A Battle Plan in the Event of Massive Air Embolism during Open Heart Surgery

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Abstract

After nearly 30 years of clinical open-heart surgery, air embolism to the coronary and cerebral arterial circulations during cardiopulmonary bypass procedures still remains a persistent hazard. The true incidence of morbidity and mortality related to air embolism is difficult to determine because of a wide spectrum of clinical presentations. The major manifestations of coronary air embolism are arrhythmia and/or reduced cardiac output. Those of cerebral air embolism are frank stroke, confusion or postoperative psychosis. A "battle plan" is proposed to outline a rational course of action in the event of massive air embolism recognized in the course of an open heart operation. If followed, the damage to the patient can be minimized, and full recovery can result. This "battle plan" represents the synthesis of the cardiac surgical literature on the prevention, recognition and treatment of air embolism occurring during open heart surgery.

Introduction

Cardiac surgeons and perfusionists continue to be surprised by the unexpected appearance of air bubbles in the course of an open heart operation. The true incidence of morbidity and mortality related to air embolism is difficult to determine because of a wide spectrum of clinical manifestations. Arrhythmia and/or reduced cardiac output are the major manifestations of coronary air embolism. ECG changes, mainly transient ST segment elevation with diphasic T waves in the distribution of the more anterior right coronary artery, are the most prominent and typical features. The major manifestation of cerebral air embolism are frank stroke, seizures, confusion or postoperative psychosis. On occasion, major cerebral air embolism can manifest itself insidiously with the patient initially awake and neurologically normal. Then, over a period of hours or days, as the consequences of cerebral ischemia evolve, the patient gradually lapses into varying states of coma.

For the cardiac surgeon, air embolism can jeopardize an otherwise excellent surgical result. Air embolism in the course of open heart surgery is fraught with medico-legal consequences and is regarded by some attorneys for the plaintiff patient as an instance of "res ipsa loquitur" (the thing speaks for itself) negligence on the part of the open heart team. As Mills pointed out, most episodes of air embolism during open heart surgery go unreported or are quietly ignored out of fear of just such litigation and personal or program embarrassment.

Perhaps the single greatest fear of many perfusionists is inadvertently "pumping air." Aside from the potential for catastrophic patient injury there is the potential for disastrous psychological consequences for the involved pump team. Profound depressions and even suicides have been noted in perfusionists who "pumped air."

For all these reasons the prevention of air embolism is the responsibility of every single person in the open heart operating room—including surgeons, perfusionists, assistants, anesthesiologists, cardiologists, scrub, monitoring and circulating nurses and even observers and visitors. As Dr. Frank C. Spencer has commented, "Nothing in an operation is neutral, it either helps or harms the outcome." Akin to the proverbial "Do not talk to driver while bus is in motion" should be similar signs in the open heart operating room.
room, "Do not talk to perfusionist while cardiopulmonary bypass is in progress." Recent reports of massive venous line gas embolism due to pressurization of the oxygenator by a visitor stepping on the waste gas vent hose, obstructing it, emphasize the need for constant vigilance in preventing and detecting potential situations in which massive air embolism can occur.

Case Report

In 1978 routine mitral valve replacement with a porcine bioprosthesis was performed under moderate systemic hypothermia (25°C.) on a 49 year old man. Warming to normothermia was accomplished and cardiopulmonary bypass was slated to be terminated in several minutes. Dual venous cannulation of the superior and inferior vena cava had been employed with a standard "Y" connector fitting to a single venous line. This venous line suddenly became kinked under the drapes obstructing venous return from the patient. Perfusion was proceeding at a 5 liter per minute rate, and in only a few seconds the oxygenator reservoir emptied unnoticed by an experienced perfusionist. Massive air embolism estimated at between 1 and 2 liters occurred via the arterial line into the ascending aorta.

The cardiac chambers became distended with air and the coronary arteries were noted to be filled with air. Cardiopulmonary bypass was stopped, and air was evacuated from the ascending aorta and cardiac chambers by disconnecting and aspirating all arterial, venous and vent cannulae. Epinephrine, metaraminol and calcium chloride were given and isoproterenol drip begun. The patient was given additional mannitol and corticosteroids. The lines were immediately filled with fluid, the pump reprimed and cardiopulmonary bypass was resumed within 3 to 4 minutes. The heart was defibrillated with the 3rd electrical shock. Ventilation was with 100% oxygen while normothermic bypass continued until cardiac tone was normal. ECG changes diagnostic of massive coronary air embolism regressed.

The pupils, which had fully dilated, returned to normal size and reactivity. Postoperatively the patient was comatose and suffered grand mal seizures for 24 hours. These responded to treatment with diphenylhydantoin and diazepam. Mannitol was infused by drip and corticosteroids were continued for 96 hours. The patient awoke starting on the second postop day. He was mentally normal on the fourth postop day. Septic sternal dehiscence was treated with debridement and resuturing on the 13th postop day. Specific antibiotic therapy dictated by wound cultures was given. Rehabilitation, with full neurologic recovery, led to discharge two months after operation. The patient remains well to this day, working full time as a cattle rancher.

Discussion

As the preceding illustrative case report dramatically emphasizes, early warning electronic fail-safe devices with automatic arterial head shutdown circuits should be attached to the oxygenator and checked for proper function prior to each perfusion run. When automatic arterial pumphead shutdown occurs, the following steps should be immediately taken by the perfusionist (Figure 1):

1. Apply clamps to the arterial and venous lines immediately upon cessation of pumping. Clamping the arterial line will prevent any upward advance of gas towards the patient due to density gradients. Clamping the venous line will prevent exsanguination of the patient by gravity drainage into the pump.
2. Purge all gas from the arterial line.
3. Refill all lines with fluid.
4. Confirm that there is sufficient fluid volume in the oxygenator in order to safely reinstitute cardiopulmonary bypass.
5. Resume cardiopulmonary bypass as soon as possible.

Figure 1: Battle Plan for Automatic Arterial Pump Head Shutdown

1. CLAMP ARTERIAL AND VENOUS LINES.
2. PURGE GAS FROM THE ARTERIAL LINE.
3. REFILL ALL LINES WITH FLUID.
4. CONFIRM SUFFICIENT VOLUME IN PUMP-OXYGENATOR.
5. RESUME CARDIOPULMONARY BYPASS AS SOON AS POSSIBLE.
**Figure 2: “Battle Plan” for Massive Air Embolism during Cardiopulmonary Bypass**

1. FOLLOW STEPS 1-5 FOR AUTOMATIC ARTERIAL PUMP HEAD SHUTDOWN.
2. SUMMON A BACKUP PERFUSIONIST, IF AVAILABLE.
3. CONFIRM ABSENCE OF OBSTRUCTION TO THE OXYGENATOR GAS VENT LINE.
4. MANUALLY ASPIRATE AIR FROM ARTERIAL CANNULA. AVOID REVERSAL OF PUMP FLOW DIRECTION TO MINIMIZE CHANCES FOR CONFUSION AND ERROR.
5. PLACE PATIENT IN DEEP TRENDENLENBERG (HEAD DOWN, FEET UP) POSITION.
6. RESUME CARDIOPULMONARY BYPASS AS SOON AS POSSIBLE. EMPLOY AORTIC ROOT VENT, PREFERABLY WITH SUCTION APPLIED.
7. ADMINISTER VASOPRESSOR (METARAMINOL OR NEOSYNEPHRINE) TO INCREASE ORGAN AND CORONARY PERFUSION PRESSURE.
8. IF HYPOTHERMIC, COOL FURTHER AND WARM SLOWLY. IF NORMOTHERMIC, MAINTAIN NORMOTHERMIA.
9. DISCONTINUE NITROUS OXIDE VENTILATION. VENTILATE PATIENT WITH 100% OXYGEN.
10. VENTILATE THE LUNGS VIGOROUSLY TO DRIVE AIR FROM THE PULMONARY CIRCUIT TOWARDS LEFT HEART AND AORTIC ROOT VENTS.
11. ADMINISTER HIGH DOSE INTRAVENOUS CORTICOSTEROIDS, 2-4 GRAMS OF METHYLPREDNISOLONE AND OR 20 MG. OF DEXAMETHASONE AND CONTINUE FOR 72-96 HOURS POSTOPERATIVELY.
12. ADMINISTER 25 GRAMS OF MANNITOL INTRAVENOUSLY AND BEGIN GLYCEROL DRIP 1.2 GMS./KG./24 HOURS FOR 48 HOURS POSTOPERATIVELY.
13. AVOID PROFOUND BARBITURATE “ARTIFICIAL COMA.” AIM FOR EARLY AROUSAL, THE BEST PROGNOSTIC INDICATOR.
14. PACK HEAD OF INFANTS AND CHILDREN IN ICE, PROTECTIVELY PADDING EARS TO AVOID FROSTBITE INJURY.
15. EMPLOY HYPERBARIC CHAMBER IF IMMEDIATELY AVAILABLE AND LOGISTICALLY FEASIBLE. TREAT PATIENT WITH 6 ATMOSPHERES 100% OXYGEN FOR 38 HOURS.
16. NEVER GIVE UP AS LONG AS PATIENT IS ALIVE. RECOVERY IS USUAL EVEN FROM MASSIVE AIR EMBOLISM IF TREATMENT IS PROMPT AND SPECIFIC.
17. PROVIDE INTENSIVE PSYCHOLOGICAL SUPPORT TO THE PERFUSIONIST THROUGH THIS DIFFICULT CRISIS.

When massive arterial or venous gas embolism occurs the following “battle plan” sequence should be initiated (Figure 2):

1. Stop the pump and fill arterial and venous lines and the pump oxygenator with sufficient priming fluid to safely resume cardiopulmonary bypass.
2. If a backup perfusionist is available in the hospital he or she should be summoned to lend support.
3. Check the oxygenator's gas vent line to determine if it is obstructed and if it might have caused pressurization of the oxygenator causing venous gas embolism. If so, remove the obstruction.
4. Manually aspirate air from the arterial cannula with a Toomey syringe. This is preferred to reversal of arterial head pumping, or to switching the arterial line to the venous cannula to retrofill the body with blood via the venous side. There is less chance for confusion in an already chaotic setting if the normal direction of perfusion is maintained.
5. Place the patient in deep Trendelenberg (head down—feet up) position to minimize the more devastating cerebral air embolism, albeit increasing the likelihood of coronary (especially right coronary) air embolism. Coronary air embolism can be more successfully treated with cardiopulmonary bypass support than can cerebral air embolism.
6. Resume cardiopulmonary bypass as soon as gross air embolism is evacuated from the aortic arch. Employ an aortic root vent, preferably with suction applied.
7. Administer intravenous vasopressor (metaraminol or neosynephrine) to increase coronary perfusion pressure and expedite clearance of air emboli.
8. Cool the patient to 20°C. or lower to encourage
solubility of gas in the blood and make the gas bubbles contract. Cerebral ischemia is minimized as well at lower temperatures. Employ slow rewarming as the situation comes under control. However, as in the case presented, if the gas embolism occurs when the patient is nearly normothermic cardiac resuscitation is less problematic and warming should probably be continued. The beating heart will aid in purging air from the cardiac chambers and coronary arteries. The rare but catastrophic massive air embolism emphasizes the rationale for routine total body hypothermia for all cardiopulmonary bypass procedures. The safety period for unanticipated circulatory arrest is extended by hypothermia.

9. The anesthesiologist should discontinue ventilation with nitrous oxide gas immediately, since N₂O equilibrates with an embolized gas bubble rapidly causing it to swell further. Ventilation should be with 100% oxygen because of oxygen's high solubility coefficient and its beneficial effect in limiting organ ischemia. The oxygen (and carbon dioxide) in a gas embolism bubble lodged in a cerebral or coronary artery is absorbed relatively rapidly. However, nitrogen and nitrous oxide are poorly absorbed and act as if they were embolized glass beads or thrombus causing ischemia of the tissues supplied by the blocked artery.

10. The lungs should be vigorously ventilated to expel air from the pulmonary veins to vent via left heart or aortic root vents preferably connected to suction to increase their efficiency by amplifying their effective vent surface area.

11. Administer 2 to 4 grams of methylprednisolone and/or 20 mgs. of dexamethasone intravenously to reduce cerebral edema and aid in protection of organs from ischemia by cell membrane and lysosomal stabilization. Continue for 72-96 hours post-operatively. Some groups, including our own, routinely add 2 grams of methylprednisolone to the priming solution of the pump on all adult open heart cases as prophylaxis.

12. Administer 25 grams of mannitol intravenously and begin infusion of glycerol for 48 hours to help reduce cerebral edema and aid in diuresis of crystalloid prime. Glycerol (1.2 grams/Kg./24 hours) is safer than mannitol (1.0 gram/Kg./24 hours) since glycerol is metabolized to glucose by the liver and does not require renal excretion which might be impaired in a critically ill patient for the initial several postoperative days. Some groups, including our own, routinely add 1 gram/Kg. mannitol to the prime of the pump for prophylaxis.

13. Do not employ profound barbiturate anesthesia (artificial coma). Some authors have recommended this modality for its cerebral protective effects; however, high dose barbiturates are myocardial depressants and may retard purging of coronary air emboli. Instead, aim for early arousal and return of normal mentation. Treat seizure activity symptomatically with diphenylhydantoin, diazepam or low dose barbiturates. The rapidity of return of consciousness and normal neurologic status after brain insult is the single most significant prognostic indicator as to ultimate neurologic recovery. Therapeutic barbiturate coma obfuscates the clinical picture and confuses adjustment of therapy. The role of calcium channel blocking drugs in minimizing brain damage after cerebral ischemia remains to be elucidated.

14. Packing the head in ice is probably of dubious value in protecting the brain of an adult because of the thickness of the skull and the presence of scalp hair. In infants and children it may provide some added protection but the ears must be carefully padded to avoid frostbite injury.

15. Consider deployment to a hyperbaric chamber with 6 atmospheres of oxygen and decompression over 38 hours as recommended by Pierce and others. However, most community hospitals are remote from a facility with a hyperbaric chamber. Transfer of a critically ill post-open heart surgery patient with all the attendant paraphernalia and personnel required to support such a complex patient represents a logistical challenge of the highest order. Also to be considered are the medicolegal consequences implicit in transferring the patient to a new care team, at a remote location, since the patient's family might erroneously interpret this as "abandonment." In my opinion, the team that has created the problem has the highest motivation to salvage the clinical situation and to provide total support, medical and psychological, to the patient and the patient's family for what frequently turns into an extended complex hospitalization for an iatrogenic catastrophe even when the outcome is satisfactory. The hyperbaric chamber, although the treatment of choice for industrial and recreational air embolism accidents (diving accidents most commonly), is unfortunately not a very practical treatment modality in most instances when the victim of air embolism is a cardiac surgery patient.

16. Don't ever give up or discontinue resuscitative efforts in the wake of massive air embolism unless, of course, the patient expires or is diagnosed as having brain death. Complete the operation, follow the above recommendations and, more often than not, the patient...
will recover. This has been amply documented by numerous reports over the years.

17. Finally, the cardiac surgeon and fellow perfusionists have a responsibility to maintain close contact and lend needed support to the unfortunate perfusionist who experiences the potentially devastating complication of massive air embolism. Like the experienced swimmer who drowns, air embolism accidents can strike the experienced and confident perfusionist as well as a neophyte. The loss of the services of a competent perfusionist in the wake of a devastating pump accident only compounds the tragedy and can usually be prevented by intense and sincere support through the crisis and its aftermath.

**Summary**

In conclusion, the best treatment is in the prevention of massive air embolism. Numerous devices and techniques have been described in the 30 years of clinical open heart surgery for this purpose.

At least on an annual basis the entire open heart surgery team should rehearse a “battle plan” for massive air embolism, for this complication can occur in spite of the team’s best efforts to prevent it. If and when this disaster strikes, the prepared team is best equipped to deal with the dramatic and frightening events in the open heart operating room. If the outcome is unsuccessful in spite of execution of the above “battle plan” at least the team can feel that it did its best. This alone is worth the effort expended.

**References**