emboli.

DR. HENRY M. SPOTNITZ
New York, N. Y.

Dr. Paul Rodigas published a paper on two-dimensional intraoperative echo from Presbyterian Hospital in The American Journal of Cardiology in 1978. We detected microbubbles in 55% of 124 patients studied at that time, none of whom had any sign of neurologic injury or other injury related to this phenomenon. Accumulated data suggest that microbubble embolization usually is detected by echo at a level far below that which is apparently clinically significant. None of our patients had neurologic injury and none of the patients in three other very large series that have been published or are in press have had any evidence of this problem.

In fact, the current trend in the use of microbubbles intraoperatively goes in the other direction; that is, deliberate
injection of microbubbles invisible to the naked eye as a contrast agent to define lesions such as ventricular septal defect or to define the absence of mitral regurgitation. For instance, in checking for mitral regurgitation, we find that the left atrium is completely nonopacified and there is no evidence of any microbubbles traveling retrogradely across the mitral valve.

I think the problem with this paper is that the technique is far too sensitive to be relevant to the clinical issue of whether retained air is significant. I believe that the retained air pockets that are clinically important are those which cause visible microbubbles or bubbles coming out of our vents, as some of the other discussants have suggested. The present observations therefore may not be directly relevant.

Before closing, I would like to ask Dr. Frater a final question about the use of the transesophageal route for routine observations in the operating room. Worldwide, the experience with the transesophageal route during operations now amounts to about 1,000 patients with no reported complications and no incidence of esophageal perforation. However, I am still unconvincing and am uncomfortable with this technique. Dr. Frater, what special techniques are used to protect your patients from esophageal perforation and how would you explain a perforation to your patient if such a problem were to occur?

DR. GEORGE E. MILLER, Jr.
Sacramento, Calif.

I wish to emphasize the ease, accuracy, and reproducibility of intraoperative echo for detecting retained intracardiac air. We reported the use of this modality to the Society of Thoracic Surgeons in 1973. We used a small hand-held probe and found in the laboratory that we could trace and remove very small amounts of air from all four chambers. We used it clinically for a short while and now, with more compact equipment, we probably should consider using it again. As Dr. Frater reported, the technique is sensitive, accurate, easy, and reproducible.

DR. FRATER (Closing)

I thank the discussants for their comments.

Dr. Borst has made a very important point. It is indeed pressure that must be used to drive air through either the coronaries or the brain if you know that air has reached there.

I agree with Dr. Roe’s extra points. We certainly do invert the atrial appendage and we place the patient in a head-down position. These are important adjuncts. I also agree that vigorous shaking is important.

Dr. Webb has a point well made. Air does cling to the trabeculae. We have concentrated on repeatedly filling the heart and letting it collapse as well as using vigorous ballottement in an effort to shake loose those bubbles of air that cling to the trabeculae.

Dr. Webb’s newer technique using carbon dioxide may well be helpful. We gave it up years ago when we noticed black particles coming out of the carbon dioxide line into our field.

Dr. Spotnitz’ points are important. I agree that we cannot with certainty assign significance to the presence of microbubbles, especially in the lower grades. Indeed, air can be seen to persist for a long time without apparent gross neurologic damage. However, when we see Grade 3 air by echocardiography, this appears to us to correlate with our ability to recognize it by other techniques and with the occasional catastrophes that occur. My late father, who was a surgeon, said, “The difference between good medicine and bad is 100% in effort and 5% in results.” I think one should not take this air lightly and let it be, but rather work vigorously to eliminate it entirely. Were I the patient, I would rather be told that there was no detectable air rather than be given the statement, “We saw a lot of air but we don’t think it matters.”

There are studies from Denmark and Sweden using far more sensitive techniques for demonstrating cerebral damage than gross neurologic examination. These studies show a still persisting incidence of significant reduction in cerebral blood flow and raised adenylate kinase levels in the cerebrospinal fluid.

None of our patients has had an esophageal injury. We do in fact obtain an informed consent specifically different from the regular consent form when we pass the transesophageal echocardioscope, because we are not sure of the legality of what we are doing. When I say legality, I mean it in the strictest sense of malpractice prevention. I do not know what I would say if we perforated the esophagus. I would apologize, obviously, and repair it.

We agree entirely with Dr. Miller’s comments, and we certainly acknowledge our predecessors in this work.