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## Oxygen Consumption in Myocardium during Experimentally-induced Aortic stenosis

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### SUMMARY

We investigated changes in oxygen consumption of the myocardium with respect to increase in subvalvular aortic stenosis, in 10 thoractomized mongrel dogs under anesthesia. A balloon catheter was advanced from the carotid artery and positioned under the aortic valve. The balloon was inflated by saline solution to produce subvalvular stenosis. Compared to pre-stenotic level the oxygen consumption remained almost unchanged at left ventricular pressure (PLV) and aortic pressure (PAo) gradient of up to 20 mmHg but showed about 15% increase at 40 mmHg and about 30% increase at 60 mmHg. Coronary blood flow (CBF) also remained almost unchanged at pressure gradient of up to 20 mmHg but was decreased about 10% at 40 mmHg and about 20% at 60 mmHg.

*Key words* : Oxygen consumption, subvalvular aortic stenosis

### INTRODUCTION

Clinical symptoms such as chest pain, dyspnea, and syncope appear with as increase in the extent of aortic stenosis. Insufficient supply of oxygen necessary to maintain the pumping action of the heart is considered as one of the causes of aortic stenosis.

We experimentally induced aortic stenosis in mongrel adult dogs and studied hemodynamics and the myocardial oxygen consumption.

## MATERIALS AND METHODS

The animals studied were 10 mongrel adult dogs, weighing 10-24kg. Intratracheal intubation was performed under intravenous anesthesia with pentobarbital 30mg/kg and a ventilator was used to maintain nearly constant pH and PO<sub>2</sub> in aortic blood.

With the animals fixed in the supine position, the pericardium was incised completely from the center to the apex following a median incision.

A catheter was introduced from the femoral artery into the left ventricle and thoracic aorta and connected to Stetham P23Db transducer for the simultaneous recording of the left ventricular pressure (PLV), left ventricular end diastolic pressure (PLVED) and aortic pressure (PAo).

The aortic blood flow (ABF) of the ascending aorta and the coronary blood flow (CBF) of the anterior descending branch of the coronary artery were also measured and recorded simultaneously with ECG. As shown in Fig. 1, a balloon catheter was inserted from the carotid artery and positioned under aortic valve. A second catheter was advanced via the vena cava and positioned at the coronary sinus.

Physiological saline solution was injected into the balloon catheter located under the aortic valve. When the balloon was inflated slowly, only the PLV rose, causing to a pressure gradient between PLV and PAo.

The aortic blood and coronary venous blood then were sampled simultaneously to estimate the oxygen consumption of the myocardium according to the formula: (aortic blood oxygen concentration - coronary venous blood oxygen concentration) / aortic blood oxygen concentration (A-V) oxygen dissociation / arterial oxygen.

Blood samples were taken before induction of stenosis (control) and while the pressure gradient was maintained for 3 min. at 20, 40 or 60 mmHg.

## RESULTS

Figure 2 illustrates the simultaneous recordings of ECG, PLV, PAo and CBF. Inflation of the balloon was initiated at the time point showed by the arrow. PLV rose markedly and PAo also rose slightly, thus the pressure gradient between PLV and PAo also increased.

On ECG, heart rate (HR) remained also unchanged and no change in ST-T was

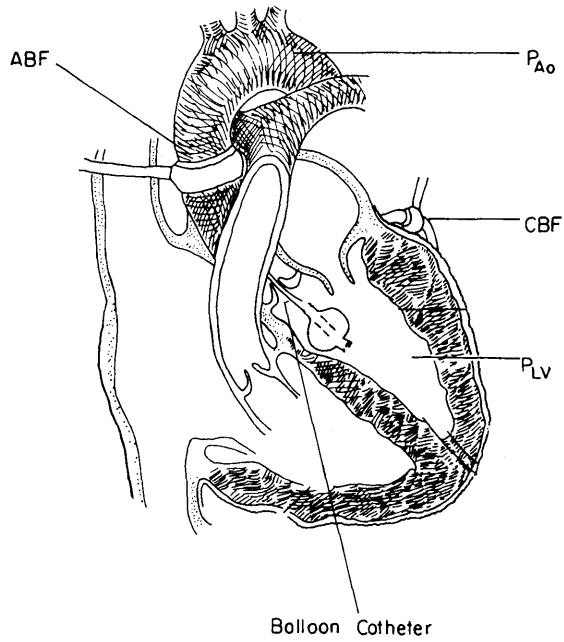


Fig. 1. A balloon catheter was inserted from the carotid artery and positioned under the aortic valve.

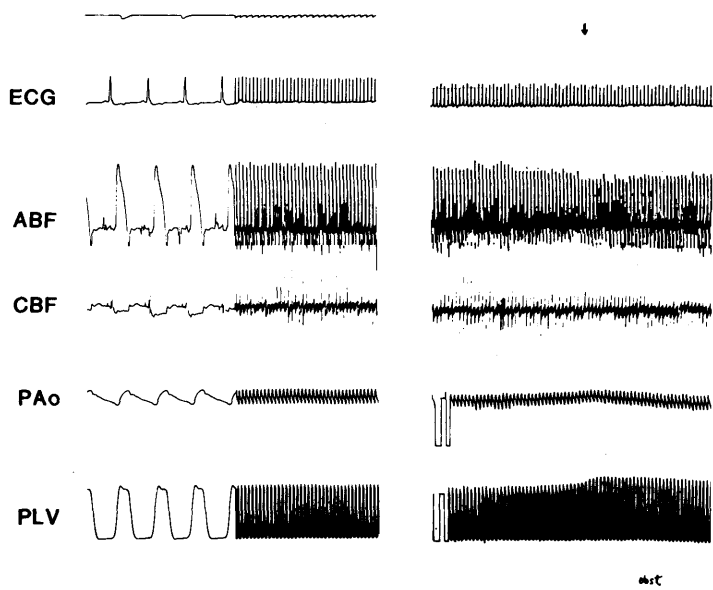
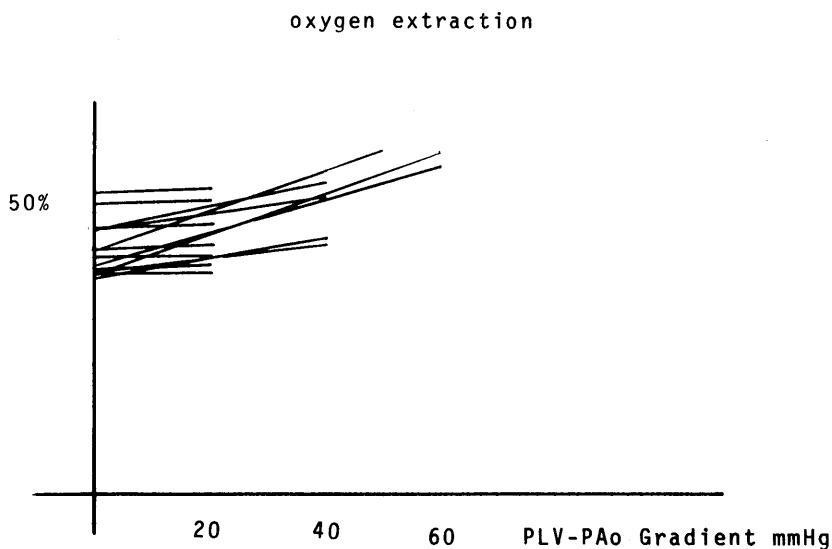


Fig. 2. The simultaneous recording of ECG, P<sub>LV</sub>, P<sub>Ao</sub> and CBF.



**Fig. 3.** The oxygen consumption remained almost unchanged at pressure gradient of up to 20 mmHg but showed about a 15% increase at 40 mmHg and about a 30% increase at 60 mmHg compared with the control level.

observed.

The oxygen consumption remained almost unchanged at pressure gradients of up to 20 mmHg but showed about 15% increase at 40 mmHg and about 30% increase at 60 mmHg compared with the control level (Fig. 3).

CBF also remained almost unchanged at pressure gradients of up to 20 mmHg but decreased about 10% at 40 mmHg and about 20% at 60 mmHg compared with the control level.

PLVED showed no significant rise at pressure gradients less than 60 mmHg.

## DISCUSSION

The pressure gradient between PLV and PAo is used as an indicator to clinically evaluate the extent of aortic stenosis. As the extent of aortic stenosis increase, findings such as chest pain, dyspnea on exertion and changes in ST-T on ECG are observed. This suggested that hypoxia or ischemia of the myocardium results from aortic stenosis.

Using mongrel adult dogs, we induced aortic stenosis experimentally by introducing a balloon catheter from the carotid artery and injecting physiological saline solution to

inflate the catheter, the extent and duration of the stenosis thus produced could be maintained as desired.

However, care should be exercised since arrhythmia might develop depending on the position of the balloon in the left ventricle.

With increasing the aortic stenosis, PLV mmHg rose but PAo mmHg remained almost unchanged, thus resulting in a pressure gradient.

The oxygen consumption of the myocardium can be approximated according to the formula:  $PLV \times HR \times LVEDV$  (LVEDV: left ventricular end-diastolic volume). LVEDV can be replaced by PLVED to some extent.

PLVED did not change significantly even with an increase in the extent of aortic stenosis in this experiment.

Thus, it is possible to derive the formula of  $PLV \times HR \times PLVED = PLV \times HR$ .

Berne et al reported a good correlation ( $r = 0.88$ ,  $P < 0.05$ ) between the rate of rise in PLV mmHg or PLV % and PLV-PAo gradient mmHg at PLV % control =  $0.7$  (PLV-PAo gradient mmHg) +  $99.8$  when a pressure gradient exists between PLV and PAo as a result of experimentally induced aortic stenosis.

From the expression the PLV % control is about 110%, 125% and 140%, respectively, at the PLV-PAo gradients of 20 mmHg, 40 mmHg and 60 mmHg. Since HR remains almost unchanged, the oxygen consumption of the myocardium is about 110% at 60 mmHg assuming that it is 100% when there is no pressure gradient between PLV and PAo.

On the other hand, the oxygen supply to the myocardium may be expressed as (A-V) oxygen  $\times$  CBF. The oxygen reserve of the myocardium is small and CBF is a major factor in the oxygen supply.

In this experiment, however, a decrease in CBF was observed with an increase in the extent of aortic stenosis.

That is, CBF showed little change at a PLV-PAo gradient of 20 mmHg but decreased about 10% at 40 mmHg and about 20% at 60 mmHg.

In general, the supply and demand of the myocardium is presumed to be balanced. When the oxygen demand from the myocardium is increased, the supply is also expected to increase. Accordingly, a decrease in CBF should cause a compensatory increase in (A-V) oxygen, or the oxygen consumption.

As the oxygen concentration of arterial blood varied slightly among mongrel dogs, oxygen consumption was used in the calculations in this experiment. At PLV-PAo gradients of 20 mmHg, 40 mmHg and 60 mmHg, the oxygen consumption remained unchanged,

increased about 15% and 30%, respectively. Thus, the oxygen supply to the myocardium was  $100\% \times 100\% = 100\%$  in the absence of a pressure gradient control of at pressure gradients less than 20 mmHg, whereas, at pressure gradients of 40 mmHg and 60 mmHg, it was 103% ie  $115\% \times (100 - 10)\%$  and 108% ie  $135\% \times (100 - 20)\%$ , respectively. The finding that oxygen supply is increased at pressure gradients of up to 60 mmHg, suggesting that oxygen consumption of the myocardium is strongly influenced by the pressure gradients.

Taking the oxygen supply-demand ratio (S/D) in the normal myocardium as 1, decrease to 0.9, 0.82 and 0.78 were observed at pressure gradients of 20 mmHg, 40 mmHg and 60 mmHg. This demonstrates that, the oxygen demand of the myocardium exceeds the supply as the extent of aortic stenosis increases.

The present study revealed that the oxygen consumption can be increased at least up to 30%.

Since these were acute experiments and since the hemodynamics of the heart under anesthesia cannot necessarily be compared to those under physiological condition, the present data can not be applied clinically.

However, it is clear that when the extent of aortic stenosis increase, CBF decreases and the oxygen consumption of the myocardium increases in an attempt to meet the oxygen demand of the myocardium.

We intend to study the so-called critical point, or point of shift from aerobic metabolism to anaerobic metabolism from the aspect of myocardial metabolism.

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