

## PULMONARY AIR EMBOLISM REAPPRAISAL OF THE IMPORTANCE IN OPEN-HEART SURGERY

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### ABSTRACT

Experience with the use of extracorporeal circulation and advancement in surgical technique apparently has lowered the mortality and morbidity following operation for repairing a cardiac defect.

Our early cases of open cardiac operation were carried out on beating hearts without occlusion of the pulmonary artery. It was customary to encounter the problem of the post-perfusion syndrome and it occasionally lead to unfortunate results. Since we have paid closer attention to the separate occlusion of the pulmonary artery when the right heart was open, the respiratory complication and the so called post-perfusion syndrome were less often encountered.

The presented experimental study was undertaken to determine the frequency of hemodynamic as well as pathological changes on the experimentally induced pulmonary air embolism in dogs. In the course of study, increased pulmonary arterial pressure and cardiac arrhythmia were noticed immediately after the injection of the air into the pulmonary artery, and lesion like interstitial pneumonia was observed later.

Through the experimental and retrospective clinical study, it became clear that the prevention of the pulmonary air embolism is equally important as that of the systemic air embolism.

Pulmonary air embolism, like other pulmonary embolism, may cause similar hemodynamic alteration and pathological findings such as pulmonary hypertension, systemic hypotension, cardiac arrhythmia, pulmonary edema and lesion like interstitial pneumonia. These changes are found to be of degree and not of kind<sup>1)2)3)</sup>.

In early cases of open cardiac operations performed on beating hearts without occluding the pulmonary artery so called post-perfusion lung syndrome and occasional fatalities were encountered. Although there have been extensive studies on this syndrome, the cause still remains unknown. In this report, importance of pulmonary air embolism in open cardiac surgery is stressed. From the data obtained on experimental studies and the clinical result, it is

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suspected that pulmonary air embolism is one of the main factors causing the post-perfusion lung syndrome.

#### METHODS

Eleven mongrel dogs, weighing from 5 to 8 kg, were anesthetized with 30 mg per kg body weight of Isozol (pentobarbital sodium) and the lungs were aerated by spontaneous respiration via an endotracheal tube. Under the fluoroscope, No. 6 F polyethylene cardiac catheter was introduced into the right or left pulmonary artery via the femoral vein. This catheter was connected to a pressure transducer and recordings were obtained. A femoral artery catheter was also inserted for systemic pressure recordings. Another set of femoral artery and vein were exposed for blood sampling. Heparin, 3 mg per kg of body weight, was administered.

For lung scanning, two dogs received the macroaggregated human serum albumin labelled with  $I^{131}$  (50 uCi), after 20 minutes following the introduction of the air into one side of the pulmonary artery.

The cortical electroencephalogram and the limb electrocardiogram were recorded on all the animals studied, while 3 ml/kg of body weight of air was introduced into one of the main pulmonary arteries via the cardiac catheter.

#### RESULT

Characteristically, the rise in pressure of pulmonary artery was promptly observed after the injection of air into one side of the main pulmonary arteries, and the highest pressure was reached within following seconds. A concurrent response was noted in the systemic hemodynamic alternations. As a rule, systemic arterial pressure declined, and in one instance, sustained hypotension caused death several hours later. In every instance, pulmonary hypertension was transient in nature, in many cases lasting less than 20 minutes, thereafter returning to the baseline or only slightly elevated level for a longer period up to 30 minutes. Right after the subsequent introduction of the air into the pulmonary artery, pressure followed the same pattern as was observed on the initial procedure (Fig. 1).

Transient cyanosis, decrease in  $paO_2$  and increase in  $paCO_2$ , was observed. Blood pH was usually high. The cyanosis as well as abnormal values of the blood, on gasmetry, returned to normal together with the pressure of pulmonary artery, so it is most likely that these changes are due to transient ventilation-perfusion inadequacy resulting from air embolism in the pulmonary artery or to the humpered pulmonary circulation. One dog, following severe hypotension, expired in the postembolic phase. Post mortem studies revealed diffuse pulmonary edema with slightly hemorrhagic infarction.

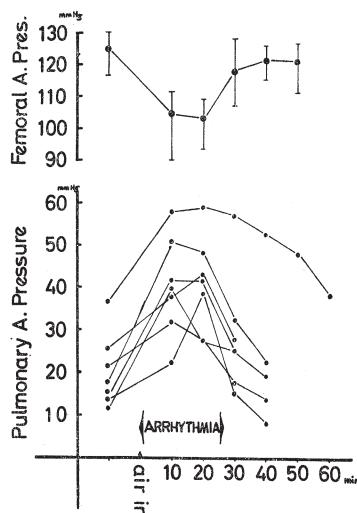


FIG. 1. Femoral and pulmonary artery pressure response to 3 ml/kg of air injected into one side of pulmonary artery. Marked elevation of pulmonary artery pressure and decrease of systemic pressure were constant feature.

Two cases were studied by lung scanning after the introduction of the air into one side of the pulmonary artery, and one case had impaired pulmonary arterial circulation on the affected side.

There was no particular change on electroencephalography.

In every case, electrocardiographic changes similar to those of usual pulmonary embolism, namely, findings of arrhythmia like bigemina, ventricular extrasystole or A-V dissociation were found immediately after the introduction of the air into one side of the main pulmonary artery. In most cases, these changes lasted for several minutes and returned to sinus rhythm spontaneously.

#### *Pathological finding:*

1. Grossly, pulmonary edema with hemorrhagic infarction was found in the air injected lobe, while the other lobe remained intact in the early stage. Immediately after the injection of air into the pulmonary artery, many patchy anemic whitish areas on the surface of the lung were observed. The immediate macroscopic change might have been present on the clinical practice when air enters into the pulmonary artery by open cardiotomy (Fig. 3).

2. Microscopically, major pathologic changes were pulmonary arteritis, intimal fibrosis, damage of elastic fiber and medial thickening. In the early stage, interstitial edema with some hemorrhages was pronounced. As time elapsed, capillary endothelial cells had proliferated in various places so as to cause thickening of the alveolar wall secondary to proliferation of the lipophages. These changes were similar to those of interstitial pneumonia, and frequently found in our early open heart cases resulting in fatality (Fig. 4).

3. Some dogs were sacrificed three or more weeks later. Their lungs showed infarction with abscess formation.

*Clinical observation:*

Previously, our open heart surgery was carried out on the beating heart without occluding pulmonary artery. As a rule, hypotension, dyspnea and high fever over 40°C followed surgery. Eventhough the systemic arterial pressure was maintained within the normal range, these symptoms appeared immediately after the termination of the heart lung bypass, and persisted for 7 hours or longer. Peripheral pulsation was difficult to palpate and the fever did not respond to the antipyretics. During this period, the pulmonary compliance was markedly decreased and oxygen tension did not increase in spite of artificial respiration and oxygen. Despite of extensive and intensive treatment of the patient, even after the patient recovered from the shock condition, arrhythmia ordinarily persisted for another days. Some cases terminated in fatalities, in which pulmonary complication and right heart failure were the commonest cause of death.

The highest temperature, respiratory rate and the lowest systolic blood pressure during the first 24 hours after the operation were compared between the 2 groups: The first group of 20 cases, on which open cardiotomy was carried out without occlusion of the pulmonary artery and the second group of 24 cases with pulmonary artery occlusion (Fig. 2).

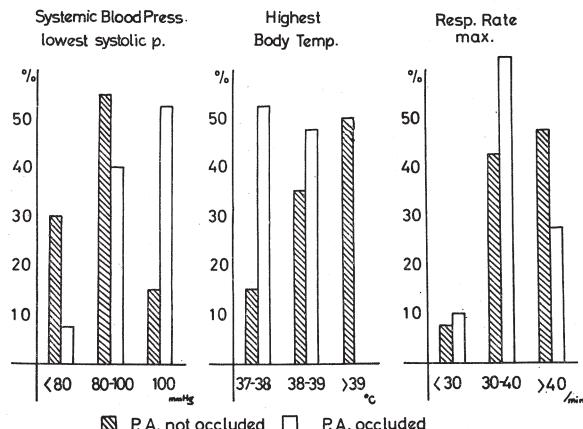


FIG. 2. Lowest blood pressure, highest body temperature and maximum respiratory rate during first 24 hours were compared between 2 group: one group of 20 cases, open cardiotomy carried on without occlusion of the pulmonary artery, another group of 24 cases, carried on with occlusion of pulmonary artery. In the latter group, decrease of hypotension, high fever and tachypnea was noted.

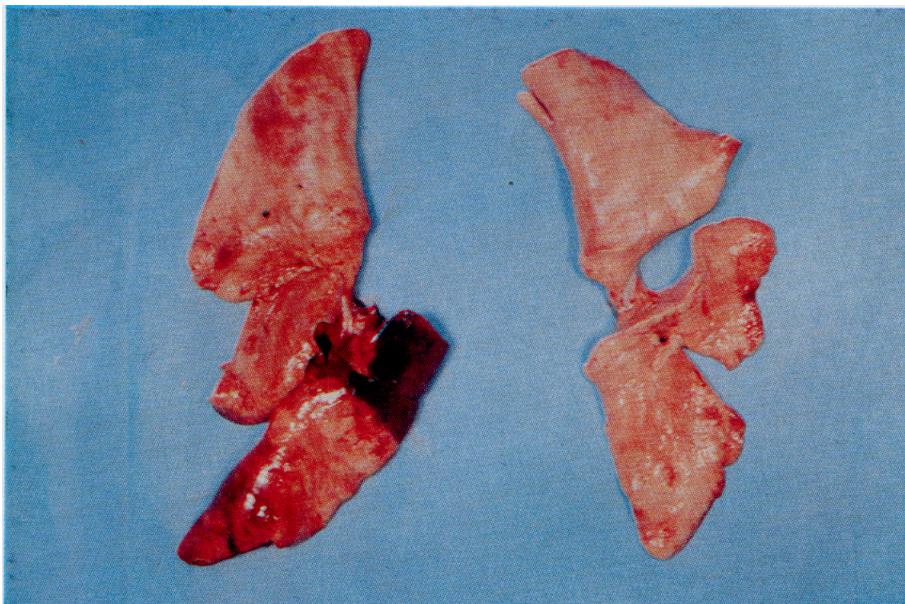


FIG. 3. Gross appearance of the lung, noted pulmonary edema with hemorrhagic infarction on the right lower lobe.

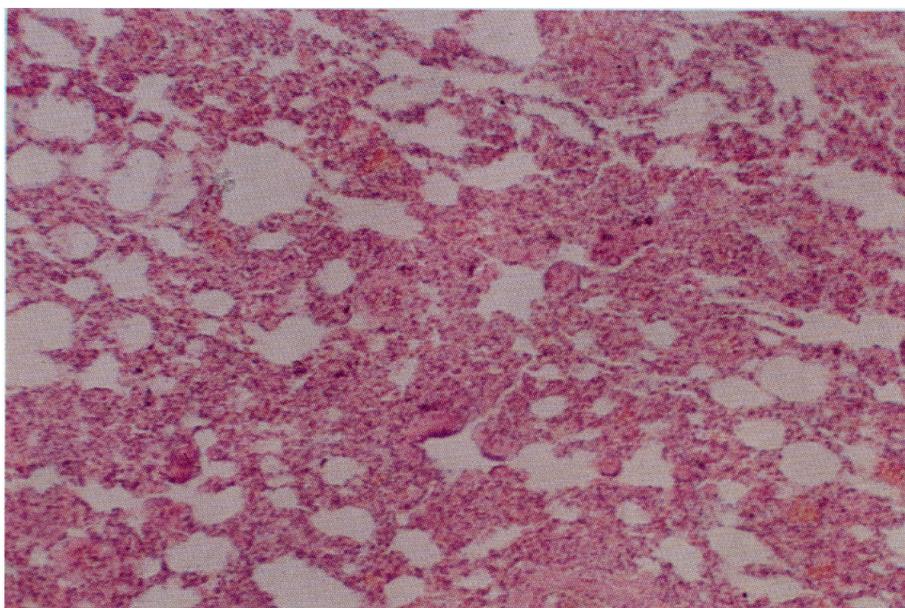


FIG. 4. Microscopic appearance of the lung, which has an injection of air into the pulmonary artery 2 days before.

As it is noted, since much care was taken in preventing pulmonary air embolism, reduction in postoperative pulmonary complications, shock syndrome, high fever and arrhythmia was achieved.

#### DISCUSSION

Pulmonary insufficiency following cardiopulmonary bypass is the most important and commonly encountered complication in open heart surgery<sup>2)4)</sup>. Although there had been extensive studies on the clinical and pathological aspect of this process as well as the influence on pulmonary function, there still remain many problems to be solved as to the pathophysiology of this postperfusion problem. The above complications were the most deteriorating factors especially when the heart disease accompanied by increased pulmonary vascular resistance was surgically treated. Clinical manifestation of the above complications were conspicuous in the postoperative course and had grave influence upon the prognosis.

In 1962, Wright<sup>5)</sup> showed and recently Anderson<sup>6)</sup> demonstrated that repeated introduction of air into the pulmonary artery causes pulmonary hypertension in experimental animals. As Freis<sup>7)</sup> and Wright proved that air bubbles in the pulmonary artery may remain for a long time enough to cause damage of the pulmonary vascular bed. Recently, Barnard<sup>1)</sup> reported that repeated intravenous injections of oxygen, nitrogen and argon gas caused arteriosclerosis of the lungs of rabbits. Arteritis, intimal fibrosis, damage of elastic fiber and medial hypertrophy were another lesions developed. Since these lesions due to the embolism of different kinds of gases were resemble those caused by the blood clot fragment, impaired tissue metabolism of the arteries caused by the substance occupying the arterial lumen to the exclusion of blood and vasospasm are suggested to be the main cause of the pathological change.

In this experiment, the pressure of the pulmonary artery was elevated following the instillation of the air into one of the pulmonary arteries. Instilled air may stay in the pulmonary artery long enough to cause various pulmonary changes, such as pulmonary edema, lesion like interstitial pneumonia and even engorged atelectatic lung. These arterial pressure elevation and pathological changes of the lung were not observed by a mechanical obstruction of pulmonary blood flow alone, thus it is strongly suggesting that the reflex also plays an important role.

Before this investigation, many factors such as drugs, anesthesia electrolytes and trauma were suspected of causing cardiac arrhythmia during and following the cardiopulmonary bypass. But, clinical experiences on more than 100 cases of open heart surgery in this clinic, far less cases of arrhythmia were seen after measures for preventing the air entering the pulmonary artery were taken. As reported on experimental cases, following instillation of air into

the pulmonary artery, arrhythmia appeared in each case. Therefore, it was considered that the pulmonary air embolism is one of the causes of postoperative arrhythmia.

Generally, in normal persons, small amount of the air injected into the pulmonary artery does not seem to cause serious ill effect. But, in the case of pulmonary hypertension, pulmonary air emboli may cause further elevation of pulmonary artery pressure due to the occlusion of the pulmonary vessel. This causes an overload on the right heart ensuring death in some cases.

Clinically, following the conclusion of the heart lung bypass, sometimes multiple whitish spots were noted on the surface of the lung. Number and severity of the cases with peripheral circulatory collapse, cyanosis, systemic hypotension and/or pulmonary complications were greater than expected. Now it became apparent that these postoperative complications were the results of pulmonary embolism, precautions have been taken to prevent pulmonary air embolism when the right heart was open.

Performing open heart surgery, using the heart lung bypass, Lillehei reported that the pulmonary artery and the aorta were occluded at the same time through the transverse sulcus to induce coronary occlusion. For the purpose of preventing pulmonary air emboli, it is felt that the occlusion of the pulmonary artery should be done separately from that of the aorta.

#### CONCLUSION

Pulmonary air embolism, like other pulmonary embolism, causes hemodynamic alternations and pathological findings similar to those of pulmonary hypertension, cardiac arrhythmia, pulmonary edema and lesion like interstitial pneumonia.

Transient increase in the pulmonary artery pressure together with the decrease in the pulmonary function, especially of the decrease in the pulmonary compliance and the right heart strain were noted when the air was introduced into one side of the main pulmonary arteries of 11 dogs.

During open right cardiotomy, the air enter the pulmonary arterial tree when forced inflation and deflation of the lung was performed. Thus, causing pulmonary hypertension and arrhythmia to appear at a critical time of the postoperative course.

Since measures were taken for prevention of the pulmonary air embolism by separate occlusion of the pulmonary artery during open right cardiotomy, the cardio-respiratory complications were less often encountered.

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