THE ROLE OF STRETCH RECEPTORS OF THE LEFT VENTRICLE IN REFLEX HEMODYNAMIC CHANGES DUE TO MYOCARDIAL ISCHEMIA

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It has been shown previously that ischemia of the feline myocardium caused by compression of the coronary artery induces reflex bradycardia, reduction of arterial pressure, minute volume and cardiac output, and a fall in peripheral vascular tone [1-2].

From our own and published findings we consider that the reflexes originate from the left ventricle. We still do not know from precisely what receptors of this region the centripetal impulses originate. A reflex of similar nature develops when the intraventricular pressure is increased [3, 5-8]. However when the myocardium is ischemia the baroceptors cannot take part in the reflex which we have discovered, because such a condition causes not an increase but a decrease of arterial pressure in the ventricle. The source of the reflex might be stretch receptors, because ischemia of the myocardium is associated with a distension, even when the intraventricular pressure is reduced [9]. The existence of such receptors has been established for the vascular zone [4]. Also, a reaction similar to the one we have observed may be evoked by stimulation of chemoreceptors of the ischemic myocardium by metabolic breakdown products (as in the Betsold-Yarish reflex).

In the present investigation we have attempted to find which of the two types of receptors plays the chief part in the reflex from the ischemic myocardium. Our starting point has been that elimination of the chemoreceptors would indicate that stretch receptors initiated the reflex. In this way their presence in the ventricular myocardium would also be demonstrated. To carry out this task we have to follow the changes in peripheral vascular tone and pulse rate in response to ischemia alone, or to tension in the left ventricle.

EXPERIMENTAL METHOD

We used the following arrangement (Fig. 1). Blood from the left ventricle was taken trhough a cannula introduced into the ascending aorta, and was led into a cylinder. To the tube we applied a screw clamp to regulate the resistance of the outflow,and thus the pressure in the left ventricle and coronary arteries. This pressure and the frequency of the cardiac contractions were recorded by a mercury manometer (not shown in Fig. 1); it was connected to the tube between the aortic cannula and the clamp. From the cylinder blood was supplied at a constant rate by means of a perfusion pump through the abdominal aorta. The tone of the peripheral vessels was recorded by a second mercury manometer connected to the output of the pump. The increase of tone was associated with an increased pressure, and vice versa (method of resistography). The distal end of the ascending aorta was ligated. In some of the experiments the ligature was placed on the aortic arch behind the left subclavian artery. The flow from the left ventricle was then directed into a cylinder through a cannula introduced into this artery. The head was perfused by the same pump through the peripheral end of the innominate trunk. The resistance of the flow from the left ventricle was adjusted so that pressure in it was 70-100 mm mercury. To establish an isolated region of ischemia in the myocardium the clamp was undone; the resistance to the flow fell greatly and the pressure in the ventricle and at the entrance to the coronary artery dropped to 0-15 ml mercury. The failure of the blood supply was confirmed by the ECG. It showed a shift of the S – T interval above or below the baseline, a negative T wave, and sometimes a complete transverse block. The other type of ischemia of the myocardium was produced by temporary closure of the descending branch of the left coronary artery applied by means of a ligature. Then the resistance to the blood flow from the left ventricle was maintained at the original level. As the contractile power of the myocardium was reduced the intraventricular pressure fell by 10-44%, and the left ventricle dilated.

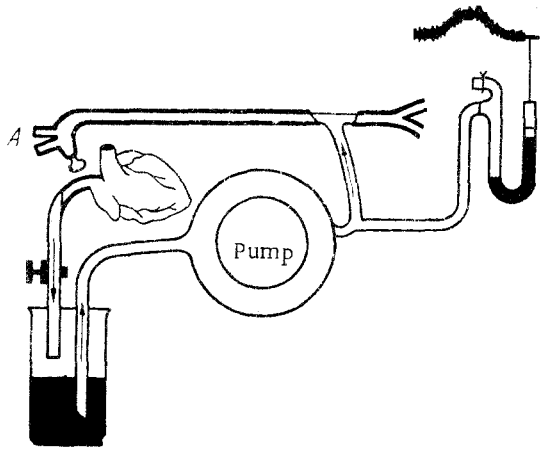

Fig. 1. Diagram of the experiment to study peripheral vascular tone during different types of myocardial ischemia. Explanation in text.

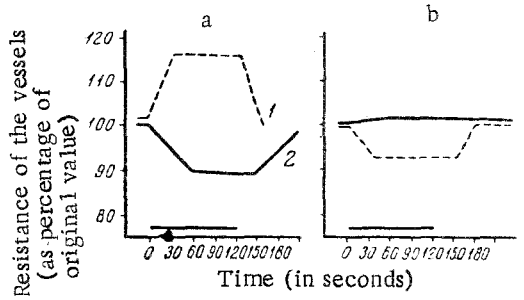

Fig. 2. Change of tone in peripheral vessels in (1) ischemia without distension and (2) ischemia with distension of the myocardium in animals (a) with intact and (b) with divided vagi. Duration of compression of coronary arteries shown by a horizontal strip along the abscissa.

The experiments were carried out on 16 cats under thiopentane or urethane anesthesia. The amounts were 0.1 and 1 g/kg respectively, and were injected intraperitoneally. After the animal had been prepared and the blood condition stabilized the temporary reduction of pressure in the left ventricle was produced (ischemia without distension), and then after recovery to the original condition the coronary artery was compressed. After 1-3 repetitions of these manipulations the vagi were divided in the neck, and the same experiments were repeated.

EXPERIMENTAL RESULTS

The changes of perfusion pressure and of pulse rate are given in Table 1 and Fig. 2 as a percentage of the original values.

From Table 1 it can be seen that when the coronary arteries were compressed, i.e., in myocardial ischemia, the associated distension of the ventricle, the perfusion pressure, and consequently the resistance and tone of the vessels of the greater circulation were reduced on an average by 11.5%. At the same time the heart rate fell on an average by 15%, and the arterial pressure by 20%. All these changes are statistically significant. In ischemia without distension the effect was different. Then peripheral tone did not fall, but increased by 16%. None of the changes in pulse rate which occur in ischemia with distension were observed.

Vagotomy eliminates or alters the effect of both types of ischemia.

From these results and from published reports we may draw up a scheme for the relationship between the four indices, namely: ischemia of the myocardium, pressure in the left ventricle, degree of distension and reflex bradycardia, and vasodilatation, for different experimental circumstances (Table 2).

It is readily seen that there is a positive correlation between distension of the left ventricle on the one hand and reflex reduction of peripheral tone and bradycardia on the other. There was no correlation between this latter reflex and myocardial ischemia or change of pressure in the ventricle.

TABLE 1. Changes of Hemodynamic Indices in Response to Various Kinds of Ischemia of the Myocardium in Animals with Intact or Divided Vagi

Experimental conditions	Index	Ischemia	
		Without distension	With distension
Intact vagi	Resistance of vessels (in mm mercury per ml per minute)	+16 (P<0.05)	−11.5 (P<0.05)
	Pressure in coronary arteries (in mm mercury)	−83 (P<0.05)	−20 (P<0.05)
	Pulse frequency	−	−15 (P<0.05)
Vagotomized	Resistance of vessels (in mm mercury per ml per minute)	−8 (P<0.05)	+3 (P<0.05)
	Pressure in the coronary arteries (in mm mercury)	−82 (P<0.05)	−5 (P<0.05)
	Pulse rate	−	−

TABLE 2. Relationship Between the Different Hemodynamic Indices under Different Experimental Conditions

Conditions of experiment	Ischemia of the myocardium	Pressure in the left ventricle	Distension of the left ventricle	Vasodilatation and bradycardia
Compression of the coronary artery	+	–	+	+
Reduction of pressure in the coronary vessels	+	–	–	–
Distension of the left ventricle	–	+	+	+
End of distension of left ventricle	–	–	–	–

Note. A cross indicates the development or a positive change of an index; the minus sign indicates a disappearance or reduction of the quantity.

Therefore the origin of the bradycardia and hypotension caused by compression of the left coronary artery and by other actions is to be found in the stretch receptors of the wall of the left ventricle. These results also demonstrate the presence of receptors in the myocardium.

We must, it seems, agree with those authors who maintain that all the baroceptors, and not only those of the blood vessels, are, strictly speaking, stretch receptors. In this case the reflex due to increase of intraventricular pressure and that due to compression of the coronary artery must be interpreted as essentially the same reflex, although they arise under different circumstances.

SUMMARY

Experiments were carried out on cats by means of a special pump for arterial perfusion at a constant rate. Myocardial ischemia and dilatation of the left ventricle was induced by compression of the base of the descending branch of the left coronary artery. Ischemia without distension was produced by reduction of pressure at the mouth of the coronary vessels to 0-15 mm mercury.

In the first case vascular tone in the systemic circulation was reduced and the heart beat slowed; in the second case peripheral tone was increased and there was no change in heart rate. Therefore the hypotension and bradycardia observed in occlusion of the coronary artery must have been due to stimulation of myocardial stretch receptors, and not to ischemia.

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