

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 pressure².

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 laboratory³. 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, serotonin or vasopressin, 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, prisolone 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 showed⁴ 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 laboratory⁵ 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 PAGE⁶ 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.

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: $2c = 5,06 \text{ \AA}$ (anstatt $2c = 6,06 \text{ \AA}$).

² W. H. HAUSS, H. KREUZINGER, and H. ASTEROOTH, Z. Kreislauforsch. 38, 28 (1949).

³ C. HEYMANS and G. VAN DEN HEUVEL-HEYMAN, Arch. int. Pharmacodyn. 83, 520 (1950). – H. MAZELLA, S. C. WANG, C. HEYMANS, and G. R. DE VLEESCHOUWER, Arch. int. Pharmacodyn. 89, 122 (1952). – C. HEYMANS and G. VAN DEN HEUVEL-HEYMAN, Circulation 4, 581 (1951). – C. HEYMANS, A. L. DELAUNOIS, and G. VAN DEN HEUVEL-HEYMAN, 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).