

depicts the somatotopical arrangement of the eye muscle representation within the semilunar ganglion. The cells innervating the superior rectus and the superior oblique muscles were localized in a more dorsal layer than those supplying the inferior rectus and the inferior oblique muscles. In the lateral portion of the proprioceptive pool responses to stretch of the lateral rectus muscle were found at an intermediate layer between the superior and the inferior muscle representations and partially intermingled with them. In the medial portion of the pool the medial rectus muscle was represented and it intermingled dorsally with the couple of the 2 superior muscles and ventrally with that of the inferior rectus and inferior oblique. Thus, in some central penetrations, it was possible to record successively responses to stretch of the superior rectus, superior oblique, inferior rectus and inferior oblique (figure 2). The result of the present investigation showing a localization of the cells innervating the proprioceptors of the extraocular muscles in the semilunar ganglion is not sur-

prising, since other researchers demonstrated a somatotopic organization in the semilunar ganglion of the cells supplying different trigeminal receptors (skin, tongue, dental pulp, vibrissae)⁸⁻¹².

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Prostaglandin A₂ (PGA₂) increases the coronary vascular resistance in the guinea-pig isolated heart preparation¹

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Summary. Prostaglandin A₂ (PGA₂) in concentrations of $1.5 \cdot 10^{-8}$ to $3 \cdot 10^{-6}$ M was found to produce concentration-dependent increase in the coronary vascular resistance of the guinea-pig isolated heart without alterations in myocardial contractile force and oxygen consumption.

PGE₂ is one of the most active and specific coronary dilating agents in the guinea-pig heart² and was found to be formed by this organ after infusion of arachidonic acid, the precursor of the bisenoic prostaglandins³. However, the question arises whether this coronary vasodilation is due to PGE₂ in itself or another derivative, such as PGA₂, which was also found to be released together with PGE₂ after infusion of arachidonic acid from the dog heart in situ⁴ and is assumed to produce profound coronary vasodilation in whole animal experiments⁵. However, the action of PGA₂ on the coronary vascular system and myocardial mechanics was not investigated systematically under better defined and more comparable in vitro conditions. Experiments were therefore performed, which were designed to evaluate the cardiac and coronary activity of PGA₂ and the possible involvement of this compound in the coronary action of PGE₂.

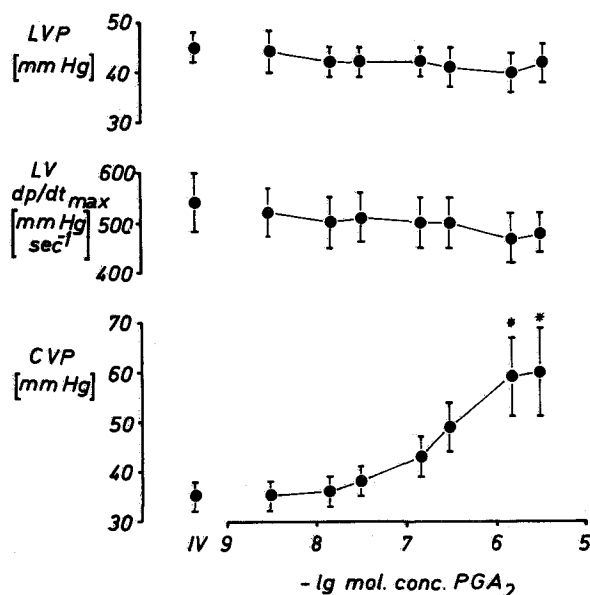
Guinea-pigs of either sex (body weight 300–380 g) were killed by cervical dislocation after pretreating the animals with heparin (10 mg/kg i.p.). The hearts were quickly removed and perfused isovolumetrically (10 ml/min) with Tyrode solution at 32°C via the aorta. The hearts were electrically driven at constant rate of 180 beats/min. Left ventricular peak systolic pressure (LVP), maximum velocity of contraction (LV dp/dt_{max}) and the coronary perfusion pressure (CVP) were measured as described earlier⁶. The CVP was assumed to be a direct expression of the coronary vascular resistance, since the perfusion rate was held constant. From PGA₂-oil⁷, a stock was prepared, containing 10 mg/ml PGA₂ in absolute ethanol. From this stock, a more diluted solution was made by addition of physiological saline, containing 10^{-4} g/ml PGA₂. This aqueous solution was stored on ice and freshly prepared each day.

Statistical analysis was performed using the t-test for non-paired samples. The mean and standard error ($\bar{x} \pm S.E.M.$) of *n* observations are quoted in the text. The level of significance was 0.05. The cumulative application of PGA₂ to the perfusion fluid of 8 hearts led to a marked increase in the CVP ($p < 0.05$), while the left ventricular mechanics remained unchanged ($p > 0.05$) (figure). This increase in the CVP was dependent on concentration, when 1.5×10^{-8} to 3×10^{-6} M PGA₂ were applied. Further increase in the concentration of PGA₂ up to 6×10^{-6} M produced no stronger responses. This coronary action of PGA₂ was reversible after wash-out and was also seen, to essentially the same extent, when the cardiac adrenoceptors were blocked previously with propranolol (5×10^{-8} g/ml) and phenoxybenzamine (1×10^{-7} g/ml), amounting 49 ± 12 at PGA₂ 6×10^{-6} M before and 52 ± 15 mm Hg in presence of the blocking agents ($p > 0.05$, $n = 4$).

For each animal the coronary reaction was calculated in % of its maximum individual response. The ED₅₀, i.e. 50% of the maximum increase in the CVP, was estimated graphically and was found to be 219 ± 45 nM. Pretreatment with indomethacin (5×10^{-7} g/ml), a potent in-

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hibitor of prostaglandin biosynthesis, also in isolated heart preparations^{3, 8, 10}, did not inhibit the coronary constrictory activity of PGA_2 ($n = 6$). There was also no influence by PGA_2 on the myocardial oxygen consumption, as seen in another 3 experiments, when the pO_2 was measured continuously in the coronary venous effluent by the polarographic technique as described earlier⁹.



Effect of PGA_2 on left ventricular peak systolic pressure (LVP), maximum velocity of contraction (LV $\text{dp/dt}_{\text{max}}$) and coronary vascular pressure (CVP) in the guinea-pig isolated heart preparation. The mean and standard error ($\bar{x} \pm \text{S. E. M.}$) are given. Each point represents 8 observations. *, $p < 0.05$, when compared with the initial value (IV) before addition of PGA_2 .

The results give evidence of a direct coronary constrictory action of PGA_2 in the guinea-pig isolated heart preparation in the absence of alterations in myocardial contractile force and oxygen consumption. Similar vasoconstrictor activity was described earlier for the vessels of the isolated rat pancreas¹¹ and the hind-paw of the dog¹², and was now also found for the coronary vessels of the isolated perfused guinea-pig heart. However, this action is only weak, as seen by comparing the ED_{50} of 219 nM for PGA_2 with the ED_{50} of about 1 nM for PGE_2 regarding its relaxing action on the coronary vessels in the same system¹³. Both this weak and vasoconstrictory activity of PGA_2 , as well as the findings that arachidonic acid and bradykinin decrease the coronary vascular resistance in the isolated heart, an effect which can be inhibited by pretreating the animals with indomethacin¹⁰, a drug which in itself increases the coronary vascular resistance in the guinea-pig isolated heart¹⁴, are consistent with the view that PGA_2 is not involved in the maintenance of the coronary tone per se, and add further evidence for the possible role of E-type prostaglandins in this process.

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Etude, à l'aide de microsphères, de la distribution de l'irrigation carotidienne chez le rat¹

Evaluation of the carotid blood distribution in the rat using radioactive microspheres

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Summary. The carotid blood distribution has been evaluated using radioactive microspheres in 13 regions of the head and in 10 cerebral structures of rats, and the intracerebral distribution of blood has been studied comparatively after injection of microspheres into the left cardiac ventricle or into the internal carotid artery. The principal results indicate that the cerebral tissue is not supplied by the external carotid blood, whereas about 30% of the internal carotid blood goes to the brain, and that the pontomedullary region receives its blood mainly from the vertebral artery.

La distribution intracérébrale de l'irrigation carotidienne ou vertébrale a été étudiée chez diverses espèces animales telles que le lapin², le chat³⁻⁵ et le chien⁴⁻⁷ à l'aide de différentes techniques utilisant des colorants, des produits de contraste, des résines et plus récemment des microsphères radioactives qui permettent d'apprécier de façon quantitative l'irrigation des différentes régions du cerveau. Ces études ont mis en évidence des différences dans les modalités de l'irrigation cérébrale selon l'espèce considérée: néanmoins, il ressort de ces travaux que l'irrigation du tronc cérébral est assurée essentiellement par le sang en provenance des artères vertébrales. Chez le rat, les modalités de la vascularisation cérébrale sont encore très mal connues et dans une étude récente, Wellens et al.⁸ rapportent que les artères vertébrales se distribuent essen-

tiellement au niveau des muscles du cou et ne participent pratiquement pas à l'irrigation du cerveau, ce qui différencierait le rat des autres espèces de laboratoire. Dans le présent travail, nous avons évalué la distribution de l'irrigation carotidienne chez le rat par injection de microsphères radioactives dans l'artère carotide, externe et interne, et étude de leur distribution dans différentes régions de la tête et du cerveau. De plus, la distribution intracérébrale des microsphères injectées dans la carotide interne a été comparée à celle qui est obtenue après injection des microsphères dans le ventricule cardiaque gauche.

Matériel et méthodes. Notre étude a été réalisée chez des rats mâles IFFA Credo d'un poids moyen de 250 g, anesthésiés au pentobarbital (45 mg/kg, i.p.).