a slit of half-length (16 mm) by which we commit a lesser mistake than with the slit of full length. For this purpose, the 16 mm slits are cut in the cylindrical mantle rectangularly with the normal slits.

In successful electrophoretic strips with a perfect partition of protein bands, we can of course obtain still more exact values, when using a very narrow densitometric slit *e.g.* 1 mm. In this case it would be an advantage to concentrate the rays on the narrow slit in putting a cylindric lens (a glass round rod of 2·5 mm diameter) close in front of the strip.

The work with this densitometric Adapter Model IV is very quick and exact. It has the great advantage that we can directly obtain the electrophoretic graph. Even a non-qualified worker is able easily to learn the manipulation of it. The only disadvantage is that the device cannot be improvised but must be made very precisely. It is equal to the special and expensive densitometric apparatuses.

Acknowledgement. I am much obliged to render thanks to Mr. A. Šmíd for a very perfect technical construction of the models of my Adapter and for a very devoted and initiative technical collaboration by the merit of which my work, after numerous experiments, has been led to a successful end.

I am also much obliged to render thanks to Dr. J. Bártek, Doctor-in-Chief of Central Laboratory of our Hospital for a friendly collaboration and precious advices.

A. Palacky

Pediatric Department of the County Hospital at Uh. Hradiste, Czechoslovakia, December 28, 1956.

Zusammenfassung

Der Verfasser beschreibt eine Apparatur zur objektiven Auswertung der Papierelektrophoretogramme. Er benützt ein Coleman-Spektrophotometer und einen besonders entwickelten Adapter.

Informations - Informationen - Informazioni - Notes

STUDIORUM PROGRESSUS

Regulation of Blood Pressure and Hypertension

By C. Heymans*

Experiments performed in different laboratories[1] have shown that blood pressure is regulated reflexly by the action of arterial pressure itself on receptors sensitive to pressure located into the walls of the blood vessels of the aortic arch and carotid sinus areas.

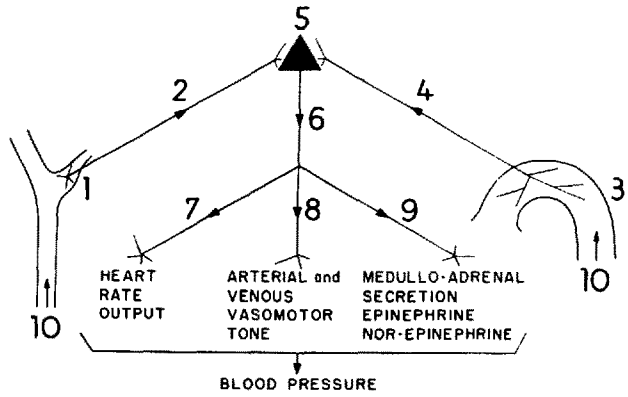
These presso- or baro-receptors are connected by means of the aortic and carotic sinus nerves with the nerve centres regulating and maintaining arterial blood pressure at normal levels. Any deviation of arterial pressure induces, by means of the aortic and carotid sinus baroreceptors, compensatory reflexes and adjustments so as to restore arterial blood pressure at its normal levels. The efferent pathways of this self-adjustment of blood pressure, of this physiological blood pressure homeostasis, are the vagus and sympathetic nerves adjusting heart rate and cardiac output, the sympathetic vasomotor nerves adjusting the peripheral vasomotor tone of small arteries and veins, and thus the peripheral vascular resistance and the circulating blood volume, and the sympathetic nerves regulating the epinephrine and norepinephrine secretion of the suprarenal glands. These hormones also act, but by an humoral way, on heart rate, cardiac output and peripheral vascular resistance (Figure).

Further experimental observations showed that the aortic and carotid sinus baroreceptors and their nerves are not only the fundamental means of the blood pressure homeostasis, but also are the reflex buffer or moderator mechanisms of the systemic arterial pressure.

* Department of Pharmacology, Medical School University of Ghent (Belgium).

[1] H. E. Hering, *Die Karotissinusreflexe auf Herz und Gefässe* (Verlag Steinkopf, Dresden/Leipzig 1927). – Eb. Koch, *Die Selbststeuerung des Kreislaufes* (Verlag Steinkopf, Dresden/Leipzig 1931). – C. Heymans, *Le Sinus carotidien* (Presses Universitaires, Paris 1929). – C. Heymans, J. J. Bouckaert, and P. Regniers, *Le Sinus Carotidien et la zone homologue cardio-aortique* (Doin et Cie, Paris 1933).

Paralysis of the baroreceptors or section of the aortic and carotid sinus nerves induces, indeed, a marked rise of blood pressure, thus a condition of acute or chronic arterial hypertension.



Schema of Self-regulation of Blood Pressure. *1* Carotid sinus baroreceptors; *2* Carotid sinus nerves; *3* Aortic baroreceptors; *4* Aortic-depressor nerves; *5* Cardio-vascular centres; *6* Efferent pathways of self-regulation of blood pressure; *7* Vago-sympathetic nerves to heart; *8* Vasomotor nerves to arteries and veins; *9* Sympathetic nerves to medullo-adrenal glands; *10* Arterial pressure acting on baroreceptors.

Everybody would agree that hypertension is a deviation of the arterial pressure, a 'resetting' of arterial pressure from normal to higher levels. In order to evaluate the origin and mechanism of this 'resetting' of blood pressure in hypertension, we ought to know in full details the physiological mechanisms maintaining blood pressure at normal levels. The physiology of blood pressure regulation is, thus, fundamental for the evaluation of the pathogenesis of hypertension.

As arterial pressure is maintained or restored reflexly at normal levels by the action of blood pressure itself on the aortic and carotid sinus baroreceptors, the question arises: how does arterial pressure act on these baroreceptors?

It has long been accepted that arterial pressure and its deviations act directly on the aortic and carotid sinus receptors. It has been stated, furthermore, that arterial pressure acts on the baroreceptors, by deforming and

thus stretching the arterial walls in which these receptors are located. Experimental evidence showed, indeed, that if deformation of the baroreceptive arterial walls by a rise of arterial pressure was prevented, the baroreceptors did not respond to the rise of pressure[2].

These experimental observations suggested that the responses of the baroreceptive arterial walls to pressure could play an important role in the mechanism of blood pressure homeostasis.

This suggestion has been investigated in our laboratory[3]. Experiments showed that an increase of tension of the baroreceptive arterial walls and, thus, an increase of pressure-response of the baroreceptive carotid sinus and aortic arterial walls induced by local application to the baroreceptive arterial walls of drugs, such as epinephrine, norepinephrine, serotonine or vasopressine, provokes a 'resetting' of the baroreceptive mechanisms of blood pressure regulation from normal to lower levels. Decrease of intramural tension and pressure-response of the baroreceptive arterial walls induced by local application of drugs such as papaverine, priscoline or nitrite, provokes, on the contrary, a 'resetting' of the baroreceptive mechanisms of blood pressure regulation from normal to higher levels.

Experiments performed on the isolated carotid sinus preparation also showed[4] that epinephrine and norepinephrine increase the pressure-response of the arterial wall and decrease their distensibility to steady and pulsatile pressures.

Further experiments also disclosed that an increased intramural tension of the arterial wall, induced into the empty but baroreceptive normal innervated carotid sinus areas, also provokes a stimulation of the baroreceptors and thus a reflex fall of arterial pressure.

These experimental observations thus showed that the pressure-response of the baroreceptive arterial walls and the degree of their intramural tension or intramural stretch or distorsion plays a fundamental role in the physiological mechanisms of blood pressure homeostasis.

Under normal conditions the degree of intramural tension, stretch or distorsion of the baroreceptive arterial walls depends on the level of arterial pressure, and mainly on the pressure-response of the baroreceptive arterial walls themselves, thus on the resistance of the baroreceptive arterial walls to deformation by the intravascular pressure. Therefore, any alteration in pressure-response of the baroreceptive aortic and carotid sinus arterial walls will induce a 'resetting' of the blood pressure homeostasis to lower or to higher pressure levels. The biological condition of the baroreceptive arterial walls, thus, plays a fundamental role in the mechanisms of blood pressure regulation. The biological condition and, thus, the response of the baroreceptive arterial walls to pressure are, indeed, more important than the level of pressure itself for the physiological mechanisms of blood pressure homeostasis. A decrease of intramural tension and resistance to distension of the arterial baroreceptive arterial walls, would induce a shift to a higher 'set' of baroreceptor function, a 'resetting' of the responses of the baroreceptors from the normal to a higher pressure level. Consequently, a higher arterial pressure would be necessary to provoke a response of the arterial pressure buffering mechanisms. This condition could be an important component in the mechanisms of arterial hypertension. Experiments of our laboratory[5] support this suggestion, as they show that in chronic renal hypertension provoked in dogs, the arterial pressure is 'reset' from high to normal or low levels when the intramural tension and the pressure-response of the baroreceptive arterial walls is increased.

Recent experimental observations of MCCUBBIN, GREEN, and PAGE[6] are also in agreement with our hypothesis. They measured baroreceptor responses by means of electroneurograms in normotensive and chronically hypertensive dogs, and showed that the baroreceptor mechanisms are, indeed, reset to the hypertensive pressure level in animals with chronic renal hypertension and that the buffer baroreceptive reflexes tend to maintain, rather than to prevent, the chronic phase of renal hypertension and are, presumably, an important component in the mechanisms of chronic renal hypertension.

Let us hope that still more detailed information concerning the physiological mechanisms of blood pressure homeostasis, mainly concerning the factors maintaining or affecting the intramural tension and the pressure-response of the baroreceptive arterial walls, will also provide more definite information concerning the origin and mechanism of hypertension.

Résumé

L'auteur résume les mécanismes de l'homéostasie physiologique de la pression artérielle et indique que la réaction à la pression intravasculaire des parois artérielles barosensibles constitue un facteur prédominant de la régulation réflexe de la pression artérielle. Une élévation du seuil d'excitabilité des parois artérielles barosensibles pourrait constituer un élément causal important de l'hypertension artérielle.

[2] W. H. HAUSS, H. KREUZINGER, and H. ASTEROTH, Z. Kreislaufforsch. *38*, 28 (1949).

[3] C. HEYMANS and G. VAN DEN HEUVEL-HEYMANS, Arch. int. Pharmacodyn. *83*, 520 (1950). – H. MAZELLA, S. C. WANG, C. HEYMANS, and G. R. DE VLEESCHHOUWER, Arch. int. Pharmacodyn. *89*, 122 (1952). – C. HEYMANS and G. VAN DEN HEUVEL-HEYMANS, Circulation *4*, 581 (1951). – C. HEYMANS, A. L. DELAUNOIS, and G. VAN DEN HEUVEL-HEYMANS, Circul. Res. *1*, 3 (1953). – C. HEYMANS and A. L. DELAUNOIS, Arch. int. Pharmacodyn. *96*, 92 (1953); Science *114*, 546 (1951). – C. HEYMANS, A. L. DELAUNOIS, and A. L. ROVATI, Arch. int. Pharmacodyn. *109*, 245 (1957).

[4] C. HEYMANS and A. L. DELAUNOIS, Arch. int. Pharmacodyn. *96*, 92 (1953); Science *114*, 546 (1951). – C. HEYMANS, A. L. DELAUNOIS, and A. L. ROVATI, Arch. int. Pharmacodyn. *109*, 245 (1957).

[5] G. MATTON, Arch. int. Pharmacodyn. *103*, 13 (1955); *110*, 472 (1957).

[6] J. W. MCCUBBIN, J. H. GREEN, and I. H. PAGE, Circul. Res. *4*, 205 (1956).

Congressus

DEUTSCHLAND

9. Internationaler Kongress für Radiologie 1959

Der *9. Internationale Kongress für Radiologie* findet in der Zeit vom 23. bis 30. Juli 1959 in München (Deutschland) unter dem Präsidium von Prof. B. RAJEWSKY, Frankfurt/Main (Deutschland) statt.

Generalsekretär ist Prof. Dr. HANS V. BRAUNBEHRENS, München. Alle Auskünfte über den Kongress erteilt das Kongreßsekretariat in Frankfurt am Main, Forsthausstrasse 76.

Dr. VIKTOR LOECK

Corrigendum

J. WILLEMS: *Orientierte Aufwachsungen auf hochpolymeren organischen Stoffen*, Experientia, Vol. XIII, Heft Nr. 7, S. 276 (1957).

Auf der rechten Spalte, unterste Zeile, sollte es heissen: $2\ c = 5{,}06$ Å (anstatt $2\ c = 6{,}06$ Å).