The Harmful Effects and Treatment of Coronary Air Embolism During Open-Heart Surgery

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ABSTRACT **Residual coronary air embolism after heart-lung bypass is an occasional cause of poor myocardial contractility and low cardiac output. To quantitate the amount of myocardial depression from given amounts of air and to explore the most efficient way to remove coronary air, 19 dog experiments were carried out. During extracorporeal circulation, balloons were inserted into the right and left ventricular cavities to measure isovolumetric myocardial contractility. Small amounts of air injected into the aortic root caused transient myocardial depression with rapid recovery. Repeated injections of small amounts of air produced an additive effect—more depression and slower recovery with each injection. A pure peripheral vasoconstrictor was not as effective as an inotropic drug such as ephedrine or isoproterenol in improving contractility. By far the most effective method of removing air from the coronary arteries and improving contractility and color of the myocardium was to increase the perfusion flow rate for one minute to one and one-half to two times normal. Large amounts of foam appeared from the coronary sinus when flow rates were increased, and hearts intractable to electrical defibrillation became pink and responded to a single shock.**

Coronary air embolization has long been a recognized hazard of open-heart operations, and numerous techniques have been devised for its prevention, including ventricular suction vents, aortic needle vents, induced ventricular fibrillation, and induced mitral insufficiency. It has been established that a small amount of air in a major coronary artery can cause a serious depression of ventricular function [1–12]. It has been generally assumed that the presence of coronary air is not of serious consequence if it occurs during artificial support of the circulation by heart-lung bypass and that it will be forced on through by the artificially maintained systemic pressure, causing little permanent damage. Therefore, this study was devised (1) to quantitate the effects of measured amounts of coronary air on myocardial contractility during bypass; (2) to determine the best

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method of removing air from the coronary circuit; and (3) to determine if residual depression of contractility remains after as much air as possible has been removed with the help of the pump.

Methods

Mongrel dogs weighing 10 to 15 kg. were anesthetized with chloralose, 100 mg. per kilogram of body weight, intubated, and artificially ventilated. Chloralose hyperreflexia was controlled by succinylcholine given intravenously as necessary. The heart was exposed by a bilateral transverse fifth intercostal incision, and bypass was established after caval and femoral artery cannulation. A disposable bubble oxygenator was used; a heat exchanger in the circuit maintained normothermia. The heart was paced a little faster than its normal rate by an electrode sutured to the right ventricle. Balloon catheters were introduced into the right and left ventricles through stab wounds, and each ventricle was vented by another catheter inserted through a separate small stab wound (Fig. 1). The balloon catheters were connected with pressure gauges, and the system was filled with saline solution. After bypass was established at 80 ml. per kilogram of body weight per minute, each balloon was inflated until a volume was found which gave an optimal systolic pressure without elevation of the diastolic pressure beyond normal; this volume in the balloon was then kept constant throughout the experiment. Systemic pressure was recorded from a carotid artery.

In 15 experiments, the effects of injecting small amounts of air into the coronary arteries were studied. One to five cubic centimeters of air was injected into the aortic root as close to the coronary orifices as possible. The

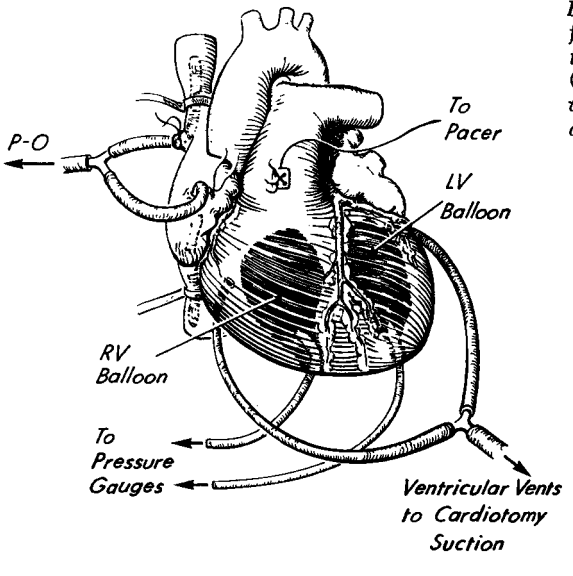

FIG. 1. The animal preparation for studying ventricular contractility by isovolumetric balloons. (LV = *left ventricle;* RV = *right ventricle;* P-O = *pump oxygenator.*)

apex of the heart was tilted up momentarily to ensure embolization. Pressure recordings were made from the isovolumetric balloons until contractility stabilized; then another bolus of air of similar size was injected into the aortic root. This was repeated until a severe degree of depression was achieved or until the heart fibrillated. Different methods of removing air while the animal was still on bypass were then studied. These methods included (1) raising perfusion pressure without changing flow rate (methoxamine hydrochloride, 3 to 20 mg., or phenylephrine, 0.5 to 1.0 mg.); (2) lowering perfusion pressure without changing flow rate (administering a trimethaphan camsylate drip until the pressure dropped 20 to 30 mm. Hg mean perfusion pressure); (3) use of inotropic drugs (ephedrine sulfate, 10 to 50 mg., or isoproterenol, 0.1 to 0.2 mg.); or (4) increasing the perfusion rate to one and one-half or two times the control (120 or 160 ml. per kilogram per minute, respectively). The results of each maneuver were assessed by observing the change in ventricular contraction as measured by the balloons, by the return of pink color to cyanotic areas of the heart, by disappearance of air bubbles from the coronary arteries, or by the appearance of foam in the right atrium from the coronary sinus. If ventricular fibrillation occurred, direct-current defibrillation was attempted.

Results

The injection of a small amount of air (2 to 5 cc.) caused a transient depression of myocardial contraction with a rapid rise back to normal in one to two minutes. Repeated injections caused an increasingly severe decrease

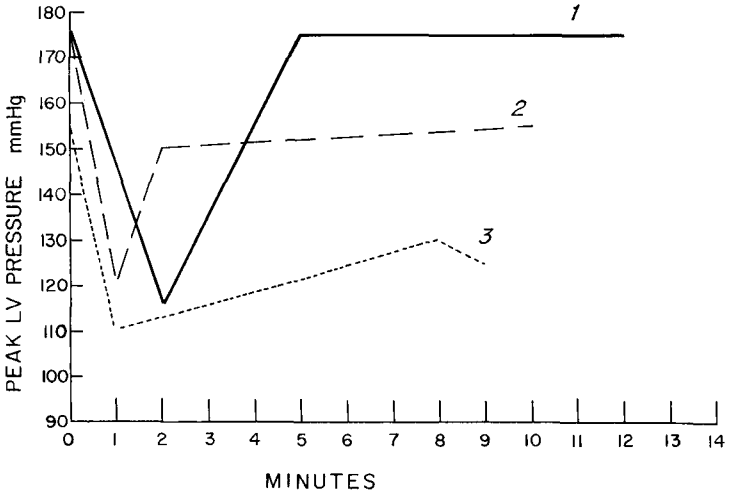

FIG. 2. A typical pattern showing decreasing contractility as indicated by the systolic peak pressure in the left ventricular (LV) *isovolumetric balloon after three sequential injections of 2 cc. boluses of air into the aortic root. Note that the effect of injection* 1 *was transient, with contractility rising back to the preinjection level in four minutes. Injections* 2 *and* 3 *each caused a more severe and sustained depression.*

in contractility, with a slower rise toward control value and with more residual depression (Fig. 2). The vasopressors methoxamine and phenylephrine caused either a slight rise or a fall in contractility of both ventricles, even when these drugs produced a slight rise in systemic pressure. Dropping the perfusion pressure by 40% with a trimethaphan camsylate drip caused a 50% drop in left ventricular contractility. The inotropic agents ephedrine and isoproterenol both caused a prompt increase in contractility and improvement in color, there being no significant difference between the two except that isoproterenol caused a few more arrhythmias in the depressed hearts.

The most rapid and effective improvement in contractility was obtained by increasing the pump flow rate by one and one-half to two times the control. This also increased systemic pressure by 20 to 30 mm. Hg. Cyanotic hearts rapidly became pink, and air quickly disappeared from the surface coronary vessels. In several experiments in which ventricular fibrillation could not be converted by multiple direct-current shocks, increasing the pump flow to twice normal resulted in a rush of foam from the coronary sinus into the right atrium, following which defibrillation was easily accomplished by a single shock.

Comment

It has been the general assumption on the part of cardiac surgeons that air which enters the coronary arteries during cardiopulmonary bypass will be forced through by the externally maintained blood pressure and will cause little permanent damage. This assumption arose from the demonstration by Geoghegan and Lam [3] that an otherwise lethal amount of air in the coronary arteries is compatible with survival if the descending aorta is clamped and the heart manually massaged to force air through the coronary circulation. Our experiments have repeatedly demonstrated that small amounts of coronary air do indeed cause only a transient decrease in cardiac contractility as long as the animal is still on bypass, which suggests that air is forced on through the coronary circulation by the pressure and flow from the pump. Larger quantities of air, or repeated small quantities, however, can cause enough coronary obstruction to significantly depress myocardial contractility even on bypass. In clinical patients, if bypass is discontinued at this point, a severely subnormal cardiac output results.

Our experiments and clinical experience both suggested that simply going back on bypass might not be enough to force air bubbles on through the coronary capillaries. In patients in whom cardiac contractions are depressed, we routinely reinstitute bypass at full flow and inject methoxamine, 2.0 mg., into the perfusion fluid; we have observed an improvement in contractions in these patients simultaneous with the usual rise in systemic arterial pressure. Our experimental animals did not always respond in this way. For reasons we cannot explain, contractility did not uniformly increase

when vasopressors were used. In animals, much greater improvement was obtained by the use of inotropic agents, but maximum removal of air and return of contractility were observed when the pump flow rate was increased. A simple method of temporarily increasing coronary flow and pressure has recently been suggested to us by Drs. John Wilson and Edward Gerety of Albuquerque, New Mexico. If the arterial return is by an ascending aortic cannula, one minute of partial constriction of the aortic arch with the fingers just distal to the cannula will greatly augment both pressure and flow in the proximal aorta and coronary circulation. This has been very effective in removing coronary air and in improving myocardial function. Care must be taken not to constrict the aorta too completely, or myocardial hemorrhage may occur from too great an elevation of coronary pressure and flow.

To summarize our clinical protocol for prevention of coronary air, the following steps are carried out. Ventricular fibrillation is always instituted before the left heart chambers are opened. If the aorta is cross-clamped, the aortic valve is held open or a needle on suction is inserted in the ascending aorta just beyond the valve as the aortic clamp is removed. A left ventricular vent is routinely used and is maintained on gentle suction. After closing the cardiotomy incision, the apex is tilted upward and the heart is allowed to refill with blood while vent suction is increased. The pulmonary veins are manually compressed, and the left atrial appendage is digitally inverted to remove any trapped air. The heart is defibrillated with the apex still tilted up, following which the vent is removed. The ascending aorta is then aspirated at its highest point with a No. 18 needle attached to suction. Bypass is then discontinued.

If cardiac contractions are weak or ventricular extrasystoles develop shortly after bypass is discontinued, the presence of coronary air is assumed to be the cause even though no air is visible in surface vessels. Bypass at full flow (2.2 liters per minute per square meter of body surface area) is reestablished, and a slow drip of isoproterenol is started. If contractions remain weak or the heart remains cyanotic or irregular, coronary flow and pressure are temporarily augmented by gentle constriction of the aorta just beyond the aortic cannula. These maneuvers seldom fail to improve cardiac contractions, and only then is bypass discontinued.

By carefully keeping coronary air to a minimum while the heart is open and by forcing all air on through the coronary capillaries until good contractions and pink muscle result, we have been able to greatly reduce the problem of low cardiac output following bypass, especially after mitral valve operations.

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Discussion

DR. RODMAN E. TABER (Detroit, Mich.): I am sure we are never going to be able to prevent air embolism completely. Drs. Justice, Leach, and Edwards have shown that very small amounts of air in the coronary system can produce profound changes, and other workers have shown that embolic carbon dioxide can produce the same effect.

The work that Dr. Justice referred to by the late Dr. Thomas Geoghegan was published in 1953. He described a method of increasing pressure in the coronary system which closely resembled that described in the clinical protocol by Dr. Justice, and it was found to be effective in experimental animals. I would imagine there is a good possibility that the increased perfusion pressure may be more responsible than flow for flushing coronary air in the experiments just outlined. Possibly Dr. Justice would comment on that.

If a patient who has had an open-heart operation exhibits impaired output after bypass is ended and it is not due to some cause such as a poorly chosen prosthetic valve or incompletely relieved disease, certainly something must have happened during the perfusion that was not going on previously. The patient's cardiac output was sustaining life before the pump was turned on, and it does very little good to ascribe poor output to something such as washing out of the adenosine triphosphate in the myocardium or a long pump run when in fact experience shows that coronary air embolism is often responsible.

Prevention of coronary air embolism is certainly much more desirable than treatment. Air trapping, we believe, occurs when air is caught in the left side of the heart after the heart has been filled and the patient is coming off bypass. It seems a reasonable preventive measure to aspirate the potential trapping sites

while the heart is still in induced fibrillation; it wouldn't do much good, of course, to aspirate it once the heart was defibrillated.

An experiment that we did illustrates the point. In a dog lying on its back with the chest closed, a catheter was inserted into the left ventricle through the aortic valve. Ten cubic centimeters of air was slowly introduced through the catheter into the left ventricular apex, and the catheter was then withdrawn above the aortic valve in order to diminish ventricular irritability. One hour later the air was still lying trapped in the ventricular apex.

DR. BENSON B. ROE (San Francisco, Calif.): First, I congratulate Dr. Justice and colleagues for making an important contribution to the armamentarium for treating a universal complication of open-heart operations.

Second, to augment Dr. Taber's emphasis on trapped air I commend to your attention the work of Fishman, who demonstrated how air becomes trapped in the pulmonary veins. Meticulous though we all are about preventive measures to remove intracardiac air, it is certain that a great deal remains unevacuated. If patients could not tolerate fairly significant numbers of air bubbles, we wouldn't be in business.

Third, I offer for your consideration a routine that we have found useful. We stop the pump briefly just before removing the aortic clamp. This prevents the nozzle effect from churning foam into the air-filled supravalvular space and allows the air-fluid level to rise so that air can be aspirated anteriorly.

DR. EDWARDS: I must emphasize once more the importance of removing all coronary air after going through the preventive measures that are now routine. Over the past few years we have become more and more convinced that if cardiac contractility is not quite normal or if serious arrhythmias develop when bypass is discontinued, they are probably due to residual coronary air; and the old syndrome of low cardiac output after a mitral valve operation was almost certainly due to the fact that air was left in the coronary circulation, even though it was not visible on the surface.

We always watch the heart for a minute or two after discontinuing bypass, and if there is any question of arrhythmia or weakness in contractility we reinstitute bypass at full flow without hesitation. We try an isoproterenol drip first, and if that doesn't improve the rhythm problem or the contractility, we constrict the ascending aorta just beyond the aortic cannula. Routinely this improves the rhythm and contractility. In fact, in the last few years, by being absolutely sure we leave no coronary air, the low-cardiac-output syndrome after heart operations has been almost eliminated.