Massive Air Embolism in a Fontan Patient

Gregory S. Matte, BS, CCP, LP; Barry D. Kussman, MBBCh; Joseph W. Wagner, BS, CCP, LP; Sharon L. Boyle, BS, CCP, LP; Robert J. Howe, BS, CCP, LP; Frank A. Pigula, MD; Sitaram M. Emani, MD

Department of Cardiac Surgery, Children’s Hospital Boston, Boston, Massachusetts

Abstract: Most institutions performing cardiopulmonary bypass for congenital heart disease patients use an integrated hard shell cardiotomy and venous reservoir attached to an oxygenator. It is of paramount importance that the integrated reservoir be vented so as not to cause pressurization. A pressurized sealed cardiotomy has been reported to occur secondary to issues with vacuum assisted venous drainage systems as well as improper venting in general. We report a case of air embolus caused by retrograde propulsion of air through the venous line secondary to a pressurized cardiotomy reservoir in a patient with Fontan circulation. The mechanism of cardiotomy pressurization is described, and the scenario simulated in a mock circuit. Keywords: pediatric perfusion, air embolism, pressurized cardiotomy.

DESCRIPTION

A 4.5-year-old patient (16.7 kg, body surface area .69 m²) with hypoplastic left heart syndrome who was status post-
Stage 1 Sano, bidirectional Glenn shunt, and extra-cardiac fenestrated Fontan presented for an atrial septectomy, tricuspid valve repair, and recreation of the Fontan fenestration. The perfusion staff set up a bypass circuit several hours before the case, as is the common practice. A Terumo Capiox FX 15–30 oxygenator, 1/4" arterial line and pump loop, and a 3/8" venous line were part of the custom tubing pack placed on a Sorin S5 heart-lung machine (Sorin Group, Milano, Italy). The oxygenator heat exchanger was water tested during setup. The yellow (vented) cap, which is normally on the reservoir vent port, was replaced with a standard (non-vented) blue cap from a cardiotomy sucker port when the original cap was misplaced. Figure 1 shows the standard Terumo FX-15–30 reservoir top, whereas Figure 2 shows the improperly capped vent port. The different internal bores of the two caps can be appreciated in Figure 3. The patient was placed under anesthesia, lined per protocol, and prepared for surgery without incident. The bypass circuit prime volume of 555 mL included 205 mL Plasma-Lyte A (Baxter Healthcare Corporation, Deerfield, IL), 350 mL fresh whole blood, 1665 units heparin, 15 mEq sodium bicarbonate, and 1500 mg calcium gluconate. The venous reservoir contained 175 mL before bypass.

A single 14 Fr Biomedicus arterial and 14 Fr and 18 Fr angled DLP cannulae (Medtronic Incorporated, Minneapolis, MN) were placed. Following heparinization to an activated clotting time of 515 seconds, the surgeon instructed the perfusionist to go on cardiopulmonary bypass (CPB). The arterial line clamp was released and the arterial pump head was turned on. When the venous clamp was released, the perfusionist noted a rapidly decreasing reservoir level and attempted to add Plasma-Lyte A while the surgeon ensured proper placement of the venous cannulae. The venous reservoir level continued to drop even after the level sensor appropriately shut down the arterial pump head. Bubbles were seen entering the bag of Plasma-Lyte A when the perfusionist tried to add volume. The reservoir level went below the low level sensor. Air was noted in the venous cannulae and heart, and the surgeon instructed the perfusionist to terminate bypass immediately. Both arterial and venous cannulae were clamped, and the arterial cannula was removed. Deep Trendelenburg was assumed with the thought that rising air would preferentially travel to the lower body and not the head. Upon identification and removal of the non-vented blue cap by a second perfusionist, a release of air was heard from the vent port. It was deduced that pressurization of the
reservoir had occurred secondary to the improperly used blue cap. Manual heart massage was performed since cardiac output was deemed inadequate. De-airing of arterial and venous cannulae and venous line was performed. Air was seen in the common atrium, and ST segment elevation with myocardial dysfunction suggested coronary air embolus. It was deduced that the air was propelled retrograde through the bypass circuit venous line and through the previously closed Fontan fenestration to the common atrium and aorta. Since the superior vena cava was not dissected free, immediate retrograde cerebral perfusion (RCP) was not possible. Traditional bypass with pH-stat blood gas management utilizing a premixed sweep gas of 96% oxygen and 4% carbon dioxide resumed with innominate vein and common atrial venous drainage. This blood gas technique maximized cerebral blood flow to help flush air out of the cerebral vasculature and maximized the gas gradient between the nitrogen air emboli and the blood, thus maximizing absorption and clearance. The patient was cooled to 18°C while the superior vena cava was dissected free. Hypothermia decreases the cerebral metabolic rate while at the same time promotes removal of emboli from the body by decreasing the air emboli bubble size.

Retrograde cerebral perfusion has been shown to be effective in clearing air from the cerebral circulation secondary to air embolism (3). Plans for RCP were discussed while the patient was cooled. After 18 minutes of cooling, RCP was instituted through the innominate vena cava for 3 minutes at a rate of 300 mL/min (.43 L/min/m²) while venting through the aortic root. Intermittent pressure on the carotid arteries was applied to facilitate air removal from the vertebral arteries. Air was seen exiting the aortic root. Methylprednisolone 30 mg/kg was administered as an anti-inflammatory agent to help prevent associated ischemic injury. Antegrade CPB resumed for 50 minutes during which the operation was completed under cardiopulmonary arrest. Upon inspection of the Fontan baffle, it appeared the fenestration that had been forced open by the air bolus remained patent. The patient was rewarmed. When the patient temperature reached 25°C, a second period of RCP commenced at 300 mL/min with central venous pressure monitoring showing 50–55 mmHg. Intermittent pressure on the carotid arteries was again applied. It was the team opinion that a second period of RCP would absolutely ensure as much air was removed with this modality as possible. Epiaortic echocardiogram was performed to confirm clearance of air bubbles from the carotid arteries during RCP. Further air was not seen. The patient was re-warmed to a rectal temperature of 32.8°C over 34 minutes with a 100% oxygen sweep gas. The patient was then successfully weaned from CPB.

Immediate and delayed hyperbaric oxygen therapy has been shown to decrease morbidity from cerebral air embolism by decreasing air emboli size and increasing the diffusion gradient for nitrogen, both of which promote reabsorption and clearance (4,5). Hyperbaric oxygen therapy also supports compromised tissue beds by increasing the blood-to-tissue oxygen gradient. Intraoperative neurology consultation was obtained and the nearest hyperbaric chamber facility was contacted to investigate the possibility of such therapy. Concerns over our ability to manage bleeding and any postoperative hemodynamic instability effectively in the hyperbaric chamber guided our decision to withhold hyperbaric therapy in the immediate postoperative period. Instead, direct transport from the operating room to the radiology department for a computerized axial tomography scan of the head was organized, with plans to consider hyperbaric oxygen therapy only if air bubbles were visualized by this imaging modality.

**POSTOPERATIVE COURSE**

The computerized axial tomography scan of the head performed immediately postoperatively did not show evidence of cerebral air, and therefore the decision was made not to pursue hyperbaric oxygen therapy. To note, delayed hyperbaric oxygen therapy has been reported in adults with positive outcomes and so may be considered if residual air is found (5). In the intensive care unit the patient was paralyzed, sedated, and cooled to 34°C to minimize the cerebral metabolic rate. Inotropic support was maintained to keep the mean arterial pressure greater than 60 mmHg to optimize cerebral perfusion. Continuous electroencephalography monitoring was instituted, and demonstrated normal brain pattern. Following 36 hours of this cerebral protective strategy, a repeat head computerized axial tomography was performed that demonstrated no evidence of cerebral edema, necrosis, or hemorrhage. At this point, the paralytic was discontinued and the patient was allowed to gradually warm to normothermia. Thorough neurologic examination performed after extubation showed no focal neurologic deficits, and the patient was discharged home on postoperative day 13.

**SIMULATION STUDY**

Davila et al. reported anecdotal evidence of cardiotomy pressurization to 20–30 mmHg causing venous air embolus (2). We were able to recreate our own scenario of improper venting with a simulated patient circuit at this and higher reservoir pressures (Figure 4). In our study, a bypass circuit was primed with blood and crystalloid. Pressure inside the cardiotomy reservoir was measured. The arterial and venous lines were connected to a separate reservoir, which served as the simulated patient. Cardiotomy suckers (open to air) were turned on. The yellow vent cap was replaced with a blue non-vented cap. Pressures in the cardiotomy
reservoir reached 450 mmHg before the occlusive blue cap on the vent port blew off. Furthermore, we were able to demonstrate a stable cardiectomy pressurization of 100 mmHg with the arterial and venous lines clamped as they would normally be prebypass and the suckers on at their normal flow. The pressure in this scenario was allowed to vent either through a slightly loosened reservoir connection or the hemoconcentrator, which was attached via pressure tubing to the reservoir top. To note, nothing sounded different at this level of pressurization. Higher levels of pressurization can lead to unique sounds in the sucker heads as they pump against pressure. We then unclamped the arterial and venous lines as they normally would be when commencing bypass. The flow of fluid (200 mL) and air retrograde to the simulated patient was rapid (5 seconds). The description and recreation of this event was videotaped for discussion at a multidisciplinary meeting, and is available internally online.

COMMENTS

Air embolism through the bypass circuit can be a lethal complication of cardiac surgery (6,7). In this case, the venous line pressure was sufficient to force open a previously closed Fontan fenestration. It is striking that an error in the type of cap used on the single vented port of an oxygenator can be so potentially catastrophic. It is also important to note that in this situation, clamping of the venous circuit is necessary to prevent further air embolus. Simply turning off the pump does not interrupt the flow of air. Moreover, it is important to note that unless the cause is identified and the occlusive cap is removed, the air embolus will recur once bypass is reinstituted.

A multidisciplinary meeting was held several days after the event. The simulation video was reviewed as well as current Perfusion Department policy regarding air embolism on bypass. The result of this meeting was that our pre bypass checklist is now updated in accordance with the American Society of Extracorporeal Technology guidelines to include ensuring proper venting of the cardiectomy reservoir (8). We have also standardized perfusion practice in that the yellow vent cap is now completely removed once the pump is primed. We believe these changes will prevent a recurrence. A literature review gave some credence to monitoring the cardiectomy pressure with an alarm value set at a minimal positive pressure, but we believe our practice changes will be sufficient (2). The potential downside of cap removal is more direct exposure of the reservoir to ambient air conditions, and potential for debris entrainment when the suckers are off. However, the risks of this appear to be outweighed by the risks of air embolus.

Finally, the authors would like to point out a major difference in the Terumo Capiox FX series reservoir tops. It strikes us as odd that the FX 05 reservoir top has the vent port on the left side (Figure 5) whereas the FX 15–30

![Figure 4. Mock circuit assembled to measure cardiectomy reservoir pressure with yellow vented cap replaced with standard blue cap. The hemoconcentrator connection to the reservoir was loosened to a level that allowed for a constant positive pressure of 100 mmHg.](image)

![Figure 5. Terumo FX-05 reservoir top with yellow vented cap on left and adjacent non-vented blue cap.](image)
(Figure 1) and FX 25 both have the vent port on the right side. While ensuring proper reservoir venting is the responsibility of the perfusionist, it would be prudent for all manufacturers to standardize vent port placement on their similar-in-appearance product lines. Perfusionists are known for identifying subtle differences in their bypass equipment or the operating room sights and sounds in general. Standardization of vent port placement and thus cap color placement may be an opportunity to help prevent future errors by perfusionists.

ACKNOWLEDGMENTS

The authors would like to express sincere appreciation to the Children’s Hospital Boston Cardiovascular Program for fostering an environment of free communication with a focus on problem resolution and not simply assigning fault. The Non-Routine Event Reporting System at our institution has allowed for effective dissemination of bypass safety issues, real or potential, to the entire multidisciplinary team (9). It is our contention that this potentially catastrophic event was handled effectively and efficiently due to the institutional culture that exists and is promoted by hospital leadership.

REFERENCES