PULMONARY COMPLICATIONS OF OPEN-HEART OPERATIONS: THEIR PATHOGENESIS AND AVOIDANCE

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PULMONARY complications, acidosis, and overoxygenation are three stumbling blocks to the success of open-heart operations. In one previous report[1] the problem of acidosis, its recognition, avoidance, and therapy, was discussed. In another report[2] the use of the Clark polarograph to avoid overoxygenation of blood in oxygenators was described. In this report, we shall present the thesis that temporary overloading of the pulmonary circulation with blood is the most important single factor in the initiation of capillary damage that marks the beginning of severe pulmonary complications after open-heart operations.

Methods

During the open-heart operation the patient's circulation was maintained by one of four types of pump oxygenators previously described: disposable membrane,[3] Melrose,[4] Björk,[5] Kay and Cross.[6] In many patients the heart was stopped with potassium citrate according to Melrose's technic.[7,8] During the usual postoperative care,[9] chest roentgenograms were made about three hours postoperatively, that night, and during the following days if necessary. If indicated, respiration was aided with positive pressure, during inspiration only, providing a mixture of air and oxygen or pure oxygen with a Bennett Respirator.* This was done via a cuffed tracheotomy tube when the need for more than several hours of respiratory assistance was anticipated.

Pathogenic Factors in Pulmonary Complications

1. *Pre-existing pulmonary vascular disease*, often present with long-standing pulmonary hypertension, seems to predispose to the occurrence of pulmonary

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**Bennett Respirator, manufactured by V. Ray Bennett & Associates, Inc., 320 South Robertson Boulevard, Los Angeles 48, California.*

complications after open-heart surgery. In the last 50 consecutive patients treated with the heart-lung machine at the Cleveland Clinic Hospital, some degree of pulmonary difficulty occurred 11 times. Of those 50 patients, 11 had pulmonary hypertension and in four of them pulmonary complications occurred.

2. *Oxygen intoxication of the alveolar and capillary cells*, from the undiluted oxygen administered by the anesthetist, has been thought possible to occur during the period of total bypass when the lungs are not perfused with blood. Exsiccation of the lungs may occur when they are continuously ventilated with dry gas during the period of total bypass, because no blood goes through them to keep them wet. Kirklin[10,11] and Patrick[12] have adopted the practice of keeping the lungs gently inflated with a mixture composed 50 per cent of helium and 50 per cent of oxygen to minimize the possibility of oxygen intoxication of pulmonary tissue. We have followed their example. The lungs should be kept gently inflated but still, to avoid the drying effect of a "breeze" of gases.

3. *Small children are especially susceptible to pulmonary complications.* One reason is that tiny bronchioles are more easily plugged with mucus or debris. Tracheotomy should be considered, but not done without good indication. The ease with which tracheotomy tubes in infants become mechanically obstructed by malposition or mucus certainly makes them a mixed blessing. Postponement of open-heart operations until the child has reached the age of two years is recommended whenever possible.

4. *Use of too much fluid.* Excessive amounts of mineral solution are present in some types of commercial blood-collecting bottles. Too much electrolyte or dextrose solution sometimes is given to keep the venous infusion flowing. In order to prevent an undesirable decrease in colloid osmotic pressure, Kirklin[10] and Patrick[12] compensate with albumin for all water used. We restrict the infusion of water and electrolyte solutions to small amounts.

5. *Air embolism* to the lungs, originating from the right heart, can easily occur. We have on occasion observed air bubbles emerge from puncture holes in the pulmonary artery following postcorrection measurement of pressure in that vessel. It probably is not so innocuous as many have thought. For example, the biologic lung* is quickly destroyed as an oxygenator because of undue resistance when small amounts of air are permitted to enter the pulmonary artery.[13] Lillehei[13] saw that small amounts of air injected into the pulmonary circulation of dogs resulted in large increases in pressure in the pulmonary artery, lasting about a half hour. South African investigators[14] have produced pulmonary arteriosclerosis in rabbits by repeated intravenous injections of small amounts of air. It may be recalled that slush (a mixture of air bubbles and blood), though it does not increase the resistance when flowing, causes a high resistance when the flow is interrupted.[15] The forces necessary to get slush moving again through the capillary bed may be greater than those available. A clamp on the pulmonary artery while the right heart is open, avoids this complication.

**The dog lung used as an oxygenator.*

6. *Temporary overloading of the pulmonary circulation with blood* seems to us to be the most important single factor. Evidence will be presented which suggests that such overloading, even for short periods of time, may initiate capillary damage with various degrees of extravasation of blood. In the postoperative hours and days the extent of these extravasations will increase by exudation and edema until they seriously interfere with gaseous interchange. Anoxia may then dictate the use of undiluted oxygen which, over prolonged periods of time, tends to add injury to insult.

Four Circumstances That Lead to Overloading of the Pulmonary Circulation

1. *Forward overfilling of the pulmonary vascular bed.* (a) During partial bypass (i.e., before the venae cavae are occluded), sudden overloading of the circulation with blood leads to increased pressure in the pulmonary arteries (Fig. 1A). This cannot occur during the period of total bypass when the venae cavae are tied off and the heart is open. (b) During total bypass, when the venae cavae are tied off but while the right side of the heart is closed, coronary sinus blood flows into the right atrium, and the pressure may build up so that blood is propelled through the right ventricle into the pulmonary artery (Fig. 1B). This becomes critical if "extravagant" coronary blood flow occurs when the occluding clamp is removed from the root of the aorta after potassium citrate arrest or after anoxia of the myocardium. The coronary blood flow may then amount to 50 per cent of the total circulation.[16] It rarely will lead to damage unless pulmonary outflow (as in circumstance 3 below) is impeded and/or unless the heart is arrested so that it cannot pump away the coronary return.

2. *Filling of the pulmonary vascular bed through collateral vessels.* (a) Patent ductus (Fig. 2A). (b) Increased bronchial arteries as in patients with tetralogy of Fallot (Fig. 2B). The occurrence of left heart overfilling first became apparent to us in open-heart operations for tetralogy of Fallot in which elective cardiac arrest was employed. Blood from the bronchial vessels to the left heart spilled through the septal defect until it was closed. On occasion the heartbeat would then return spontaneously because the left heart pressure had reached a level sufficient to flush the coronary circulation and to remove the potassium citrate mixture. Muller, Littlefield, and Dammann[15] have given convincing experimental proof of the importance of collateral circulation. They first provoked the development of collateral vessels by tying off one pulmonary artery; at a later date, during extracorporeal maintenance of the circulation, they clamped both the main pulmonary artery and the root of the aorta, and within minutes the pretreated lung was engorged with blood. Among our last 50 open-cardiotomy patients there were 10 patients each of whom had tetralogy of Fallot, and in two of them pulmonary complications developed.

3. *Retrograde overfilling of the pulmonary vascular bed: excess blood in pulmonary veins caused by impeded venous outflow or by backflow.* (a) Mechanical obstruction of pulmonary veins, or of a single pulmonary vein may occur following surgical attempts to correct transposition of the great vessels or following correction of

atrial septal defects with associated anomalous pulmonary venous drainage (Fig. 3A). (b) Obstruction of the mitral ostium in mitral stenosis, regurgitation of blood in mitral insufficiency or in mitral and aortic insufficiency will cause backflow into the pulmonary veins (Fig. 3B). Aortic regurgitation will not occur when the patient is maintained on the heart-lung machine if the aorta is clamped for arrest, but will commence upon removal of the clamp from the aorta. (c) Dynamic obstruction may occur in ventricular fibrillation or with an ineffective left ventricular beat (Fig. 3C). When the patient is returned to his own circulation, the right ventricle initially may develop a more effective beat than that of the left ventricle. High pressures measured in the left atrium indicate that this may occur after elective potassium arrest.

4. *Overfilling of the pulmonary vascular bed by a combination of the above factors.*

We have analyzed our experiences with more than 140 patients who underwent open-heart operation, and we shall discuss the technical problems that have led to the development of pulmonary complications.

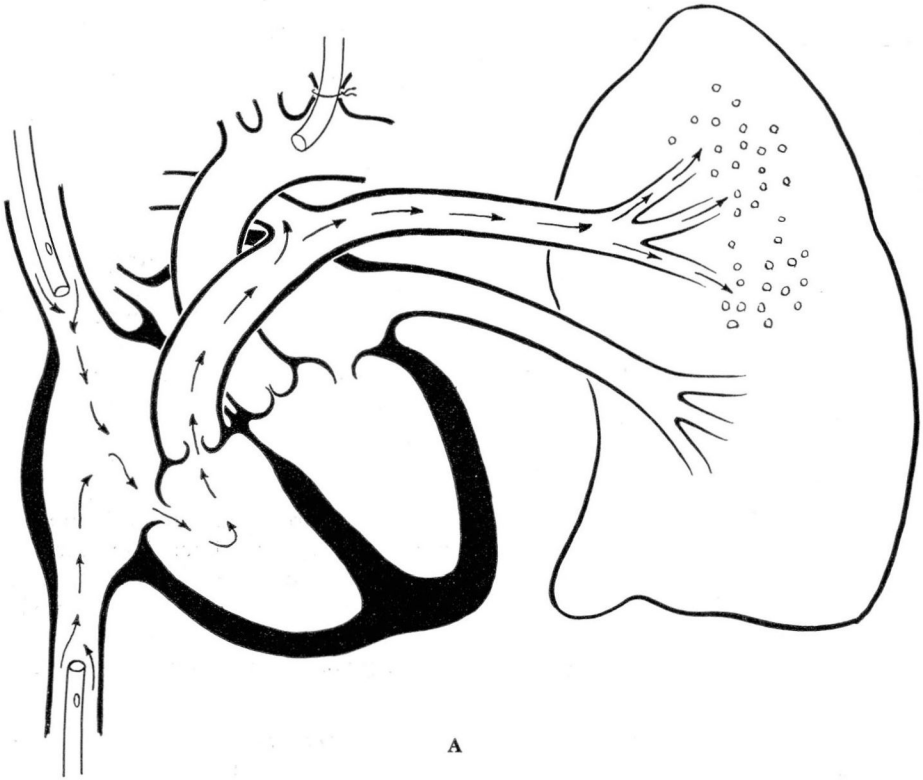


Fig. 1. (**A**) Forward overfilling by increased blood volume of the patient occurs during partial bypass. Increased blood volume in the right atrium is transmitted to the lungs.

Case Reports

Case 1. A six-year-old boy underwent open-heart operation for correction of a large interatrial septal defect. When the heart-lung machine was started and before the venae cavae were tied off, a technical error was made. Although it was promptly corrected, blood was pumped into the venae cavae, distending the right heart and entering the pulmonary arteries. This must have led to rupture of the capillary system in the lungs, since within minutes bloody fluid was recovered from the tracheal tube.

During the subsequent time that the child was maintained on the heart-lung machine, the lungs seemed to recover, and no more blood came from the trachea. However, the damage was done; roentgenograms made shortly after the operation showed many focal densities in both lungs (Fig. 4). The acute changes were consistent with diffuse intrapulmonary hemorrhage and associated edema. A roentgenogram made that evening showed progressively severe changes, and on one made the next day the lungs appeared solidly opaque. The child died in anoxia despite administration of progressively stronger mixtures of oxygen via the positive-pressure (Bennett) respirator.

At necropsy the lungs grossly resembled hepatic tissue, being firm and engorged with blood. A minimum of air-containing tissue was left. Microscopic findings were unusual.

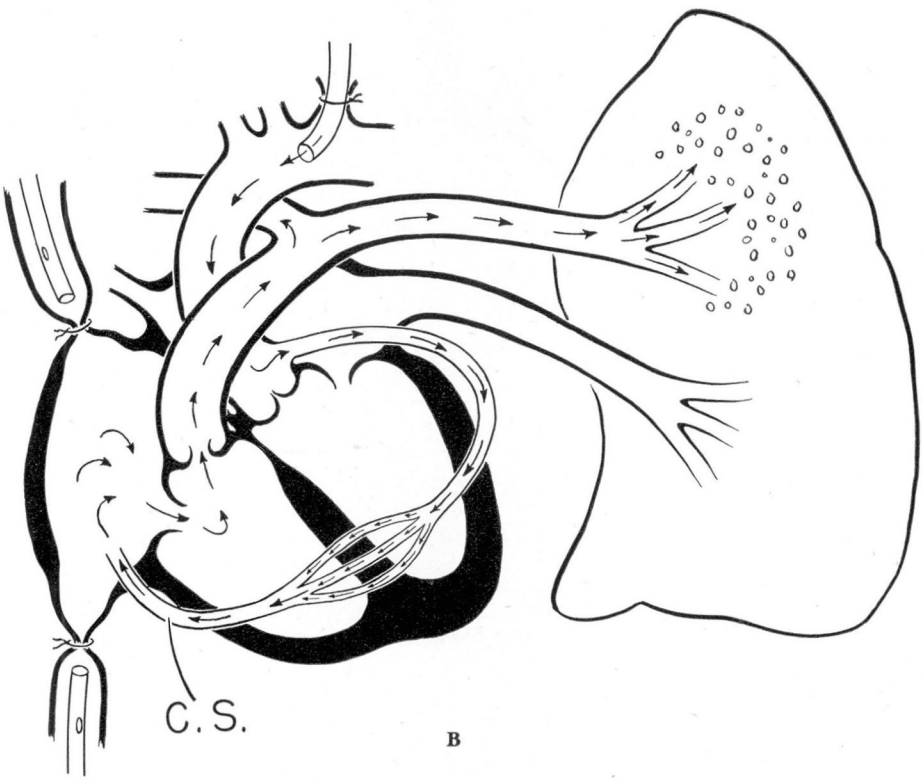


Fig. 1. (**B**) Forward overfilling occurs during total bypass (the venae cavae are tied off). Blood coming from the coronary sinus fills the right atrium, right ventricle, pulmonary artery, and pulmonary vessels.

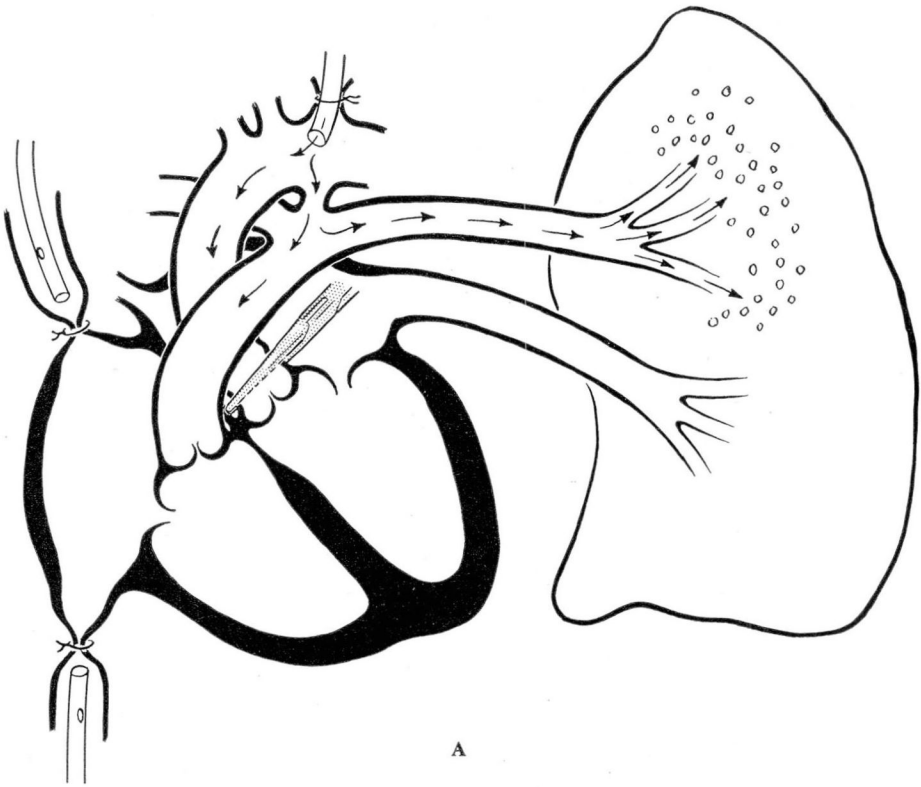


Fig. 2. (**A**) Filling of the pulmonary vascular bed occurs through the patent ductus.

In some areas the alveoli were filled with blood; in others there was a mixture of blood, fibrin, and granular coagulin. Elsewhere there were large masses of fibrin in the alveoli. Most striking, however, were large areas where the fibrin coated the alveolar membrane as in hyaline membrane disease of the newborn (Fig. 5). This reminded us of the tragically correct remark made by the child shortly before he died: "I can breathe, but I cannot get the oxygen out."

For this case there exists no doubt concerning the pathogenesis of the changes. They are due to forward overfilling of the pulmonary vascular bed. The progress of the changes could be followed both roentgenographically and clinically. The temporary improvement of the pulmonary changes during the time that the patient was maintained on the heart-lung machine should be kept in mind, and may become of practical importance when heart-lung machines are able to sustain a human being for several days without inflicting damage to the clotting mechanism or to other functions.

Case 2. A 12-year-old boy, who weighed 45 kg., underwent open-cardiotomy with potassium citrate arrest for repair of anomalous drainage of the right pulmonary veins and an interatrial septal defect. Pulmonary arterial pressure was 42/10 mm. of Hg. It was found that the veins of the right upper and middle lobes drained into the right

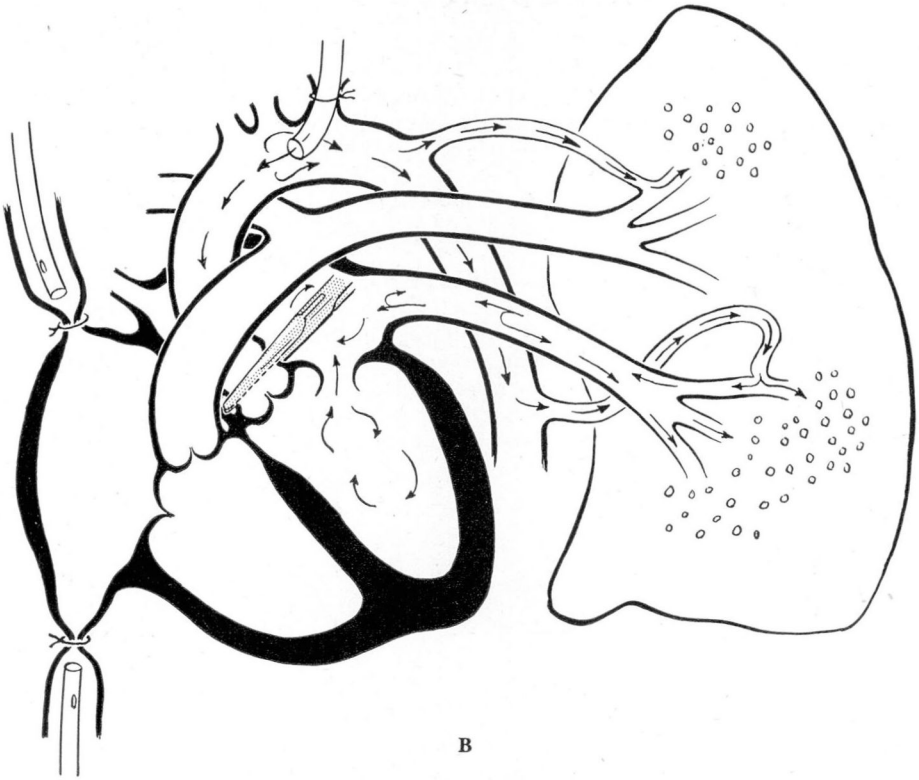


Fig. 2. (**B**) Filling of the pulmonary vascular bed occurs through the collateral blood vessels.

atrium. In order to make them drain into the left atrium, the defect was closed, shifting the atrial wall to the right of the veins.

During the bypass the child was overtransfused with 600 ml. of blood which later was removed. Nevertheless, it could be seen that all of the pulmonary veins were distended and the pressure in the left atrium was palpably high. After the run, the venous pressure was 18 mm. of saline solution, and the mean arterial pressure was low, varying from 40 to 70 mm. of Hg. The postoperative roentgenogram demonstrated massive opacification in the right upper and middle pulmonary lobes and lesser similar changes in the left lung field, probably the result of extravasation of blood (Fig. 6). That evening, fearing thrombosis or mechanical obstruction of the venous drainage, the upper and middle lobes were surgically removed. No mechanical obstruction to the pulmonary veins could be demonstrated. The child died in spite of the pulmonary resection.

At necropsy the same type of engorgement was present in all remaining lung tissue. The microscopic changes of a hemorrhagic pulmonary edema were present in the excised right upper lobe. There was no recognizable fibrin, but a granular coagulum was in the alveoli. Sections of the left lower lobe showed essentially the same changes. The myocardium, while grossly normal, microscopically showed multiple areas of fresh focal necrosis.

It is possible that the pulmonary changes began in the lobes in which the venous outflow was transposed because of some impedance of outflow, but the high left atrial pressure must have been due to left ventricular failure. We consider this to have been retrograde overloading of the pulmonary vascular bed by mechanical and dynamic obstruction. The etiopathogenesis of the myocardial necrosis, undoubtedly the cause of the left ventricular failure, we were unable to determine.

Case 3. A 53-year-old man underwent open cardiotomy for correction of aortic stenosis. While the circulation was maintained with a heart-lung machine, the aorta was clamped and the heart was arrested with potassium citrate for 16 minutes. After correction of the stenosis, a certain degree of aortic insufficiency remained. After the clamp on the aorta was released, blood coming from the heart-lung machine flowed back into the left ventricle, which soon became hard and distended. Ventricular fibrillation occurred and lasted 35 minutes. We assume that in this fibrillating heart the pressure in the left ventricle was further transmitted to the left atrium and, since there was no vent in the left atrium to release it, pressure was built up in the pulmonary veins. The anesthetist noted that bloody fluid came from the endotracheal tube. Many electric shocks were administered before a sinus rhythm became established.

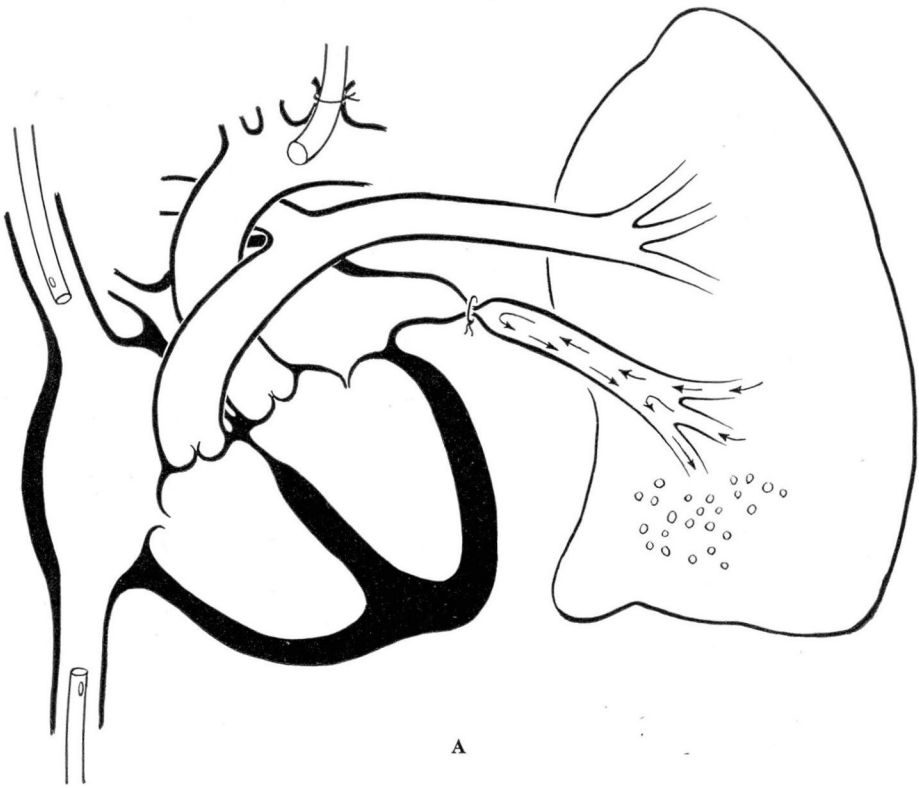


Fig. 3. (**A**) Retrograde overfilling of the pulmonary vascular bed occurs through mechanical obstruction of a pulmonary vein.

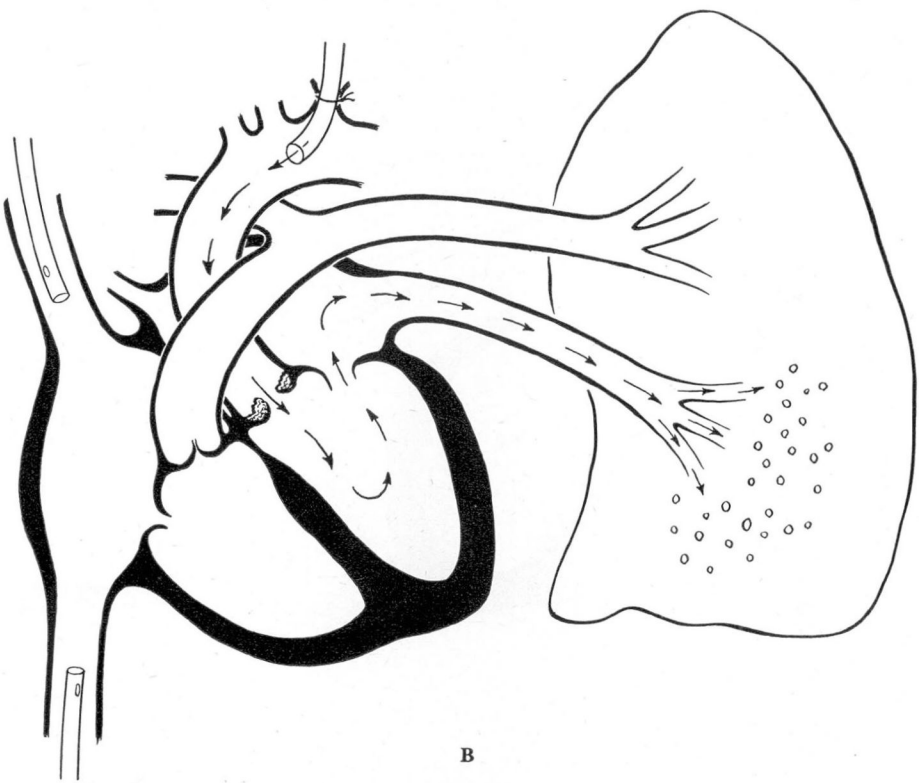
The postoperative chest roentgenogram demonstrated widely distributed areas of increased density bilaterally (Fig. 7). A roentgenogram made several hours later demonstrated progression of the areas of density. In view of the fact that these changes were noted a few hours after the operation, the areas of density probably represented pulmonary hemorrhage as well as pulmonary edema.

This, then, is an example of mechanical (aortic insufficiency) and dynamic (ventricular fibrillation) obstruction. It could have been counteracted if a large drainage tube had been inserted into the left atrium to relieve the pressure.

Case 4. A 9-year-old girl had an interventricular septal defect, severe kyphoscoliosis and mild pulmonary hypertension (47 mm. of Hg). Multiple small hemorrhages in the lungs were observed when the chest was opened. They were considered an indication of a "brittleness" of pulmonary vessels.

After surgical closure of the defect during potassium citrate arrest, the patient had a period of ventricular tachycardia resulting in ineffective circulation that required continued use of the heart-lung machine. An electric shock restored a normal sinus rhythm. In retrospect it seems that backing up of blood in the left side of the heart occurred during the period of arrhythmia which lasted 16 minutes.

Postoperative chest roentgenograms demonstrated an area of moderately increased density involving at least one third of the right lung, the appearance of which is con-

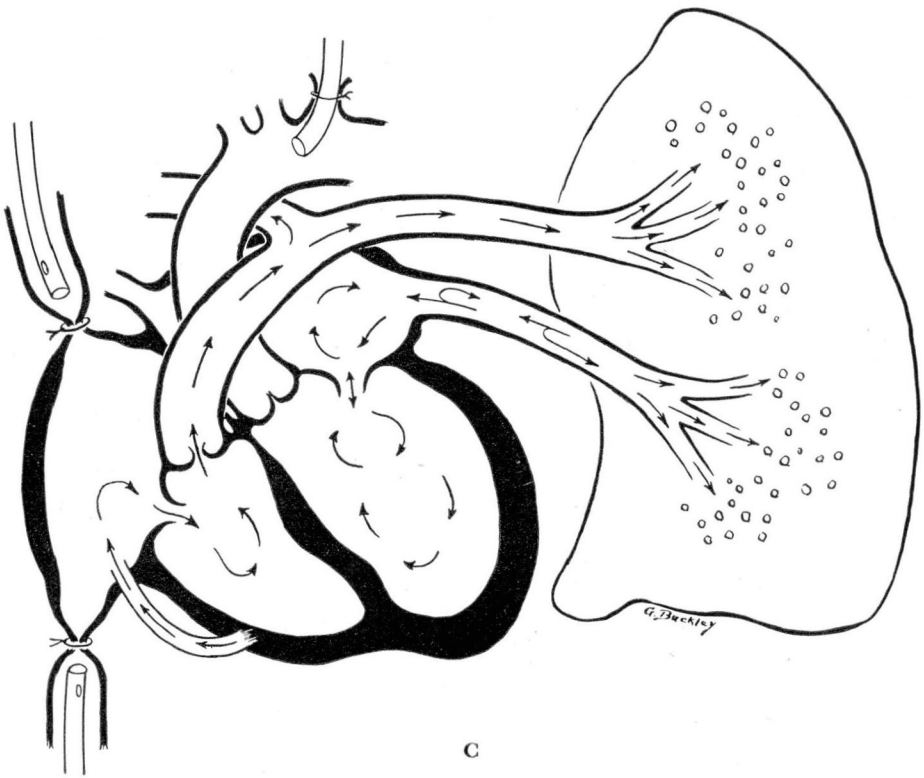


B

Fig. 3. (**B**) Retrograde overfilling of the pulmonary vascular bed occurs because blood from the heart-lung machine flows through incompetent aortic and mitral valves into the pulmonary veins.

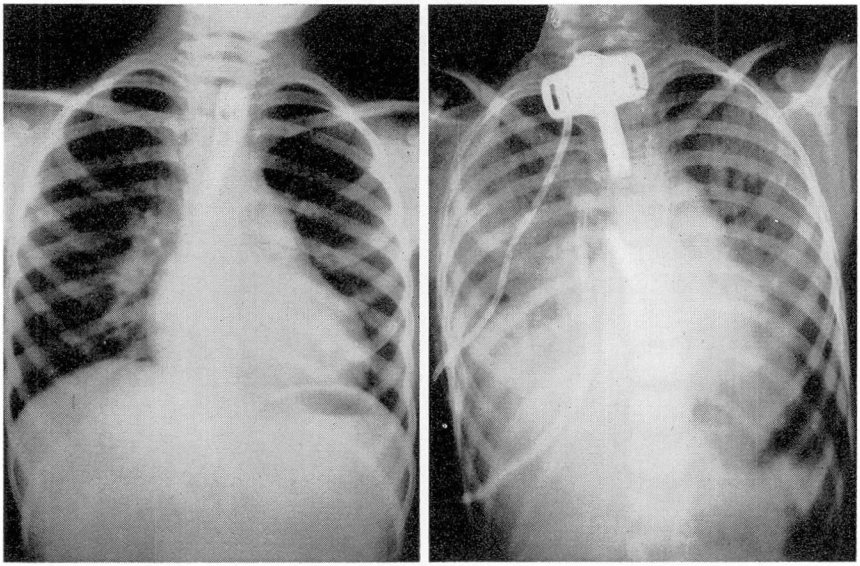
sistent with intrapulmonary hemorrhage (Fig. 8A, B). A roentgenogram four days later showed progression of these changes, which then involved both lungs (Fig. 8C). After a temporary improvement her clinical condition deteriorated. It became increasingly difficult to elevate the blood pH. She became completely dependent on the positive-pressure respirator (Bennett), and died in uremia. The lungs at necropsy were intensely congested. There were focal intraalveolar hemorrhages, and some granular coagula but no fibrin deposits in the alveoli.

Case 5. A 34-year-old man had an interatrial septal defect and high pulmonary artery pressure, 102/46 mm. of Hg. Cyanosis was not present. Pulmonary arteriolosclerosis was proved by lung biopsy. The interatrial septal defect was closed through the opened right atrium. There was no evidence of overloading of the circulation shortly before, during, or after treatment with the heart-lung machine. The duration of cardiac arrest was 13 minutes, and the heart rate restored itself within one minute after release of the aortic clamp. Some air emerged from the puncture hole in the pulmonary artery, after its pressure had been measured, indicating that more may have been present. As there was no catheter in the left atrium, the possibility exists that backing up of blood took place during the recovery phase of cardiac arrest, perhaps enough to initiate changes



C

Fig. 3. (C) Retrograde overfilling of the pulmonary vascular bed occurs because dynamic obstruction due to ventricular fibrillation or inefficient left ventricular beat causes blood from the coronary circulation or from elsewhere to pool in the left ventricle and finally to back up into the pulmonary veins.



A B

Fig. 4. Case 1. (**A**) Preoperative chest roentgenogram shows clear lung fields. (**B**) Roentgenogram made shortly after the operation shows focal areas of increased density throughout both lungs. These acute changes represent diffuse intrapulmonary hemorrhages and associated edema.

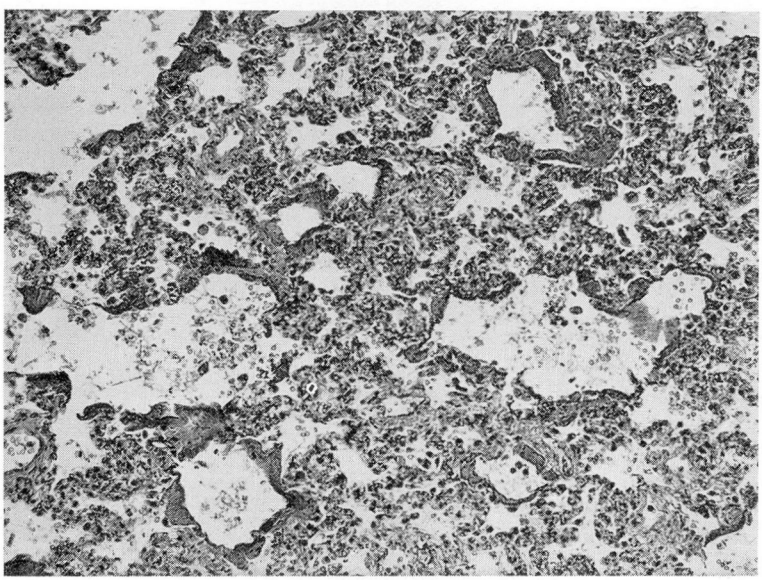
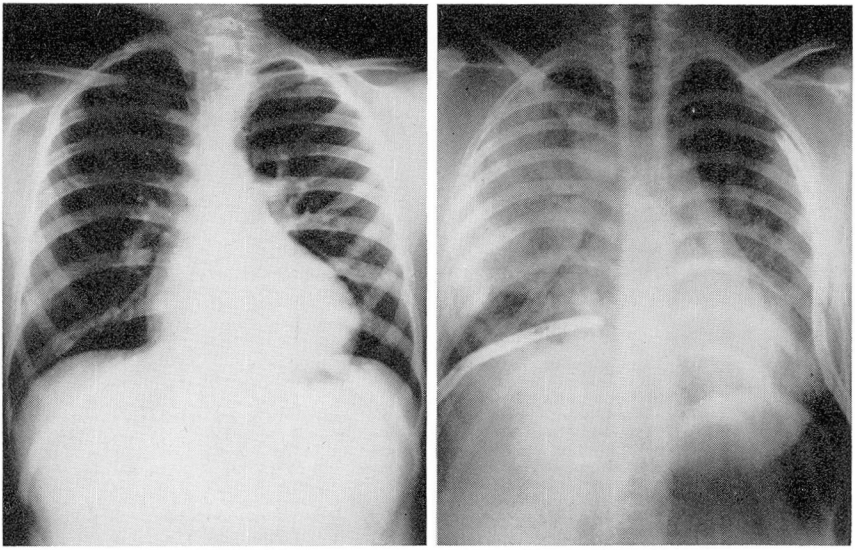
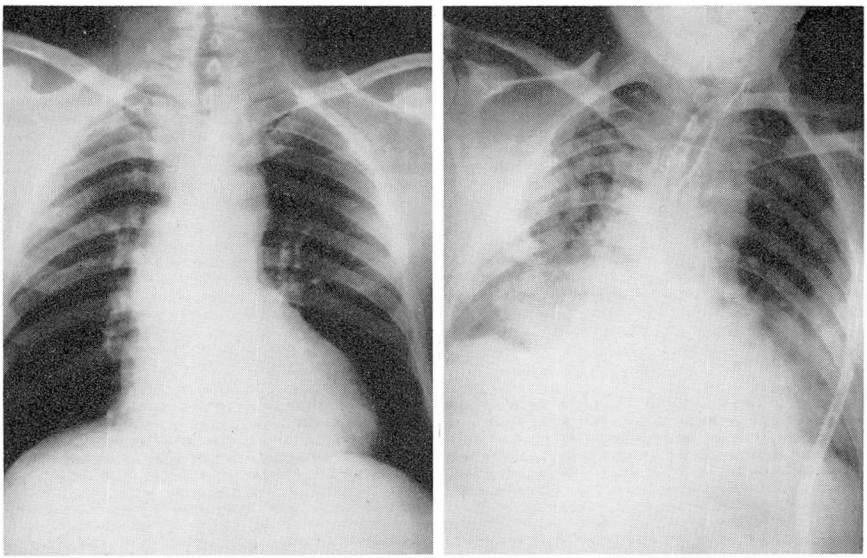


Fig. 5. Case 1. Photomicrograph of lung findings at necropsy: a waxy material, which stains positively with fibrin stains, coats many of the alveolar membranes. Hematoxylin-eosin and methylene blue stain; X 100.



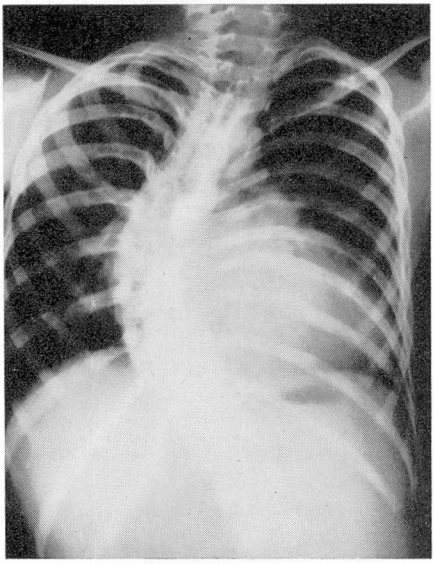
A B

Fig. 6. Case 2. (**A**) Preoperative roentgenogram shows clear lung fields with normal vascular markings. (**B**) Postoperative roentgenogram shows an area of increased density obscuring at least two thirds of the right thorax. Note that lesser changes are also apparent on the left side.



A B

Fig. 7. Case 3. (**A**) Preoperative chest roentgenogram shows cardiac enlargement and clear lung fields. (**B**) Postoperative chest roentgenogram demonstrates areas of increased density bilaterally and widely distributed.



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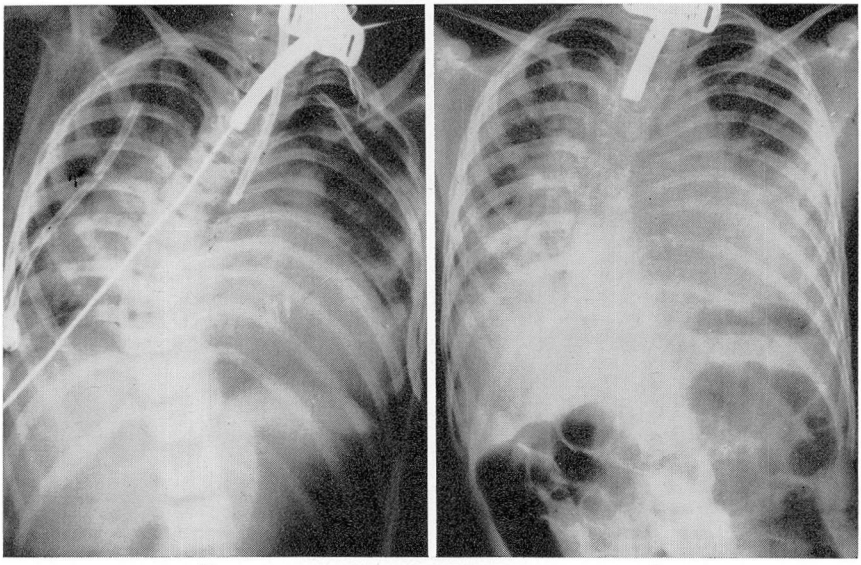


B C

Fig. 8. Case 4 .(A) Preoperative chest roentgenogram demonstrates a marked kyphoscoliosis and probable cardiac enlargement. Lung fields are clear. **(B)** Postoperative chest roentgenogram shows a moderate increase in density involving at least one third of the right lung field, the appearance of which is consistent with intrapulmonary hemorrhage. Two days postoperatively some improvement of the changes in the lower right lung had occurred. **(C)** Progress roentgenogram four days later shows progression of the pathologic changes that now involve both lungs.

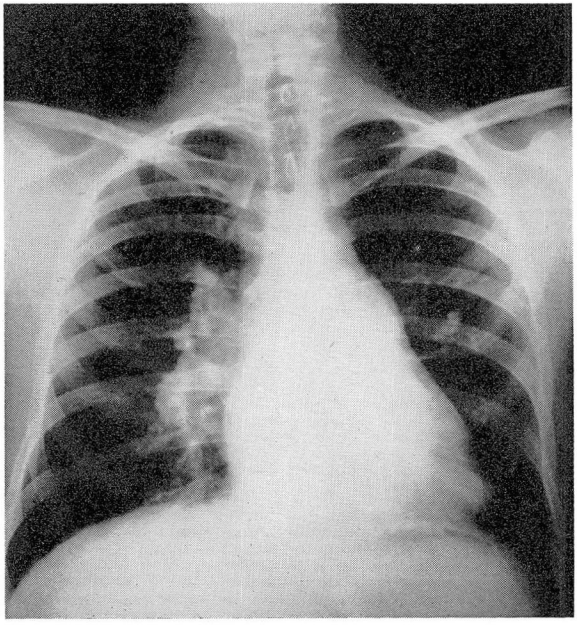
that became more evident the following day (Fig. 9). The immediately postoperative roentgenogram showed no evidence of intrapulmonary hemorrhages, but later, patchy areas of increased density as well as haziness over the left lung were noted. Their development during seven days would tend to indicate extensive edema or inflammatory processes rather than pulmonary hemorrhage. The patient was maintained on a respirator (Bennett) with positive pressure for approximately 14 days, and finally recovered.

This patient was considered a bad operative risk, but was accepted for surgery in view of the poor prognosis without surgical correction. We did not use left atrial decompression at the time. The patient's survival can be attributed to the use of the Bennett respirator.

Avoidance of Pulmonary Complications Due to Overfilling of the Pulmonary Vascular Bed

If the hypothesis is accepted that temporary overloading of the pulmonary circulation can initiate capillary damage in the lungs and a sequence of serious events, every attempt should be made to avoid this overloading, however short the duration.

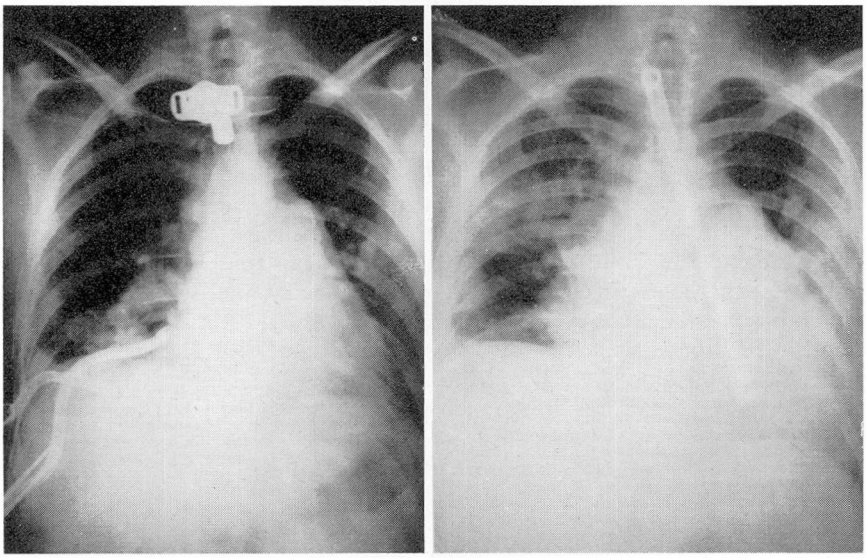
1. *Forward overfilling of the pulmonary vascular bed.* (a) Overfilling by volume changes during partial bypass can be avoided: (1) Rigid maintenance of a



A

Fig. 9. Case 5. (A) A preoperative roentgenogram demonstrates a moderately enlarged heart with prominent pulmonary artery segment and prominent right hilar mass that is considered to be due principally to dilated pulmonary vessels. The periphery of both lung fields as well as the bases are relatively clear.

constant volume of the heart-lung machine will permit increase of the patient's blood volume only by intentional transfusion. The Gibbon type of oxygenator[17,18] and the Clark and Gollan[19] oxygenator have an electronically controlled constant volume. Our own disposable membrane oxygenator[2] has an accurate volume control although it is visually maintained. Mr. Frederick Olmsted has provided our Kay and Cross oxygenator with an automatic volume control. A disadvantage of a rigidly controlled volume of the machine is that blood loss anywhere, unless replaced, will be reflected in decreased output of the artificial-heart pump and, if severe, in reduced circulation, shock, and anoxic changes on the electroencephalogram. We shall accept these disadvantages when overfilling of the vascular bed of the lungs can be avoided with certainty. (2) Prevention of "suck in" is desirable. In some machines, temporary difficulty with venous outflow can lead to negative pressures that suck the walls of the venae cavae into the openings of the venous cannulae, blocking them and leading to volumetric changes. This can be avoided by the use of a venous reservoir in which a slight (4 mm. of Hg) negative pressure is automatically maintained,[17,18] or by the use of an open venous reservoir that can be moved up and down so that the degree of suction can be regulated by the height of the



B C

Fig. 9. Case 5. (**B**) An immediately postoperative roentgenogram shows no apparent change in the cardiac size or silhouette. Increased markings at the right base are partially due to incomplete inspiration and probably are associated with areas of segmental atelectasis. The left lung though not sharply defined is considered clear. (**C**) A roentgenogram seven days later shows a marked increase in the over-all size of the heart. Patchy areas of increased density and haziness over the chest laterally on the left side are noted. It is apparent that these pulmonary changes have progressed. Their delayed development tends to indicate extensive edema or inflammatory processes rather than pulmonary hemorrhage. Increase in cardiac size suggests probable right heart failure.

siphon.[13] We prefer the latter device. (3) Monitoring of central venous pressure. This is an important safeguard. A rising venous pressure suggests blood-pooling in the caval system. As previously stated, however, during total bypass and when the right heart is open, the lungs are protected against forward overfilling even if the venous pressure is elevated.

(b) Accumulation of coronary sinus blood can occur only when the right side of the heart is closed. Releasing of the caval occlusion may or may not suffice for draining the sinus blood out toward the venous cannula of the heart-lung machine; a cannula in the right atrium itself may be necessary, according to animal experiments conducted by Dr. T. A. Akutsu in our laboratory.

2. *Filling of the pulmonary vascular bed through collateral vessels.* An open ductus must be closed before starting, or at the onset of, extracorporeal circulation. Increased bronchial circulation may lead to a large influx of blood into the lungs. We no longer believe that maintenance of free reflux through the pulmonary valve into the open right ventricle provides adequate decompression of the pulmonary circulation; we currently prefer to have a cannula in the left atrium to drain off the excess blood.

3. *Retrograde overfilling*, whether mechanical (obstruction) or dynamic (fibrillation or insufficient left ventricular contraction). A large cannula in the left atrium can prevent retrograde overfilling except that which is due to obstruction of a pulmonary vein (Fig. 10). This cannula is easily inserted from the right side immediately behind the junction of the right atrium and posterior atrial septum.

A blood-filled U tube connected to the cannula indicates the pressure in the left atrium. When there is obstruction, the pressure in the left atrium in a few seconds may rise to 50 cm. or higher on the manometer. Under such circumstances the cannula is promptly vented to the venous reservoir which is under the level of the heart. A constant flow of bright-red blood will be observed if there is obstruction. *Warning:* Negative pressure should not be applied to the cannula while the left heart is open to the atmosphere, even if it is only via an open foramen ovale to the right atrium, because residual blood will be sucked out and replaced by air, resulting in air embolism when the heart beats.

Discussion

As already mentioned, the problems of acidosis and overoxygenation have been recognized. Pulmonary complications in a given case may result from any combination of errors relative to acidosis, excessive oxygenation, and overfilling of the left heart. Although we do not have experimental evidence to support this concept, it is possible that the metabolic changes associated with excessive oxygenation may promote pulmonary vascular fragility and, in turn, make the lungs susceptible to damage from relatively mild overfilling of the vascular bed.

There seem to be cases where each of the above-listed factors played a role in the development of pulmonary lesions.

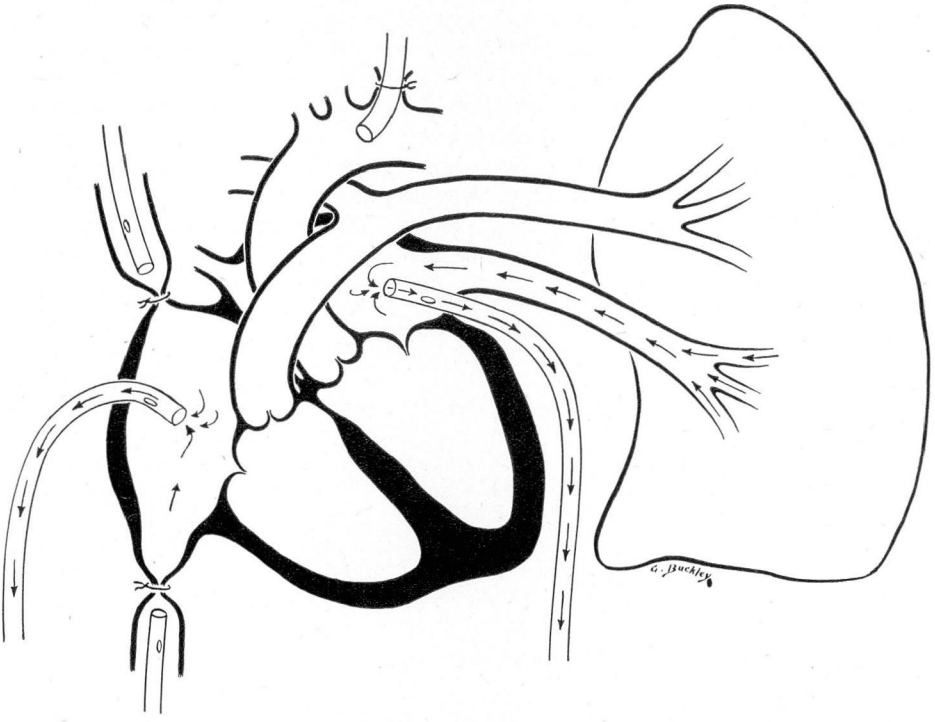


Fig. 10. Safeguard against overfilling of the pulmonary vascular bed: a cannula is in the left atrium. Another cannula in the right atrium may be indicated when the right heart is not opened and the cannulae in the venae cavae do not provide sufficient decompression of the right atrium. This latter cannula has not been used clinically. *Warning:* suction on either cannula when the heart is open must be avoided as it may introduce air into the heart cavities.

Overfilling of the pulmonary vascular bed during operations using extracorporeal circulation is most likely to occur when heart chambers are not open. It can be avoided by placing an adequate drainage tube in the left atrium. After discontinuing the extracorporeal circulation, this same tube should be used to measure the pressure in the left atrium, in order to facilitate recognition of left ventricular failure. Elevated left atrial pressure may indicate that the left ventricle is not ready yet to take over the burden of the circulation.

Summary and Conclusion

The thesis is developed that temporary overloading of the pulmonary circulation with blood is the most important single factor in the initiation of capillary damage that marks the beginning of severe pulmonary complications after open-heart operations.

Pathogenic factors in pulmonary complications are: Pre-existing pulmonary vascular disease; oxygen intoxication of the alveolar and capillary cells from undiluted oxygen administered by the anesthetist when there is no blood going through the bypassed lungs; exsiccation of the lungs as a result of their being continuously ventilated during this period. Small children are more susceptible to having pulmonary complications. Use of too much fluid predisposes to pulmonary edema. Air embolism forming slush in the pulmonary capillary bed will increase the resistance in certain areas in the lungs, but the authors believe that temporary overloading of the pulmonary circulation with blood is the most important factor.

Schema of overloading of the pulmonary circulation:

1. By forward overfilling
   (a) During partial bypass if a sudden increase in the patient's blood volume occurs
   (b) During total bypass by accumulation of extravagant coronary sinus blood in the right side of the heart
2. Through collateral vessels
   (a) Patent ductus
   (b) Increased bronchial circulation
3. By retrograde overfilling
   (a) By mechanical factors
      (1) Pulmonary venous obstruction
      (2) Mitral stenosis
      (3) Aortic and mitral regurgitation
   (b) Dynamic obstruction
      (1) Ventricular fibrillation
      (2) Ineffective left ventricular beat

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