Water movement between myocardial tissue and capillary blood during and after coronary reactive hyperemia as studied by continuous measurement of colloid osmotic pressure of cardiac venous blood

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Summary. Colloid osmotic pressure (COP) of blood from the great cardiac vein was continuously measured by the use of a membrane colloid osmometer during reactive hyperemia resulting from temporary occlusion of the anterior descending branch of left coronary artery. The COP increased sharply but transiently after the release, then it decreased below the initial level and gradually recovered. It was concluded that a measurable amount of water moved from the capillary blood into the myocardial tissue and then flowed back slowly into the capillary blood.

During renal arterial occlusion water moves from renal tissue into capillary blood according to Starling's law. The haematocrit value of the renal venous blood clearly decreases when the arterial occlusion is released and renal perfusion is reestablished[2]. However, it is still unknown whether water moves between capillary blood and surrounding tissue during coronary arterial occlusion and post-occlusive reactive hyperemia in the myocardial capillary bed. In the present study, tissue-capillary water movement was studied by means of continuous measurement of the COP of the blood from the great cardiac vein. A membrane colloid osmometer was constructed with acryl plastics according to the description of Prather et al.[3] and Aukland and Johnsen[4] with a slight modification, which will be reported elsewhere. 4 mongrel dogs were anesthetized with nembutal (30 mg/kg i.v.). Ventilation of the dog was maintained by a positive pressure respirator (250 ml/kg min). The heart was exposed by a thoracotomy made at the fourth intercostal space. The anterior descending branch of left coronary artery (LAD) was threaded with a thin suture for coronary arterial occlusion (LAD-occlusion). A soft polyethylene tube (OD 2.0 mm, ID 1.4 mm, length 30 cm) was introduced into the great cardiac vein via the left carotid vein (figure 1 A). The proximal end of this tube was connected with a tylon® tube (OD 3.0 mm, ID 1.5 mm and length

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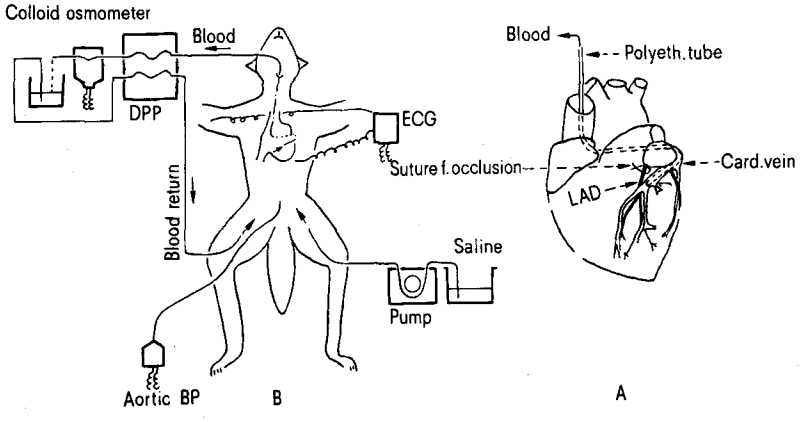

Fig. 1. Schematic illustration of experimental arrangement. LAD and DP Pump stand for anterior descending branch of left coronary artery and double peristaltic pump used for continuous sampling and infusion of cardiac venous blood, respectively.

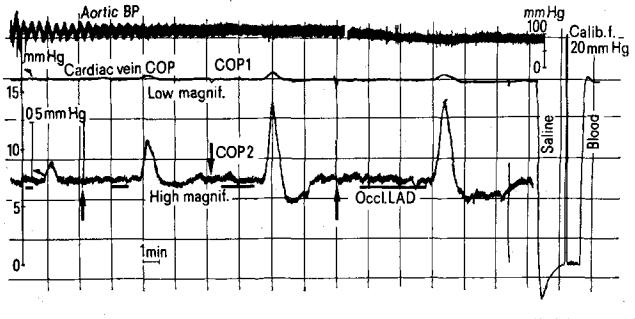

Fig. 2. Typical experimental recording. Curves from top to bottom; aortic blood pressure, colloid osmotic pressure of cardiac venous blood recorded with low (COP 1) and high (COP 2) magnification. Horizontal bars below COP 2 tracing indicate the LAD-occlusion period. Open arrows indicate transient interruption of recording.

30 cm, Imamura Rubber Co, Tokyo), which was fixed to a double peristaltic pump (DPP in figure 1). The proximal end of the tylon tube was connected with the colloid osmometer. After heparinization of the dog, the peristaltic pump was turned on and adjusted to pump blood at the rate of 4.0 ml/min. Thus, the venous blood flowed through the colloid osmometer allowing continuous measurement of COP. The blood sample returned into the right femoral vein through another tylon tube fixed to the DPP. The COP transducer was calibrated with albumin-water solutions whose COP were determined by the use of a freezing point depression osmometer (Knauer, Federal Republic of Germany). Since the COP rose steadily because of the thoracotomy, saline was continuously infused into the left femoral vein at the rate of 5.0 ml/min. Aortic blood pressure was recorded via a polyethylene tube placed in the descending aorta. The complete experimental set-up is shown in figure 1 B.

A sample recording is shown in figure 2, where the curves from top to bottom represent aortic blood pressure (Aortic BP) and COP with low and high magnifications (COP 1 and 2). The recordings were transiently interrupted at the thick arrows. The thick horizontal bars indicate the time period of the LAD-occlusion (30, 60, 120 and 240 sec). When the occlusion was released, the COP signal rose transiently from 30 to 90 sec, then it became lower than the initial level and recovered gradually. The period during which COP was lower than the initial level was longer, when occlusion time was prolonged. The right side of the recording shows where the colloid osmometer system was switched to draw saline instead of cardiac venous blood. The COP 1 curve quickly fell and showed an overshoot, reaching a plateau within 90 sec, while the COP 2 curve went off scale. An electric signal which corresponded to 20 mm Hg of COP was recorded for calibration. Then the colloid osmometer system was again switched for sampling of cardiac venous blood. The COP 1 curve quickly rose and returned to the initial level after an overshoot. During this series of measurements, the peristaltic pump continued to draw blood from the great cardiac vein. In another series of measurement, the peristaltic pump was stopped during the LAD-occlusion. In this case, the COP signal rose slightly during the pump arrest, because red blood cells flowing through the osmometer were deposited on the membrane[5]. But when the drawing action of the pump was restored, the COP signal returned quickly to the initial level. After returning to the original level, the COP signal showed a sharp rise followed by a slower transient drop below the normal level. The amplitudes of COP changes of both the sharp increase (ΔCOP), and the following decrease below the initial level ($-\Delta$COP) as a function of occlusion time, are summarized for 4 dogs in figure 3. A characteristic saturation curve seems to relate ΔCOP and occlusion time (upper half of figure 3). The rate of increase in ΔCOP with increasing occlusion time is reduced with occlusion times longer than 3 min. With occlusion times longer than about 2 min, $-\Delta$COP remained almost constant (lower half of figure 3). The control value of COP observed in 4 dogs was 15.6 $\pm$ 0.6 mm Hg (mean $\pm$ SD).

The myocardial ischemic area caused by the LAD-occlusion was partially perfused by the blood flowing into this region through collateral anastomoses from the coronary circumferential branch. The peripheral perfusion pressure of LAD during LAD-occlusion was found to be in the range of 25 to 30 mm Hg in 2 dogs. This low peripheral perfusion pressure could probably hold the capillary blood pressure at the level which was exerted by the normal perfusion. The low peripheral pressure was directly applied to the capillary vessels, since arterioles in the ischemic area were fully dilated. Thus, no water moved between plasma and tissue during LAD-occlusion in a different manner from the renal arterial occlusion.

The initial increase and the following gradual decrease in COP is interpreted as follows. During the post-occlusive reactive hyperemia, the arterioles were fully dilated. The capillary pressure rose and water moved from plasma into tissue. The saturation curve (figure 2, upper half) suggests a limitation of water absorption capacity by myocardial tissue. As hyperemia ceased, capillary pressure decreased, and the excess amount of water which had been absorbed by the tissue flowed back slowly into the capillary blood. When the sample for COP measurements was changed from blood to saline, the COP signal showed an overshoot. This was caused only by a large change in the quality of the sample fluid. No such overshoot appeared on the COP signal when an albumine solution was added to the sample blood flowing through the osmometer. Therefore, no error due to the overshoot was included in the COP recording of cardiac venous blood. As another cause of COP changes, the effects of hypercapnia in the ischemic area were tested. The swelling of red blood cells exposed to a P_{CO_2} increase of 30 mm Hg in vivo caused a COP change of less than 1%[6], which seems too small to be a major contribution to the COP change observed in the present study.

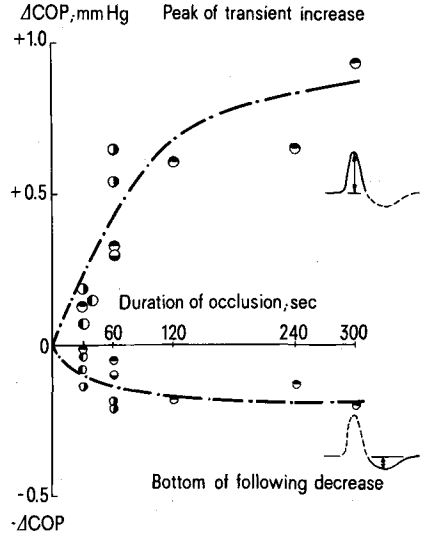

Fig. 3. Relationship between COP changes and duration of LAD-occlusion. Peak of COP increase (ΔCOP, upper half) and bottom of decrease ($-\Delta$COP, lower half) as schematically shown with arrows in the figure are plotted on the ordinate. Since repetition and prolongation of coronary occlusion readily caused arrhythmia, occlusion tests were made only a few times in each dog and the results in 4 dogs are pooled in this figure.

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