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Haemodynamics in the First Seconds After Coronary Artery Occlusion

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Summary

The changes in cardiac and in total haemodynamics, occurring during the first seconds of occlusion and the subsequent desocclusion of coronary arteries were studied on 28 dogs. The most intensive changes were observed after the trunk occlusion of the left coronary artery. Simultaneously with decreasing blood inflow into the myocardium its contractility and the systolic pressure in the left ventricle and the outflow from the coronary sinus began to fall rapidly . The systolic pressure in the left ventricle decreased within the first 10 s from 24 to 13–15 kPa (180 to 100–110 mm Hg), which means that the systolic pressure fell about 1 kPa (7–8 mm Hg) per second, or 0.5–0.6 kPa (4–5 mm Hg) per systole. At the same time the enddiastolic pressure in the left ventricle also increased from zero to 3–4 kPa (25–30 mm Hg). After the trunk desocclusion of the left coronary artery the systolic pressure in the left ventricle proceeded to fall by about 2–3 kPa (15–22 mm Hg). Only then, 20–25 s after the desocclusion, blood flow in the left coronary artery began to rise intensively and 4–6 s later the myocardial contractility and the systolic pressure in the left ventricle also increased. After unclamping (50–60 s), there was an overshoot of haemodynamic values above preocclusive values and then followed the compensatory phase. This phase lasted 80–90 s and on its peak the pressure and flow parameters increased by about 50–60 % above preocclusive values. During the occlusion of *ramus interventricularis anterior* or *ramus circumflexus* for 30–60 s the haemodynamic parameters changed only slightly. The same was observed during trunk occlusion of the right coronary artery (30–60 s), but in that case many extrasystoles occurred.

Key words

Occlusion of coronary arteries – Haemodynamics after coronary occlusion – Haemodynamics after desocclusion – Coronary blood flow – Myocardial contractility

Introduction

In spite of the number of clinical and experimental studies about ischaemic heart disease (Tennant and Wiggers 1935, Gregg et al. 1939, Rein 1954, Driscol and Eckstein 1967, Harley *et al.* 1968, Schaper 1979, Zimmer *et al.* 1989, Schoemaker *et al.* 1990), the changes in haemodynamics occurring during first seconds after occlusion of coronary arteries have not been explored sufficiently until now.

From the point of view of heart diseases in man, this initial stage of acute myocardial ischaemia can be considered as the most critical. Furthermore, physicians can observe this stage very rarely because they only see the patients later. We have therefore tried to analyze this initial stage in experiments on anaesthetized animals. Either the trunks of coronary arteries or both branches of the left coronary artery (*ramus interventricularis anterior* and *ramus circumflexus*) were occluded.

Following questions were studied in these experiments:

1. Which changes occur in the myocardial blood flow, in heart contractility, and in ventricular and left atrial pressures during occlusion of coronary arteries ?
2. What is the course of the main haemodynamic parameters after the desocclusion of coronary arteries ?

Material and Methods

The experiments were carried out on 28 mongrel dogs – (16 males and 12 females) with the average weight 19.8±5.6 kg under thiopental anaesthesia (40–50 mg/kg) and artificial ventilation (Chirolog). The chest was opened by the middle sternotomy. The pressures in both ventricles, in the left atrium and in the femoral artery were measured and recorded by electromanometers (Statham). The blood flow in coronary arteries and the blood outflow from the coronary sinus were recorded by electromagnetic flowmeters (Nykotron). Beside that, the myocardial contractility of the left ventricle (dp/dt) and the electrocardiogram were monitored. All haemodynamic parameters were continuously registered on a 8-channel Physioscript (Schwarzer).

The coronary arteries were occluded by means of clamps for 15–60 s. The fall of systolic pressure in the left ventricle to 7–8 kPa (55–60 mm Hg) was mostly taken as the criterion for releasing the occlusion. This criterion was chosen, because the fall of blood pressure in the left ventricle continued further, even after the release of the occlusion. In such cases, the myocardial blood flow was not restored spontaneously after the desocclusion, the heart contractility did not rise, and the animal died. The recordings were expressed as the mean values ± S.E.M. Altogether, we performed 24 occlusions (lasting 15–20 s) of the trunk of the left coronary artery, 21 occlusions (30–60 s) of *ramus circumflexus*, 16 occlusions of *ramus interventricularis anterior* and 18 occlusions (30–60 s) of the right coronary artery.

Simultaneous records of eight haemodynamic parameters enabled more exact analysis of the haemodynamic responses to occlusion of arteries supplying the myocardium with blood.

Results

The effect of occlusion of the left coronary artery is shown in Fig. 1. Although the occlusion of the trunk lasted only 18 s, all recorded haemodynamic parameters were already changed markedly from the very beginning.

Obviously, the blood flow through the left coronary artery began to drop concomitantly with the occlusion. Within one or two seconds after the occlusion, the blood outflow from the myocardial bed through the coronary sinus dropped by 60–70 % as well. At the same time the contractility of the left ventricle steeply declined. Simultaneously, the systolic pressure in the left ventricle fell to 8–9 kPa (60–70 mm Hg) and in the femoral artery to 7–8 kPa (55–60 mm Hg). Later (5–6 s) blood pressure decreased in the left atrium by 10–15 mm H_2O and after 3–4 s it increased in the right ventricle by 1.5–2.0 kPa (10–15 mm Hg).

When the drop of the systolic pressure in the left ventricle reached 8 kPa (60 mm Hg) (within 18 s), the occlusion of the left coronary artery was released. In spite of the desocclusion, the blood flow in the left coronary artery slightly

increased, but the systolic pressure in the left ventricle proceeded to fall to 5 kPa (40 mm Hg). Only 25 s after the occlusion had been released, the spontaneous and rapid increase of myocardial blood flow occurred. Few seconds later (3–4 s) the myocardial contractility and the blood pressure began to rise in the left ventricle until the preocclusive values were reached and then surpassed. During the following compensatory phase, all the monitored parameters increased, but the restitution to initial values did not occur until 150–180 s after occlusion had been discontinued. The compensatory phase lasted 80–90 s and, on its peak, the pressure and flow values surpassed the preocclusive ones by about 50–60 %. The original record in Fig. 1 shows that, during the occlusion, the enddiastolic pressure in the left ventricle increased from zero values to 4–5 kPa (30–35 mm Hg). The emptying of blood from the left ventricle is much worse during this period because of the low contractility of the myocardium.

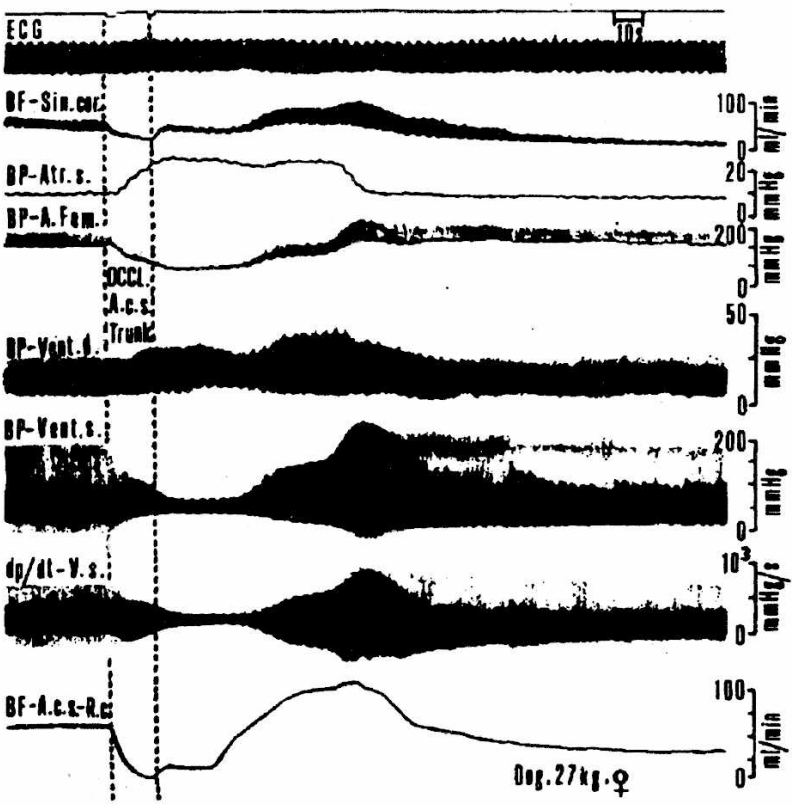

Fig. 1

Haemodynamic changes during the occlusion (lasting 18 s) and the desocclusion of the trunk of the left coronary artery (OCCL. A.c.s. Trunk) in the anaesthetized dog. Traces from above: ECG – electrocardiogram, BF-Sin.cor. – blood flow in the coronary sinus, BP-Atr.s. – blood pressure in the left atrium, BP-A.Fem. – blood pressure in the femoral artery, BP-Vent.d. – blood pressure in the right ventricle, BP-Vent.s. – blood pressure in the left ventricle, dp/dt-V.s. – contractility of the left ventricle, BF-A.c.s.-R.c. – blood flow in the left coronary artery *(ramus circumflexus)*. The dark middle and clearer margins in the records of blood pressures and dp/dt are caused by the non-equality of the systolic-diastolic amplitude. The dark central parts thus represent twice more oscillations than the margins of the records. This phenomenon deserves a special analysis.

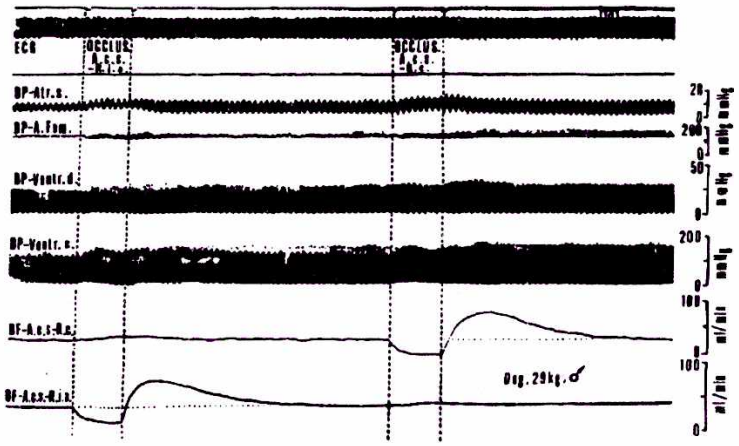

Fig. 2

Haemodynamic changes during the occlusion (lasting 30 s) of branches of the left coronary artery in the anaesthetized dog. OCCLUS. A.c.s.-R.i.a. – the occlusion of *ramus interventricularis anterior* of the left coronary artery, OCCLUS. A.c.s.-R.c. – the occlusion of *ramus circumflexus* of the left coronary artery. Traces from above: ECG – electrocardiogram, BP-Atr.s. – blood pressure in the left atrium, BP-A.Fem. – blood pressure in the femoral artery, BP-Ventr.d. – blood pressure in the right ventricle, BP-Ventr.s. – blood pressure in left ventricle, BF-A.c.s.-R.c. – blood flow in *ramus circumflexus* of the left coronary artery, BF-A.c.s.-R.i.a. – blood flow in *ramus interventricularis anterior* of the left coronary artery.

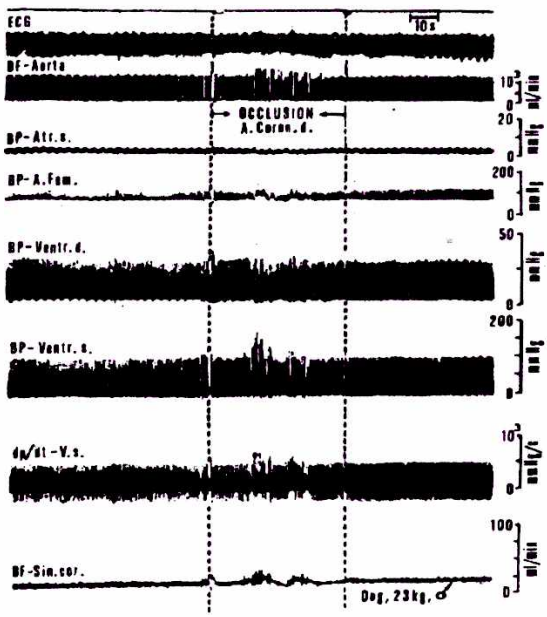

Fig. 3

Haemodynamic changes during the occlusion (lasting 45 s) of the right coronary artery (OCCLUSION A. Coron. d.) in the anaesthetized dog. Traces from above: ECG – electrocardiogram, BF-Aorta – blood flow in the ascendent aorta, BP-Atr.s. – blood pressure in the left atrium, BP-A.Fem. – blood pressure in the femoral artery, BP-Ventr.d. – blood pressure in right ventricle, BP-Ventr.s. – blood pressure in left ventricle., dp/dt-V.s. – contractility of the left ventricle, BF-Sin.cor. – blood flow in the coronary sinus.

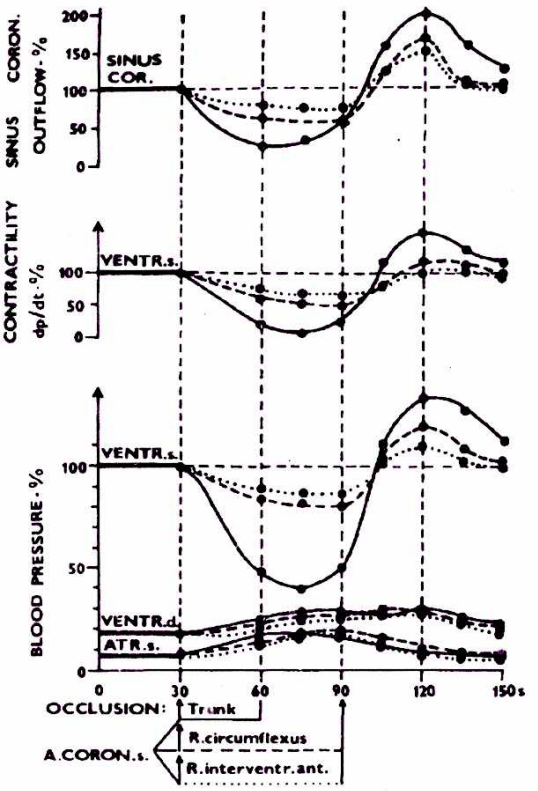

Fig. 4

Average changes in blood pressure in both heart ventricles (VENTR.s., VENTR.d.) and in the left atrium (ATR.s.), in the contractility of the left ventricle and in blood outflow from the coronary sinus after the occlusion and the desocclusion of the trunk of the left coronary artery and of its both branches in anaesthetized dogs. Number of occlusions (number of dogs): Trunk of the left coronary artery – 24 (21), *ramus circumflexus* – 21 (12), *ramus interventricularis anterior* – 16 (9). Standard deviations from the mean values (in percent): coronary sinus outflow ± 12–18 %, contractility of the left ventricle ± 9–17 %, blood pressure in both ventricles and in the left atrium ± 16–23 %.

The effect of 20 s lasting occlusion of the main branches of the left coronary artery is shown in Fig. 2. The occlusion of *ramus interventricularis anterior* or *ramus circumflexus* did not evoke more marked changes in haemodynamic parameters within 20 s. Since the blood flow in both branches of the left coronary artery was recorded, the respective curves show that the occlusion of one branch decreased its blood flow to zero values and simultaneously slightly increased the blood flow in the other branch. After release of the occlusion, a clear reactive hyperaemia lasting 30–35 s occurred in both cases. Since occlusions lasting 20 s evoked only slight changes, we also applied lasting occlusions in our experiments (up to 60 s). In that case, more distinct haemodynamic changes were registered (Fig. 4).

Fig. 3 presents the results of 45 s lasting occlusion of the trunk of the right coronary artery. Even in this prolonged occlusion greater changes in the recorded parameters could not be distinguished. In most cases, disturbances in the impulsogenic activity of the sinoatrial node took place during occlusion of the right coronary artery. It is possible to see these disturbances as numerous extrasystoles with compensatory pauses.

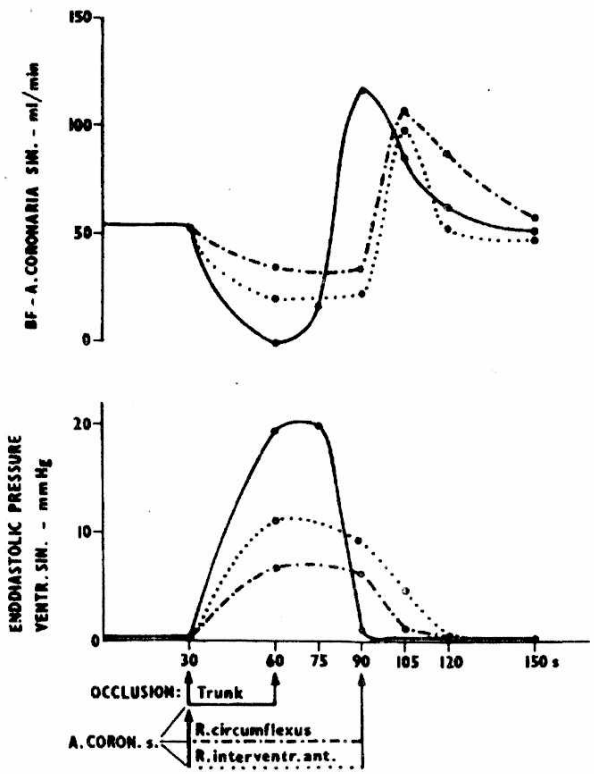

Fig. 5

Average changes of the enddiastolic pressure in the left ventricle and of the blood flow in the left coronary artery after the occlusion and desocclusion of the trunk and both branches of the left coronary artery in anaesthetized dogs. The numbers of occlusions and of dogs are the same as in Fig. 4. Standard deviations from the mean values: left coronary blood flow ± 12–19 %, enddiastolic pressure in the left ventricle ± 14–22 %.

The mean values of haemodynamic changes induced by trunk occlusion of the left coronary artery and by separate occlusions of its branches are illustrated in Fig. 4. We have evaluated pressures in both ventricles and in the left atrium, the myocardial contractility of the left ventricle, and the venous blood flow in the coronary sinus. Our experiments confirmed the expectations that the fastest and greatest changes in haemodynamic parameters were caused by trunk occlusion of the left coronary artery. Non-significant changes were found when *ramus circumflexus* and especially when *ramus interventricularis anterior* were clamped. Deviations from the average magnitude of changes after arterial occlusion depend mainly on the morphological variability and on the contribution of single arteries to the myocardial blood supply of the left ventricle.

Fig. 5 shows the relation between the enddiastolic blood pressure in the left ventricle and blood flow in the left coronary artery. This evaluation confirmed that the contractility of the myocardium decreased proportionally to the lower blood supply and the left ventricle emptied less effectively. The enddiastolic pressure in the left ventricle therefore rose from zero values up to 4–5 kPa (30–35 mm Hg).

Discussion

Our experiments confirmed the well-known fact that marked changes occur not only in local haemodynamics of the myocardium, but also in the systemic circulation after occlusion of coronary arteries (Gregg 1950, Wüsten 1979, Dzhavakhishvili *et al.* 1982, Olsson and Bugni 1986). The speed and the intensity of these changes depend on the extent of the vascular bed of individual coronary arteries, on the efficiency of collateral anastomoses, on the myocardial activity and on the importance of different regions of the heart for the total blood circulation. Since the greatest part of the myocardium is located in the walls of the left ventricle and its blood supply comes mainly from the left coronary artery, the most rapid and greatest haemodynamic changes occur after the occlusion of the left coronary artery. After the occlusion of one of the two main branches of the left coronary artery (*ramus circumflexus* or *ramus interventricularis anterior*), considerably smaller changes occur in cardiac haemodynamics. Slight changes in local and especially in general circulation can also be observed after occlusion of the right coronary artery. However, clear interindividual differences were found because there exists considerable interindividual variability in the area supplied by one coronary artery (Wiggers 1954, Schaper *et al.* 1976, Vanhuis *et al.* 1985, Antal 1986). It should also be mentioned that, after the occlusion of main branches of the left coronary artery and also after the occlusion of the right coronary artery lasting for a longer time (several minutes), distinct changes in haemodynamics can be registered. As it has already been pointed out, the greatest changes in the local and the total haemodynamics appear during first seconds after the occlusion of the left coronary artery and then after the release of occlusion. Therefore, our discussion deals mainly with comments on these two phases.

The time course of haemodynamic deterioration during the first seconds after the occlusion of the left coronary artery

The immediate consequence of the occlusion is the decreased blood flow in the corresponding artery and in its vascular bed. Then, 0.5–1.0 s later, the myocardial contractility begins to fall and this decreases the systolic pressure in the left ventricle. Naturally, the drop of blood pressure in the femoral and other arteries as well as the fall of the outflow from the coronary sinus follows.

The rapid fall of blood pressure in the left ventricle has an immediate consequence in other haemodynamic aspects. The registered curves demonstrate that the most rapid drop of pressure takes place within the first 10 s after the occlusion. The time course of the pressure drop proved that, during the first 10 s, the pressure falls almost linearly and the magnitude of the decrease is about 9–11 kPa (67–80 mm Hg). This drop of pressure represents 0.5–0.6 kPa (4–5 mm Hg) per one systole or about 1 kPa (7–8 mm Hg) per second. After 18 s lasting trunk occlusion of the left coronary artery, the systolic pressure decreases by about 110 mm Hg, i.e. from 180 to 70 mm Hg.

Simultaneously, during first 10 s the enddiastolic pressure in the left ventricle increases by about 2–3 kPa (15–23 mm Hg) what represents about 0.1 kPa (0.7–0.9 mm Hg) per systole. This calculation also demonstrates the actual speed of the evolution of myocardial ischaemia and the rate of the decrease in the contractile

force of myocardial cells. This large decrease of heart contractility shows that the muscle cells do not accumulate large supplies of energy metabolism and anabolites and cannot work on a longer oxygen and metabolic debt. On the other hand, this rapid decrease of contractility seems to indicate that the heart muscle requires for its activity not only adequate, but also time-punctual nutrition in order to be able to work at its full contractile performance.

The course of haemodynamic changes after the release of left coronary artery occlusion

The first effect after release of the occlusion concerns the blood flow in the desoccluded vessel. This immediate rise of blood flow is clearly shown in Fig. 1. However, this increase in the first seconds is very small, because the blood pressure in the left ventricle is also low at this time. Therefore, in spite of the desocclusion of the left coronary artery, the contractility of the myocardium and the left ventricular pressure proceeds to fall further. The maximum decrease of both mentioned parameters will take place only 20 s after the desocclusion. Afterwards, the blood flow in the desoccluded coronary artery suddenly begins to rise more steeply and 2–3 s later the myocardial contractility begins to rise more rapidly as well as the blood pressure in the left ventricle. According to the metabolic hypothesis of coronary blood flow regulation, myocytes produce vasodilatory metabolites in proportion to their ischaemia and thus, in effect, regulate their own blood supply (Olsson and Bugni 1986, Hoffmeister *et al.* 1988). We can suppose that 20 s after the occlusion, the concentration of vasodilatory substances in the myocardium is already so high, that it evokes sudden and large vasodilation and a steep increase of the local coronary blood flow. Only 40–50 s after releasing the occlusion, the initial values of the registered parameters were attained and followed by the compensatory phase lasting 50–60 s. The values of haemodynamic parameters on the peak of the compensatory phase were about 30–50 % higher, as compared with the preocclusive values. Altogether it took 105–120 s until the preocclusive monitored parameters were restored after the desocclusion. Naturally, during the longer occlusion, the phase of resuscitation was also prolonged, or this phase did not occur at all and the animal died.

It is remarkable, that the myocardial contractility and the left ventricular pressure continued to fall down to critical values of about 5.2 kPa (40 mm Hg) and lower even after the arterial occlusion had been released. With such low values of left ventricular and aortic pressure, the contractility of the heart muscle could not recover spontaneously. Then, in most cases, the resuscitation of myocardial blood flow and the restoration of total circulation could only be achieved by artificial massage of the heart. We have to emphasize that, in all cases in which the resuscitation of myocardial activity took place, myocardial blood flow was the first to rise and later myocardial contractility and blood pressure in the left ventricle began to increase gradually.

The most important findings can be summarized as follows: 1. The unexpected speed of the decrease of the haemodynamics during the first seconds after the occlusion and a further drop of haemodynamic parameters, even after the desocclusion of the left coronary artery trunk; 2. Besides this, the most important factor for the resuscitation of myocardial contractility is the spontaneous increase of blood flow about 20 s after the desocclusion of the left coronary artery.

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Reprint requests

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