stomachs were removed, opened along the greater curvature and rinsed in ice-cold saline. The mucosa of the oxyntic gland area was scraped off and homogenized in ice cold 0.1 M phosphate buffer, pH 6.9–7.0, to a final concentration of 100 mg (wet wt)/ml.

Aliquots (0.5 ml) of the homogenate were incubated with 4×10^{-4} M 1-^{14}C-L-histidine (1.3 mCi/mmole; New England Nuclear) in the presence of 10^{-5} M pyridoxal-5-phosphate and 5×10^{-4} reduced glutathione at 37 ° C for 1 h under nitrogen. Total reaction volume was 0.53 ml. The enzyme reaction was stopped by acidification and the amount of $^{14}CO_2$ produced was determined as described in detail elsewhere [11,13]. Enzyme activities are expressed as pmoles $^{14}CO_2$ produced per mg tissue (wet wt)/h. Comparisons between groups were made using Student's t-test for unpaired observations. Differences with a p-value of less than 0.05 were considered significant.

3 groups of rats were examined. Control rats received an i.v. infusion of saline and simultaneously an intragastric perfusion with deionized water. In a 2nd group the rats received an i.v. infusion of gastrin and an intragastric perfusion with water. In this group gastric histidine decarboxylase activity was markedly increased compared with the control rats. A 3rd group of rats received the same dose of gastrin in the gastric perfusate whereas the i.v. infusion was saline. In this group the enzyme activity did not differ from that seen in the control group. The data are summarized in the figure.

Data are accumulating that upon stimulation of the gastrin cells gastrin is released into the lumen of the stomach as well as into the circulation[2-7]. Whether this phenomenon is physiologically significant or not remains to be established. There have been suggestions that luminal gastrin may exert a biological action from within the lumen of the gut[5,9]. In the rat, activation of histidine decarboxylase in the oxyntic mucosa is one of the functions of gastrin[11]. As expected, i.v. infusion of a large dose of gastrin raised the histidine decarboxylase activity. Intragastric administration of gastrin, however, failed to reproduce this effect. This could mean either that the endocrine cells containing the enzyme do not respond to gastrin from the lumen or that they are inaccessible to gastrin from the lumen under the conditions of the experiment. However, it should be realized that the failure of luminal gastrin to reproduce the effect of circulating gastrin on histidine decarboxylase does not necessarily mean that other target cells are inaccessible or non-responsive to luminal gastrin.

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Effect of sudden reductions of the arterial blood pressure on the mean diastolic coronary resistance

D. Gattullo, G. Losano, O. Pinotti and G. Vacca[1]

Department of Human Physiology, University of Turin, Corso Raffaello 30, I-10126 Torino (Italy), 4 August 1980

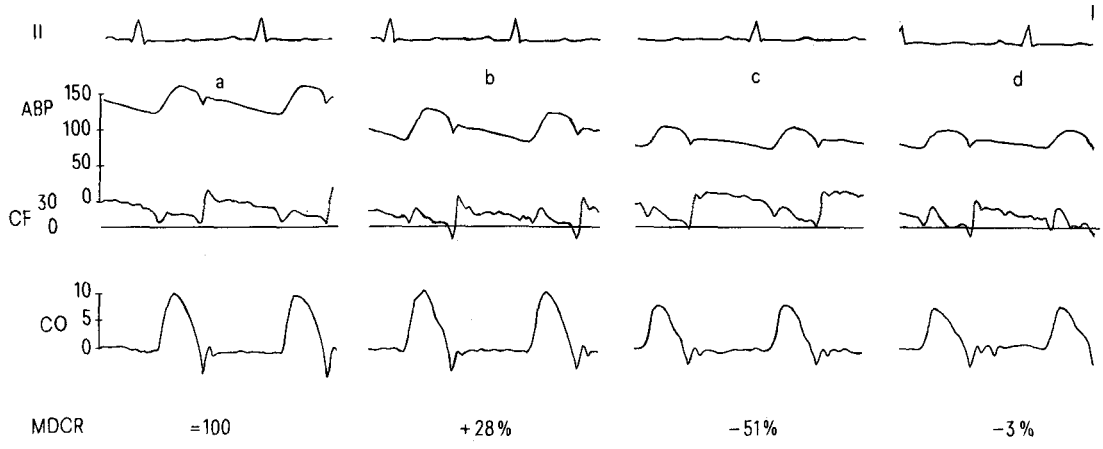
Summary. At the beginning of a 10 sec arterial haemorrhage, vascular elasticity induces an increase of mean diastolic coronary resistance. Then, the increase is counteracted by the relaxation of the vascular musculature, which causes a coronary hyperaemia when, after the haemorrhage is arrested, the vascular wall is stretched by a sudden though slight increase of blood pressure.

The value of the mean diastolic coronary resistance (MDCR) is a reliable index of the coronary vascular resistance (CVR). It can be calculated from the ratio of the mean diastolic aortic blood pressure to the diastolic coronary flow.

In addition to metabolic, humoral and nervous factors, both vascular elastic distensibility and a myogenic mechanism take part in the regulation of CVR[2-8]. Since the elastic and the myogenic mechanisms are affected by the transmural coronary pressure, this research was planned to investigate how they interact upon the regulation of MDCR during and after abrupt reductions of the aortic blood pressure (ABP) in the absence of corresponding changes of the baroreceptor activity. In a previous study it was seen that abrupt but transient falls of ABP can be effectively produced by inducing and then stopping a brief multiple arterial haemorrhage[9].

Materials and methods. In 8 open-chest dogs under general barbiturate anaesthesia and artificial ventilation, the probes of 2 electromagnetic flowmeters were placed around the ascending aorta and the left circumflex coronary artery in order to record stroke volume (SV) and coronary flow (CF) respectively. ABP was derived by means of a plastic catheter connected with an electromanometer and pushed into the aorta via the left common carotid artery.

To produce a multiple arterial haemorrhage, 3 silicon tubes filled with heparinized saline solution and clamped on 1 side, were inserted into the right common carotid and the 2 femoral arteries.



A, Control; B, 5 sec after the beginning of the haemorrhage; C, 2 sec after the arrest of the haemorrhage; D, 20 sec after the arrest of the haemorrhage. Note in C a well evident coronary hyperaemia. II, ECG from the 2nd limb lead; ABP, aortic blood pressure in mmHg; CF, coronary flow in ml/min; CO, cardiac output in l/min; MDCR, mean diastolic coronary resistance. Paper speed = 100 mm · sec^{-1}.

During the experimental procedure, interference due to the baroreceptors was prevented by both the intubation of the 2 common carotid arteries and the section of the vagi nerves.

Results and discussion. In the animals thus prepared ABP was rather high (140 ± 17 mmHg) in the control. As soon as a 10-sec haemorrhage was started, the opening of the 3 silicon tubes produced a sudden reduction of the total peripheral resistance. As a result, a sharp fall of ABP ($39 \pm 9\%$ after the first 2 sec and $47 \pm 10\%$ after the first 5 sec) occurred together with a slight increase of SVs, the latter effect being a consequence of the lowered aortic impedance to the ventribular ejection (fig. 1). Owing to the suppression of the baroreceptor interference, no change in heart rate was observed. In the last part of the haemorrhage, during the entire course of which 160–200 ml of blood were collected, ABP decreased further ($62 \pm 10\%$ with respect to the control), whereas SVs fell below the control as a consequence of the reduction of the venous return to the heart.

After the haemorrhage was arrested by clamping the tubes, SVs did not vary any more, but continued to display the same amplitude observed just before the arrest. On the contrary, immediately after the clamping of the tubes, ABP began to increase progressively, although even 2 min later it was not back to the control value.

After the first 2 sec of haemorrhage MDCR increased significantly ($p < 0.05$) in all experiments by $23 \pm 13\%$, while after the first 5 sec an average increase of $62 \pm 111\%$ was not significant ($p > 0.05$) owing to the tremendous rise ($+330\%$) recorded in one experiment and the reduction observed in another one. Later on, at the end of the haemorrhage, MDCR was below the control in 4 experiments, back to the control in one, and further increased in three. Among these 3 experiments, CF fell to zero in diastole in the dog in which MDCR was increased by 330% after the first 5 sec. With respect to the control, Tension Time Index (TTI)[10] was significantly ($p < 0.05$) reduced in all experiment by $55 \pm 12\%$.

At the clamping of the tubes, when ABP increased quickly by $32 \pm 20\%$ with respect to the instant prior to the end of the haemorrhage, MDCR fell abruptly below the control in all experiments with an average highly significant ($p < 0.01$) decrease by $40 \pm 13\%$. As a result of such a decrease a clearly evident coronary hyperaemia occurred. It is note-worthy that the fall of MDCR and the related hyperaemia occurred also in the 4 experiments in which MDCR was already below the control immediately before the clamping of the tubes. However, both the fall of MDCR and the hyperaemia were completely over about 20 sec later. Similar data were obtained when a 2nd haemorrhage was produced after reinfusion of the blood and beta-adrenergic receptor blockade.

The above results suggest that the initial effect of the transmural pressure fall was the elastic reduction of the diameter of the coronary arterial vessels. In the experiments in which MDCR decreased or returned to normal during the last part of the haemorrhage, it seems that after the first 5 sec a low-pressure induced myogenic vasodilatation counteracted the effect of the elastic recoil.

In the experiments in which MDCR increased until the end of the haemorrhage, the activity of the vasodilatory mechanism was probably masked by the prevalence of a remarkably conspicuous elastic recoil of the arterial wall.

As TTI decreased together with ABP, metabolic factors should not be involved in the relaxation of the coronary smooth muscle fibres[10]. These factors, however, must have played some role in the experiment in which no flow was present at the end of the haemorrhage.

In all experiments, the occurrence of a relaxation of the coronary vascular musculature, caused by the decrease of ABP, is believed to have been responsible for the fall of MDCR which occurred when the clamping of the tubes caused a distension of the arterial wall by a sudden though slight increase of the transmural pressure. However, the fall was over when the transmural pressure itself induced the vascular musculature to contract again about 20 sec after the arrest of the haemorrhage.

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