The Resistance of Pulmonary Blood Flow and Cardiac Output

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Although many factors have been presented to explain mechanisms of cardiac performances, many unknown factors which would control other cardiac phenomena could possibly be explored. In this report a clinical and experimental work to establish a relationship between pressure loading of pulmonary artery and cardiac output was investigated.

1) Pulmonary Inflation Test
As vascular bed of the lung is attached directly to the alveola, pressure of respiratory space should be immediately transmitted to and influence upon pulmonary artery resistance.

Under general anesthesia tracheal tubing was inserted into a patient and a double lumen catheter was introduced into the pulmonary artery of the patient. Left atrial puncture via the right atrium was performed at the same time. Pulmonary wedge pressure, pulmonary artery pressure and left atrial pressure were recorded simultaneously by a polygraph. Cardiac output was measured by means of dye dilution technique in the patient under artificial respiration in administration of muscle relaxant (Succinyl choline chloride) after keeping alveolar pressure at a constant level one and half minute for obtaining steady state by inflation of the patient’s lung. When the alveolar pressure was remained at atmospheric pressure a difference of pulmonary and wedge pressure was 6 mmHg.

![Fig.1. Staged inflation to alveolen in a patient. Alveolar pressure was risen for about one-half minutes to obtain stable state by the inflation of air through a tracheal tubing under artificial respiration. Cardiac output decreased linearly following the risen pulmonary arterial wedge pressure, left atrial and pulmonary arterial pressure, but even in this occasion, pulmonary arterial pressure did not rise to so high.](image)

**UNILATERAL PULMONARY ARTERY OCCLUSION**

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**TYPE # 4**

![Fig.2. Unilateral pulmonary artery occlusion test in a patient. Right heart decompensation was occurred after 30 second of obstruction of right pulmonary artery by the cause of low functional reserve in left lung. Aortic pressure rose scarcely even in occurrence of heart decompensation.](image)

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**Item:**
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Fig. 3. Cross circulation system
A hind leg of the left dog is perfused by the arterial blood of another dog (dog B).

Fig. 4. The effect of injection of physiological saline solution into pulmonary artery. Injection pressure was 14 kg/cm² and volume was 3 ml/kg.
Top: Systemic arterial pressure of the donor dog.
Middle: Change in perfusion flow of a hind leg of donor dog.
Bottom: Constant perfusion pressure of hind leg.
Diagram of Perfusion System

Fig. 5. Perfusion system of isolated right heart preparation
The coronary blood supply was maintained by femoral arterial blood of donor dog. Right atrium-right ventricle-pulmonary artery circulation was arranged with two reservoirs of which hydrostatic level could be changed respectively.

and a difference of wedge and left atrial pressure was 4 mmHg. And when alveolar pressure was risen up to some level left atrial pressure was not remarkably changed, whereas pulmonary wedge pressure, however, rose rapidly following elevation of alveolar pressure. At the level of 35 cmH₂O in alveolar pressure, wedge pressure approached the level as well as pulmonary arterial pressure. Cardiac output was, however, decreased linearly in related to increased alveolar pressure and at 30 cmH₂O of alveolar pressure the volume of cardiac output became less than 1 liter per minute.

2) Unilateral Pulmonary Artery Occlusion Test
At an occasion of unilateral pulmonary artery occlusion test of a patient right ventricular failure was experienced probably due to lack of necessary functional reserve of non-occluded side lung. In that case cardiac insufficiency appeared without any serious sign of overloading upon pulmonary artery (Fig. 2). As shown in Fig. 2, the pulmonary arterial pressure of the patient during the occlusion test rose up merely to 72 mmHg at maximum. In the above two experiments in 1) and 2), unexpected pressure elevation at right heart failure was assumed to be induced that rapid decrease of cardiac output occurred by some other unknown regulation mechanisms one of which we have postulated as a baroreceptor reflex.

3) Pulmonary Arterial Injection Test
To examine an effect of sudden pressure loading upon pulmonary artery to various hemodynamics, injection of isotonic saline solution into pulmonary artery and cardiac chambers was done by high pressure injection. As shown in Fig. 3, a catheter was inserted into the pulmonary artery of one dog of which femoral artery was perfused by the femoral artery of the other dog. Blood flow of the femoral artery of the recipient dog was drained back into donor dog through jugular vein. Injection of 3 ml per kilogram of isotonic saline solution into pulmonary artery was done by the pressure of 14Kg per cm². Systemic pressure change measured in the descending aorta rose rapidly within 3 to 4 beats after the injection. This phenomenon was thought to be induced simply because of increased of left ventricular stroke volume which was made by the injection of saline into the pulmonary artery. But elevated aortic pressure soon returned to the previous level before injection within 2 to 3 strokes of the heart beat followed by rapid drop of aortic pressure. This

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Fig. 6. The relation between loading pressure of pulmonary artery and right atrial inflow in constant right atrial filling pressure. Right atrial inflow decreased disproportionally to argumented pulmonary arterial loading in any constant atrial filling pressure. At the level of 45–50 cmH₂O pressure loading, cardiac output is almost diminished and rather began regarigation; but in higher loading pressure, cardiac output increased transiently again.

Along with elevation of atrial pressure from 0 to 20 cmH₂O. On the other hand under a condition of constant atrial filling pressure cardiac output was rapidly decreased by the pressure loading of pulmonary artery starting 0 cmH₂O to 70 cmH₂O. At the level of 50 cmH₂O of pulmonary artery pressure cardiac output was almost disappeared irrespective of level of atrial pressure. But at the level of the pulmonary artery pressure a little higher than that mentioned above cardiac output was transiently appeared. This was, however, not acceptable that reduction of cardiac output was resulted from decompensation of the heart by overdistention of the heart muscle because slight elevation of the pulmonary artery pressure beyond that non-output point supplied again minimal transient cardiac output. This evidence that disappearance of cardiac output occurred at the pressure under 50 cmH₂O in the pulmonary artery, was so much interested to discuss. We considered it as a characteristic phenomenon of right ventricular performance mediated by the baroreceptor reflex in right heart. It also shall be understood that low cardiac output syndrom which are occasionally seen after chest surgery would be the same sort of mechanism.

Conclusion
Decrease in cardiac output by the pressure load on pulmonary artery was understood to consist following two items: Decrease of cardiac output was 1) influenced by the lessening of systemic blood flow resistance induced by the baroreceptor reflex in the pulmonary vessel; 2) induced by the specific function of heart action which was mediated by the baroreceptor reflex situated in the right heart.

References
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