Physiologisches Institut I, Universität Düsseldorf

Alpha-adrenergic vasoconstriction in arterial and arteriolar sections of the canine coronary circulation*)**)

Alpha-adrenergische Konstriktion arterieller und arteriolärer Abschnitte des Koronarkreislaufs. Untersuchungen an Hunden

G. Ertl and M. Fuchs

With 5 figures and 2 tables

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Summary

Using a method which allowed the in-situ measurement of segmental coronary vascular resistances, the reaction of arterial and arteriolar sections of the coronary vascular system to α-receptor stimulation was studied in anaesthetized dogs. The left coronary artery was cannulated, and the perfusion pressure was kept constant. Allowance was made for extravascular and metabolic influences of α-stimulation on coronary vascular resistances. On an average, submaximal α-stimulation with xylometazoline increased the arterial resistance by about 60 % and the arteriolar resistance by about 90 %. The cannulation of the left coronary artery increased the sympathetic reactivity of the arterioles. Moreover, xylometazoline increased the extravascular component of the coronary vascular resistance by about 4 %. Thus it can be assumed that under normal in-vivo conditions α-receptor vasoconstriction might be less different in coronary arteries and arterioles. Since the arterial resistance ranges from 20 % to 50 % of total coronary resistance, a sympathetic vasoconstriction of this vascular section might lead even to a critical limitation of coronary blood flow. On the other hand, a predominant constriction in arterioles leads to an increase in peripheral coronary pressure, i.e. to a "reverse coronary steal phenomenon".

Sympathetic coronary vasoconstriction is well recognized and it could be demonstrated that it is mediated by the adrenergic α-receptors (5, 10, 13, 27). Some authors suppose that α-receptor induced coronary vasoconstriction is capable to compete with metabolic coronary vasodilation (3, 14, 23, 24, 28). In-vitro studies have shown that sympathetic vasoconstriction occurs predominantly in large coronary arteries (1, 4, 22, 39). There are only a few studies on in-situ heart preparations concerning the reaction of

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different coronary vascular sections to α-adrenergic stimulation (2, 17, 20). These studies were performed on large coronary arteries without a significant vascular resistance. Gerová et al. (17) and Bassenge et al. (2) found that large coronary arteries can be constricted by stellate ganglion stimulation and the application of norepinephrine. Kelley and Feigl (20) concluded from their experiments that, in contrast to investigations on in-vitro preparations, sympathetic vasoconstriction in large coronary arteries is relatively less than in the entire coronary vascular bed.

When the coronary arteries and arterioles are constricted to a different extent, a change in peripheral coronary pressure must occur (11). Since peripheral coronary pressure is the driving force for the collateral blood flow (11), α-receptor vasoconstriction could either lead to a coronary steal or to a phenomenon called “reverse coronary steal” by Chiarello et al. (8).

The quantitative evaluation of a direct sympathetic vasoconstriction in coronary vessels is difficult since pharmacological and neuronal sympathetic activations alter myocardial performance and change therefore indirectly the coronary vascular resistance. Another source of problems concerning the quantification of sympathetic vasoconstriction results from the dependence of sympathetic reactivity on the present sympathetic tonus.

It was the aim of the present study to evidence and to quantify the α-adrenergic constriction in arterial sections of the coronary vascular system and to compare the reaction of the arterial sections with that of the arterioles. The arterial sections, which were under study, were distinguished by a considerable vascular resistance. Special attention was paid to interferences from metabolic and extravascular influences on coronary vascular resistances as well as to the dependence of sympathetic reactivity on control conditions. The effect of α-receptor vasoconstriction on the peripheral coronary pressure, i.e. the perfusion pressure of coronary collateral vessels, was of particular interest.

Methods

General preparations

Mongrel dogs weighing 25 – 40 kg were anaesthetized with chloralose (40 mg/kg i.v.), urethane (250 mg/kg i.v.), and morphine (3 mg/kg s.c.), and they were ventilated with room air at an expiratory pressure of 5 cm H₂O. Arterial P₀₂, Pₐ₎₂, and Pₕ were controlled half-hourly (BMS2 Radiometer, Copenhagen). Left ventricular and aortic pressures were measured with a double-tip manometer (PC 780 Millar). Heart rate and left ventricular pressure rise (dP/dt) were recorded. Left ventricular diastolic pressure was recorded with high reading accuracy (± 0.1 mm Hg). A 7F Courand catheter was advanced from the right jugular vein into the coronary sinus. Coronary sinus blood pressure was measured. The right position of the catheter tip was verified post mortem. Left thoracotomy at the 5th intercostal space was performed and the pericardium dissected. The dogs were anticoagulated with heparin (750 U/kg i.v. initially, and 430 U/kg i.v. two-hourly).

Coronary perfusion

A steel cannula was advanced from the right carotid artery into the common branch of the left coronary artery and carefully tied up with minimal lesion of the