POSTOPERATIVE PULMONARY INSUFFICIENCY IN CARDIAC SURGERY AND ITS TREATMENT

YOHTARO IUOMASA, TAKESHI SHIMIZU, TAKASHI KOGURE, NAGAYOSHI OKAMURA, HIROSHI YOKOCHI, and IWAO FUKUTA

1st Department of Surgery, Nagoya University School of Medicine
(Director: Assist. Prof. Itsuro Fukukei)

The most common postoperative complications in cardiac surgery are low cardiac output syndrome, renal shutdown and respiratory failure. Respiratory insufficiency is often unrecognized clinically and effective treatment is undertaken at too late a stage. One of the authors previously investigated postoperative pulmonary function in experimental thoracotomy and cardiopulmonary bypass from the point of view of respiratory mechanics and reported that tidal volume and compliance decreased, and respiratory work, frictional resistance, frequency and minute volume increased in early postoperative period. Pathologic basis of these alteration was that mucus in the large air way was responsible for increased respiratory frictional resistance while mucus in the bronchioles and atelectasis were responsible for decreased compliance. Increased frictional resistance and decreased compliance produced more work to maintain pulmonary ventilation. Atelectasis may increase pulmonary shunt and lead to eventual hypoxia.

In order to explain the cause of postoperative respiratory failure in cardiac surgery, respiratory mechanics, lung volume, pulmonary shunt, He-clearance delay and pulmonary surfactant were investigated on clinical patients.

This study may contribute to the management of postoperative pulmonary insufficiency.

MATERIALS AND METHODS

The patients with cardiac disease at University Hospital of Nagoya were subjected to the pulmonary function test both preoperatively and postoperatively. Surgery on these patients comprises radical operations of atrial septal defect, ventricular septal defect, tetralogy of Fallot, mitral commissurotomy and valvular replacement with prosthetic heart valve in aortic valvular lesion and mitral valvular lesion. The age of the patients ranged from 10 to 40 years of age.
1) Respiratory mechanics

During the week before surgery, preoperative tests were carried out. After the surgery the tests were done four times; 5 hours, 24 hours, 48 hours, and 1 week postoperatively. Tidal volume and respiratory flow which were obtained from wedge spirometer (Model 170, Med. Sience) were recorded simultaneously on multiple monitor recorder, Nihon-Koden Industry with intraesophageal pressure, which was taken through esophageal balloon. The equipment is shown in Photo. 1.

PHOTO. 1. Wedge spirometer, gas collecting bag and multiple monitor recorder with accessory tubings are shown.

Frequency, tidal volume and compliance were derived from this record. A pressure volume loop was displayed on dual beam oscilloscope for respiratory work. Respiratory frictional resistance was measured on the same oscilloscope by means of Mead’s method.

2) Arterial blood analysis

Arterial blood was taken from femoral artery. pH, pCO₂, base excess and pO₂ was measured by Astrup microapparatus while each test of mechanics was being done.

3) Pulmonary shunt

Gaseous isotop Kr was dissolved in cardiobule dye, and this mixture was injected intravenously quickly followed by continuous withdrawal of arterial blood at constant speed. Withdrawal of blood was stopped before recirculation of the dye.

\[
R = \frac{C_{ak}/C_{aD}}{C_{IK}/C_{ID}} \times 100
\]

R: percentage of pulmonary shunt to cardiac output
CaK: concentration of Kr in arterial blood (cpm/ml)
CaD: concentration of dye in arterial blood (mg/ml)
CIK: concentration of Kr in the injectate (cpm/ml)
CID: concentration of dye in the injectate (mg/ml)
The test was done preoperatively and on the day of surgery or within one week after surgery.

4) **Helium clearance delay**
Uneven distribution of inspired gas may occur in the early postoperative period. With Helium open circuit method, gas mixture containing 20% Helium was inspired until equilibrium of gases in alveoli was obtained, then number of breathing that needed to wash out 90% of Helium inspired was measured using catapherometer (Godalt, Holand). Helium clearance delay percentage introduced by Fowler was calculated with the equation below:

\[
\frac{\text{actual average breath no.} - \text{ideal average breath no.}}{\text{ideal average breath no.}} \times 100
\]

Functional residual capacity was also measured along with this test. These tests were carried out preoperatively, 24 hours and one week postoperatively.

5) **Pulmonary surfactant**
Lung extract was obtained from alveoli washed with saline through trachea. Cournand cardiac catheter with 2 cm of cuffed rubber tube on its tip (Photo. 2) was prepared and inserted through endotrachial tube into one segment until it was wedged. The cuff was inflated with 1.5 ml of air and 60 ml of saline was injected to the region. The syringe was pumped three times. Surface tension of the lung extract was measured by means of modified Wilhelmy balance (Photo. 3). When this test was carried out, patients were anesthetized with barbiturate and endotracheal tube was inserted into the trachea to have a secure airway.

**RESULTS**

1) **Respiratory mechanics** (Fig. 1)
Respiratory, minute volume, frictional resistance and power increased in
FIG. 1. In the early postoperative period respiratory frequency, minute volume, frictional resistance and power increased significantly. On the contrary, tidal volume and pulmonary compliance decreased remarkably.

the early postoperative period. Mean value indicates maximum at 24 hours postoperatively, which, however, gradually decreased within one week after surgery. Percent increase of frequency from preoperative value at 24 hours postoperatively is 165%, that of minute volume is 123%.
On the contrary, tidal volume and pulmonary compliance decreased significantly in the early postoperative period. Mean value of tidal volume and pulmonary compliance were minimum at 24 hours after surgery. Percent decrease of tidal volume and pulmonary compliance at that time were 68% and 41.9% respectively. These values showed the tendency of recovery later, but even one week after surgery they were still by far lower than preoperative value.

2) *Arterial blood test*

a) Acid-base balance (Fig. 2)

PH and base excess took almost the similar course postoperatively. On the day of surgery, they decreased remarkably, but within 24 hours they were back to preoperative value and continued to increase. Even at one week after surgery, the value was still higher than preoperative value. However, as to $\text{paCO}_2$ the value was essentially unchanged.

![Fig. 2. PH and base excess decreased remarkably on the day of surgery. 24 hrs postoperatively, the value was higher than the preoperative value.](image)

b) Arterial blood oxygen tension (Fig. 3)

$\text{PaO}_2$ became lowest at 24 hours or 48 hours after surgery. The mean value at that period was 75 mmHg. Minimum value was 57 mmHg. One week after surgery, it recovered to 82 mmHg, which was still low compared with preoperative value.

3) *Pulmonary shunt* (Fig. 4)

Cases of atrial septal defect showed slight increase of pulmonary shunt on
the day of surgery. Two days postoperatively, the values were back to normal. However, in the cases of tetralogy of Fallot, postoperative pulmonary shunt continued to increase until several days after surgery.

4) **Helium clearance delay percentage** (Fig. 5)
Normal clearance delay is below 20%. However, preoperative value in this study ranged from 20% to 85%, which increased in 24 hours postoperatively and within one week became normal except in the cases which had prolonged intrapleural effusion. Functional residual capacity, which was measured at the same time of helium clearance delay, decreased in the early postoperative period. At 24 hours after surgery, the value ranged from 95% to 40%.

5) Pulmonary surfactant (Table 1)

Minimal surface tension of lung extract from normal man is about 2 to 3 dynes/cm. That of cardiac patient was 3.7 dynes/cm. Postoperative minimal surface tension of cardiac patient was higher than preoperative tension, and the mean value was 11.0 dynes/cm (Fig. 5).

Stability index before and after surgery displayed inverse relation to the minimal surface tension (Fig. 6).

### Table 1. Surface tension of lung extract from normal lung, from preoperative cardiac patients and from postoperative cardiac patients is shown

<table>
<thead>
<tr>
<th>Name</th>
<th>Disease</th>
<th>Normal</th>
<th>Preoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>S.T.</td>
<td>S.I.</td>
<td>S.T.</td>
</tr>
<tr>
<td>I. Y.</td>
<td>P.S.</td>
<td>1.3-46.8</td>
<td>1.89</td>
<td>7.9-53.4</td>
</tr>
<tr>
<td>K. K.</td>
<td>M.S.</td>
<td>2.6-57.5</td>
<td>1.82</td>
<td>8.2-51.5</td>
</tr>
<tr>
<td>T. Y.</td>
<td>T./F.</td>
<td></td>
<td></td>
<td>13.9-54.5</td>
</tr>
<tr>
<td>I. M.</td>
<td>P.D.A.</td>
<td>2.2-50.5</td>
<td>1.83</td>
<td>7.4-48.5</td>
</tr>
<tr>
<td>K. M.</td>
<td>A.I.M.S.I.</td>
<td>4.1-54.7</td>
<td>1.72</td>
<td>12.0-53.1</td>
</tr>
<tr>
<td>O. Y.</td>
<td>M.I.</td>
<td>0.5-42.1</td>
<td>1.93</td>
<td>15.6-55.6</td>
</tr>
<tr>
<td>T. Y.</td>
<td>T./F.</td>
<td>7.8-49.7</td>
<td>1.45</td>
<td></td>
</tr>
<tr>
<td>N. A.</td>
<td>T./F.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>1.95-52.1</td>
<td>1.85</td>
<td>3.7-49.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>11.0-52.8</td>
<td>1.32</td>
</tr>
</tbody>
</table>

**FIG. 6.** Minimal surface tension increased significantly after cardiac surgery.
DISCUSSION

It is almost impossible for the postoperative patient to hold his breath or take forced respiration. Therefore, it is natural that the method to be used after surgery is limited. Study of respiratory mechanics is not only adequate for this mean, but also fundamental for the research of respiratory function. Wedge spirometer was introduced in this study. Simultaneous recording of volume, flow and esophageal pressure was made possible to obtain pulmonary compliance, frictional resistance and respiratory work. Decreased compliance after cardiac surgery may probably be due to atelectasis of the lung or mucus in bronchioles as reported previously. Nature of atelectasis on postoperative period is mainly disseminated microatelectasis which is not usually recognizable on chest x-ray film. The fact that PAO₂ was lowered postoperatively coincides with the finding of atelectasis and decrease in pulmonary compliance. The decreased ventilation may be partly responsible for decreased PAO₂. However, it is considered that over perfusion and poor ventilation in alveolar level may be mainly responsible for it. Increased postoperative pulmonary shunt and delayed Helium clearance which was obtained in this series of study are evidence of increased non-ventilated or poor ventilated alveoli. However, those parameters do not give us clear cut answer of the cause of atelectasis. In the cases that bronchial suction and humidification are inadequate, mucus or blood may plug the bronchioles. The distal region of the lung may collapse. In the postoperative patient care, even though suction, nebulizing and position change were intensively carried out, pulmonary compliance and PAO₂ continuously decreased until 48 hours postoperatively.

Pattle reported in 1955 that pulmonary edema foam is very resistant to anti-foam agents and that bubbles expressed from the lung into air saturated water are stable for long period. He deduced from these observations that the lung contains a powerful surfactant which reduces the surface tension of alveoli nearly to zero. Clements measured surface tension in 1957 as a function of changes in surface area by using Wilhelmy balance demonstrating that when the surface was increased, the tension was relatively high, but when surface area was decreased, the tension fell to 10 dynes/cm and first pointed out that such a reduction in surface tension during deflation of the lung would tend to stabilize the air spaces by permitting them to remain open at low lung volume. Mead reported close relationship between surface tension and pulmonary volume-pressure hysteresis. 1959 Avery measured the surface tension of lung extract from infants, children and adults and confirmed the presence of a very surface active substance. Higher surface tension in lung extract of very small premature infants and infants dying with hyaline membrane disease was explained as deficiency of surface active material. Later in 1961, Tooley and Finley investigated the effect of blood from a pump oxygenator on lung. Blood subjected to long term perfusion was transfused to normal dogs, which
had respiratory distress within 12 hours. Their lung was atelectatic and the minimum surface tension of the lung extract increased strikingly.

In our clinic, pulmonary surface activity was investigated on the cardiac patients. In the early postoperative period, pulmonary surface tension of lung extract increased compared to the preoperative value. Minimal surface tension of lung extract from normal man is below 3 dynes/cm in our technique. Minimal surface tension of preoperative cardiac patients is higher than that of normal man. Surface activity of alveolar lining substance decreases not only after cardiopulmonary bypass, but also after simple thoracotomy. Pure oxygen inhalation reduces pulmonary surface activity. Some anesthetic agents are considered to impair alveolar lining substance. It is reported that pulmonary arterial ligation and bronchial ligation make activity of pulmonary surfactant reduced in that region. There are many factors to reduce the pulmonary surfactant. Consideration can be extended to lysosomal membrane stability. During cardiac surgery involving cardiopulmonary bypass, particularly following inadequate blood flow, lysosomal membrane might be destroyed and lysosomal enzymes might be released. Among those enzymes, acid phosphatase probably destroys dipalmitoyl lecithin which is the main component of pulmonary surfactant. Denaturation of plasma proteins caused by unfolding of the globulin molecule due to cardiopulmonary bypass may increase blood viscosity resulting in aggregation consequently causing sludging of microcirculation. Denaturation of plasma protein also comprises hemolysis and release of free lipids which may bring about fat globulinemia and fat embolization of the lung. Disturbance of pulmonary blood flow causes hypoxia of alveolar cells and inhibits metabolism of pulmonary surfactant. Reduced pulmonary surfactant causes alveoli to collapse. Eventually, pulmonary compliance and paO₂ decrease. In Fig. 7, Block diagram of postoperative development of respiratory insufficiency in cardiac surgery is shown. Recently, in our Clinic, lecithin solution inhalation has been carried out on postoperative cardiac patients with pulmonary distress by means of ultrasonic nebulizer connected with Bird respirator. After inhalation of lecithin solution, pulmonary compliance and paO₂ were significantly ameliorated. In the case that has pulmonary hypertension and is anticipated to have postoperative pulmonary failure in high probability, preoperative administration of lecithin with C.D.P. cholin is recommended (Fig. 7).

SUMMARY

Measurement of pulmonary function were performed before and after cardiac surgery.

In the early postoperative period, respiratory frequency, minute volume, respiratory work, frictional resistance, and pulmonary shunt increased while tidal volume, pulmonary compliance, pH, arterial paO₂ and surface activity of
Development of respiratory insufficiency  
Recovery of respiratory insufficiency

**FIG. 7.** Block diagram of postoperative development of respiratory insufficiency and course of its recovery are shown.

lung extract decreased. Helium clearance was delayed. These parameters were worst between 24 hours and 48 hours after cardiac surgery and gradually returned to normal afterwards. In the cases of death these deteriorated progressively.

One of the main causes of postoperative pulmonary insufficiency is decreased surface activity of surfactant.

Treatment of postoperative respiratory failure is discussed in this paper.

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