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Coronary Blood Flow and Oxygen Consumption by the Heart Following a Hemorrhage

Przepływ wieńcowy i zużycie tlenu przez serce w czasie skrwawiania

Коронарное кровообращение и потребление кислорода сердцем во время кровопотери

Although considerable evidence from hemodynamic (4, 5, 6, 10, 23, 25, 27, 29, 30), electrocardiographic (18) and metabolic (7, 8) studies has been presented by many workers indicating that the impairment of myocardial function is at least in parts responsible for the hemorrhagic shock syndrome, relative little information is available regarding alternation of coronary hemodynamics in this syndrome. Studies on dogs generally reveal a fall of coronary resistance following hemorrhagic hypotension, with either a decrease or no change in the coronary blood flow (8, 11, 14, 22). Present investigations were designed to study events in the left coronary hemodynamics throughout the course of bleeding and hemorrhagic hypotension in anaesthetized dogs.

MATERIALS AND METHODS

The experiments were carried out on 15 dogs weighing 15—22 kg, anaesthetized intravenously with 100 mg/kg chloralose. Coagulation of blood was prevented by the intravenous administration of 5 mg/kg heparin initially and 2.5 mg/kg every hour thereafter. In all experiments artificial respiration by Starling's pump was accomplished. Blood pressure was measured in the femoral artery with a mercury manometer. The rate of the coronary blood flow in the descending branch of the left coronary artery was continously recorded by the photohemotachometer method of Cybulski and Klisiecki. The oxygen content in blood samples taken from the coronary artery and coronary sinus was determined by the method of Van Slyke. Fig. 1 shows the schema of the arrangement used for measuring of the coronary blood flow. The results obtained made it possible to calculate the total volume of

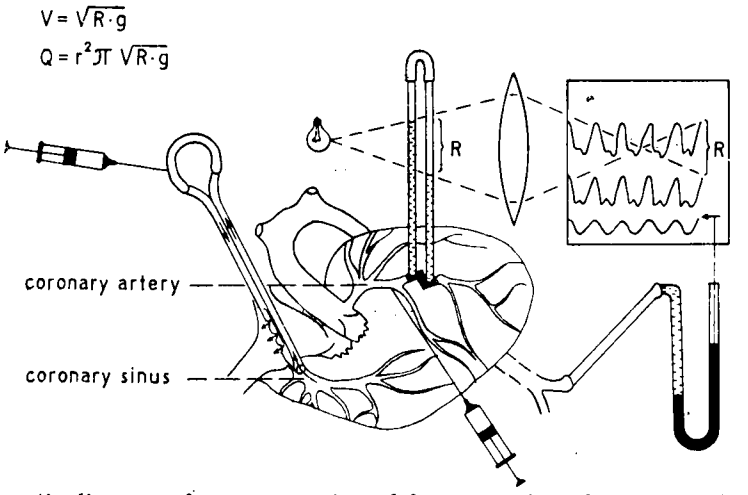

Fig. 1. Schematic diagram of arrangement used for measuring of coronary blood flow; V — velocity of the blood flow in mm/sec, Q — volumetric blood flow in ml/sec, R — difference of levels of air-water manometer, g — earth gravity, r — radius of the cannula of Klisiecki inserted into the anterior ramus descendens a. coronariae sin. Syringes — withdrawing blood samples

blood flow in the left coronary artery per minute and per 100 g of tissue, coronary flow resistance and oxygen uptake by the left ventricle per minute and per 100 g. A graded, intermittent hemorrhage was produced under close observation by bleeding from carotid artery. Two ways of bleeding were used in experiments: the first — slow, 25 — 50 ml every 20—30 min. and the other, more rapid, 100—200 ml every 5—10 min. The total volume of shed blood was 15—30 ml/kg.

RESULTS

Fig. 2 illustrates in graphic form the typical changes in the studied parameters during the course of a slowly performed hemorrhage. As can be seen in the diagram there is a progressive decrease in the arterial blood pressure from 97—15 mm Hg, that is by 85% of the control value. During the early period of bleeding (55 and 30 ml) despite the decrease of blood pressure the coronary blood flow slightly increases (from 77 to 81 ml/ /min/100 g) then slowly decreases until it reaches the values of 53 ml/min/100 g at the end of observation, which is 68,8% of the control. It can be seen in Fig. 2 that during the course of hemorrhage there is a more rapid decline in the arterial blood pressure than in the coronary blood flow response. Thus the coronary flow resistance, which is calculated as the ratio of mean arterial pressure to mean coronary blood flow, decreases gradually. The heart rate does not change at the beginning of hemorrhage and then is slightly slowed. The coronary oxygen arterio-venous difference decreased from 11,55 vol% to 4,48 vol%, which was

caused mainly by a decrease in oxygen content in the arterial blood. Oxygen consumption by the heart dropped from 8,89 to 3,65 ml/min/100 g of the left ventricle.

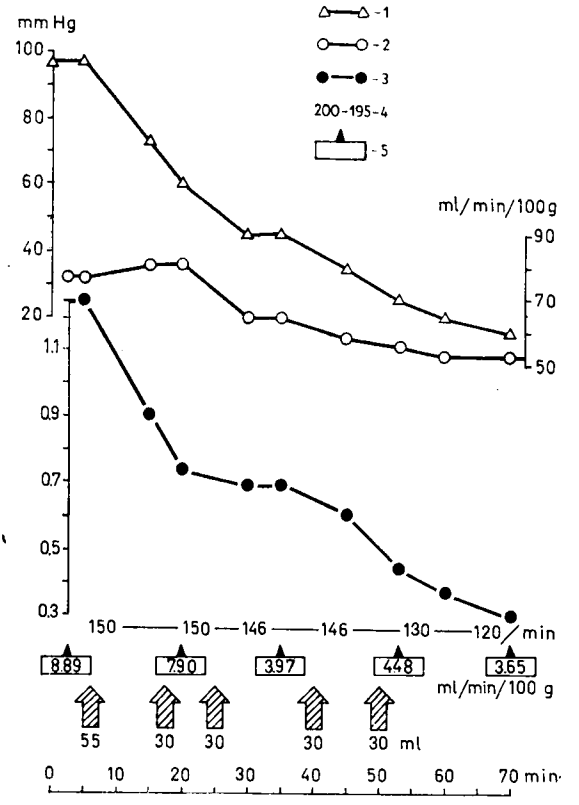

Fig. 2. Graph illustrating trends of blood pressure; 1 — blood flow in the descending branch of left coronary artery, 2 — mean coronary resistance, 3 — in units $\frac{\text{mm Hg}}{\text{ml/min./100 g}}$ 4 — heart rate (numbers —200—195—), 5 — oxygen consumption by the heart in the course of hemorrhage. Arrows with numbers below them indicate volume bleed-out. Time in minutes. Experiment No 6. Dog weighing 18 kg

A somewhat different type of coronary response to hemorrhage is shown in Fig. 3, observed in experiments with larger bleed-out volumes. In this experiment each bleeding was 100 ml and the total volume of shed blood was 450 ml. There was also a graded fall of blood pressure. The coronary blood flow at first decreased and then rose above the prehemorrhagic level after next bleeding of 100 ml. An additional hemorrhage of 100 ml did not change the flow within 30 min. The third bleeding of 100 ml and further, each of 50 ml, decreased coronary blood flow to 42 ml/min/100 g. The mean coronary resistance increased initially

from 0,99 to 1,08 in response to the bleed-down of 100 ml and then progressively dropped following consecutive hemorrhage. After a bleed-down of a total of 350 ml the coronary resistance rose rapidly to the prehemorrhagic level (1,0). This rise was transient and was followed by a fall to about one third of the control values. The heart rate did not change initially and then progressively slowed from 200 to 102/min. The coronary sinus 0_2 content was nearly the same during the bleed-down (7,5—8,0 vol%) but that of the arterial blood continuously dropped (16,5—12,2 vol%). Thus the coronary arterio-venous oxygen difference decreased (9,3—5,1 vol%). Oxygen uptake by the heart initially decreased a little from 8,46 to 8,16 ml/min/100 g and then more rapidly to 2,14 ml/min/100 g.

In experiments in which bleeding was performed rapidly, 200—300 ml each, there was a sudden and deep fall of blood pressure and coronary resistance. The coronary blood flow lowered too. The heart ratio initially

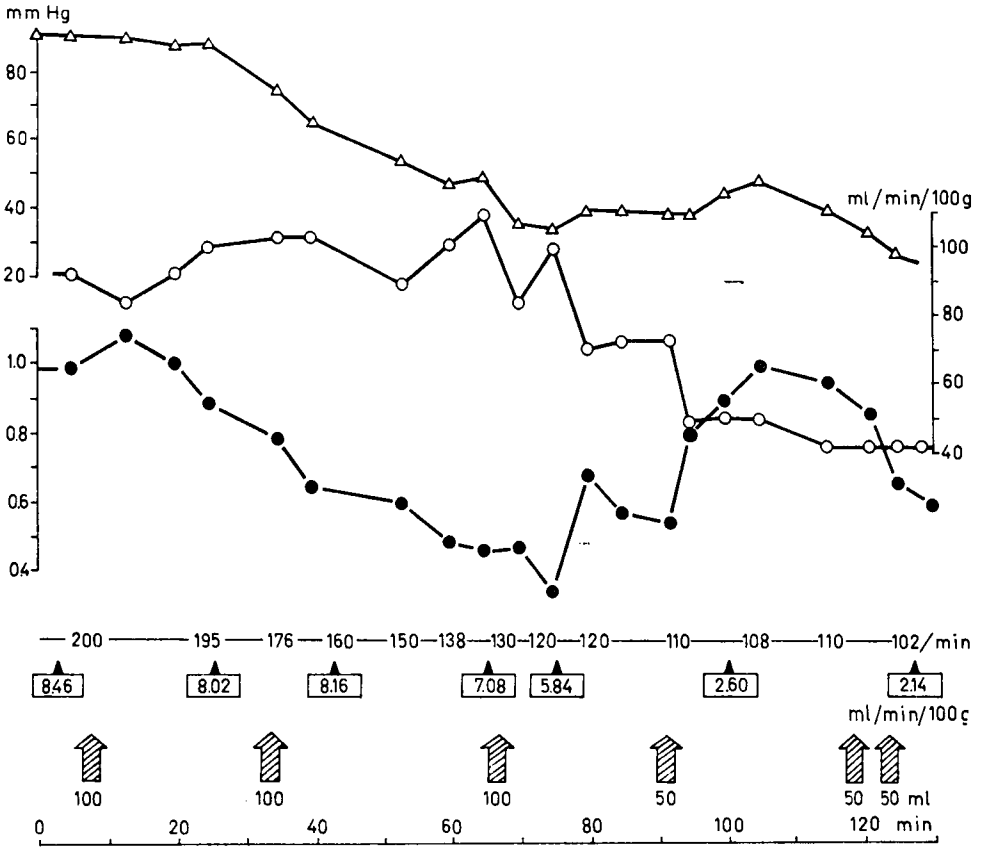

Fig. 3. Graph representing results of experiment with larger and more rapid bleed-out. Explanations the same as in fig. 2. Experiment No 13. Dog weighing 16 kg

mildly increased and then decreased. Oxygen consumption by the heart fell down considerably. In those experiments in which the blood pressure dropped rapidly to very low values, reinfusion of all withdrawn blood did not improve the coronary circulation causing anly a transient and short-lasting rise of blood pressure and coronary flow followed by their fall. Just prior to cardiac standstill and death the mean coronary resistance rose sharply, paralelling the drop in coronary blood flow. Sometimes ventricular fibrillation appeared.

DISCUSSION

The results of present investigations indicate that in the anaestetized dogs the blood pressure decreased continuously in the course of hemorrhage. The coronary blood flow generally decreased too. The rate of decline of the blood pressure was much greater than that of the coronary blood flow. Therefore the mean coronary resistance fell down. Such a situation may be due either to a decrease in the extravascular impedance to flow or to an active relaxation of smooth muscles of coronary vessels. It seems likely that both components superimpose each other: extravascular compression decreases and simultaneously coronary vessels dilate. The results of present work show that coronary vasodilatation occurs. It is of interest that during the period of hemorrhage the end-diastolic flow rate exceeds that of systolic one, which can be seen in the photohemotachogram. The fall down of arterial blood pressure is a result of the decrease of stroke volume and cardiac output. But the depression of circulation in long-lasting lowered blood pressure and in posthemorhagic shock cannot be attributed solely to a decrease of circulating volume of blood and small venous return. Present experiments show that despite the reinfusion of all withdrawn blood to animals being in postoligemic shock does not improve the ciruclation. The problem is still open. Impairment of myocardial function has been suggested as a contributing factor to the irreversibility of postoligemic shock by many workers. Lefer *et al.* (20) demonstrated that papilary muscles isolated from cats in postaligemic shock exhibited depressed excitability and contractility. This loss of myocardial contractility is a consequent upon on insufficient coronary flow.

C r o w e l l *et al.* (7) have shown some irreversible damage to a metabolic system in myocardium and presume that irreversibility in hemorrhagic shock is related to loss of purine base for restoration of cellular ATP. Macro and microscopic evidence of myocardial injury appears in hemorrhagic shock (13).

The decrease in the stroke volume and cardiac output together with

the reduct arterial oxygen content results in a reduction of oxygen transport to the tissue. In present experiments the decrease of oxygen uptake by the heart was observed. The coronary arterio-venous 0_2 difference first remained the same after the beginning of bleeding and then decreased due to the fall in the arterial 0_2 content generally was unchanged and thus there was some reserve of oxygen in the venous blood. This suggests the loss of ability to extract oxygen from the blood by the heart. As the bleeding continued the coronary blood flow fell to a greater extent than did the blood pressure.

Present investigations confirm previous findings of authors that of all arterial beds only the coronary shows a decreased vascular resistance during hemorrhagic hypotension and shock (8). Futhermore, the present work demonstrated that despite the gradual decrease of blood pressure, parallel to bleeding, the coronary blood flow can increase above the control value. Thus the coronary flow fraction of cardiac output increases. Such observations substantiate the view that the coronary circulation is preferentially supported following hemorrhage. Available evidence indicates that this may occur at the expense of the visceral organs and limbs (9, 11, 12, 15, 24, 26, 28). The whole picture in hemodynamics and the data of other investigations indicate that one of the major compensatory mechanisms of the organism in hemorrhage is the increased activity of the sympathetic nervous system (1, 2, 3, 11, 19, 21, 28, 31) with catechol amines discharge into the blood stream (16, 17).

CONCLUSIONS

1. In the course of hemorrhage the blood pressure gradually decreases.

2. Coronary blood flow increases at the beginning of bleeding and then decreases but to a smaller extent than does the blood pressure.

3. Coronary resistance declines gradually. In experiments with larger bleed-out volumes it rises sharply just prior to cardiac standstill and death. Sometimes ventricular fibrillation appears.

4. The arterial oxygen content decreases gradually and that of coronary sinus blood does not change. The oxygen uptake by the heart falls down.

5. On the basis of the obtained data one can suggest that the contractility of myocardium decreases and coronary vessels dilate. It seems likely that the heart loses the ability to extract oxygen from the sinus blood.

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STRESZCZENIE

W doświadczeniach na psach z otwartą klatką piersiową, uśpionych chloralozą i heparynizowanych, przeprowadzono skrwawiania w dwojaki sposób: powoli i szybko. Całkowita objętość upuszczonej krwi wynosiła 15—30 ml/kg. Mierzono przepływ krwi w gałązce zstępującej lewej tętnicy wieńcowej fotohemotachometrem Cybulskiego-Klisieckiego i zużycie tlenu przez serce na podstawie zawartości tlenu w krwi pobranej z t. wieńcowej i z zatoki wieńcowej. Wyniki doświadczeń wykazały, że ciśnienie tętnicze opadało stopniowo. Przepływ wieńcowy z początku wzrastał, a następnie stopniowo malał, ale w porównaniu z ciśnieniem spadek ten był mniejszy. W konsekwencji opór naczyń wieńcowych stopniowo malał. W grupie zwierząt, w której objętość upuszczonej krwi była większa, przepływ wieńcowy z początku spadał na krótko, a następnie zwiększał się ponad poziom kontrolny. Pod koniec obserwacji przepływ wieńcowy malał. Częstość skurczów serca nieznacznie malała. Zużycie tlenu przez serce spadało.

Wyniki doświadczeń sugerują, że kurczliwość mięśnia sercowego zmniejsza się, a naczynia wieńcowe rozszerzają się w czasie postępującego skrwawiania. W ten sposób wzrasta procentowo część krwi z objętości minutowej serca, przypadająca na krążenie wieńcowe.

РЕЗЮМЕ

В опытах с открытой грудной клеткой, проведенных на собаках, наркотизированных хлоралозой, кровопускание проводилось двояким способом: медленно и быстро. Общий объем пущенной крови составлял 15—30 мл/кг. Измеряли кровоток в левой коронарной артерии и потребление кислорода сердцем. Результаты опытов показали, что артериальное давление снижалось постепенно; коронарный кровоток сначала увеличивался, а потом постепенно уменьшался, но по сравнению с артериальным давлением менее резко. Вследствие этого сопротивление коронарных сосудов постепенно уменьшалось. В группе животных, потерявших значительное количество крови, коронарный поток сначала на короткое время уменьшался, а потом увеличивался и превышал контрольный уровень. К концу наблюдений коронарный кровоток уменьшался. Частота сокращения сердца незначительно падала. Потребление кислорода сердцем уменьшалось.

Результаты экспериментов показывают, что сила сердечных сокращений уменьшается, а коронарные сосуды во время прогрессирующей кровопотери расширяются. Таким образом происходит процентное увеличение части крови из минутного объема сердца, протекающая через коронарные сосуды.