EFFERENT AND AFFERENT IMPULSATION IN THE RENAL NERVES DURING THE DEVELOPMENT OF EXPERIMENTAL NEUROGENIC HYPERTENSION

Yu. L. Pines

From the Laboratory of Electrophysiology (Head – V. E. Delov) of the I. P. Pavlov Institute of Physiology (Dir. – Academician V. N. Chernigovskii) of the Akad. Nauk SSSR, Nauk SSSR, Leningrad
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According to the contemporary theory on hypertension [11, 14], the primary reason for the development of this disease is overexertion of higher nervous activity, leading to constant excitation in the vasculomotor centers of the cerebral cortex and the subcortical structures. The theory holds that as a result of intensification of the vasoconstrictive neutral influences, ischemia occurs in a number of internal organs, including the kidneys. Under these conditions, the latter secrete pressor substances of the renin and renol type, which, continuously entering the blood and acting on the vessels, support and reinforce the hypertonic condition.

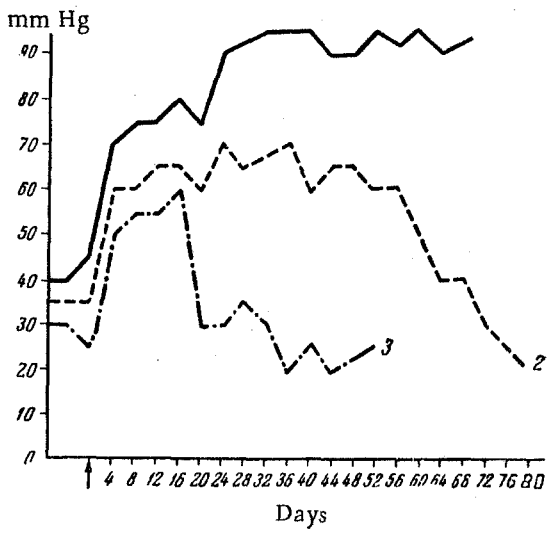

Fig. 1. Changes in the arterial pressure with the development of experimental neurogenic hypertension. The arrow notes bilateral transection of the nerve depressors and the sinocarotid nerves.

At the present time, however, data have been accumulated which show that the renin pressor mechanism is initiated reflexly as a result of intensification of neural influences on the kidneys from the centers [25]. Neuroreflexic initiation of this kidney mechanism in the pathogenesis of hypertension is particularly suggested by the easing of experimental neurogenic, centrogenic, and full-blown renal hypertension following denervation of the kidneys, or by inhibition of its development after preliminary denervation of the kidneys [3, 17, 18, 20, 22, 25].

Initiation of the kidney mechanism in the development of hypertension suggests an intensification not only in efferent nerve influences on the kidney, but also in the afferent impulsations from the kidney receptors. Thus, it was shown that pressor substances secreted by the kidneys in hypertension, not only manifest a direct vasoconstrictive activity, but also a reflex one, through excitation of interoceptors [2, 4, 12]. Therefore, hypertension must be regarded as a neurogenic illness not only in the initial stages of its development, but also in the subsequent ones, when the renal humoral factor is brought into play. In light of this, hypertension must be considered a cortico-visceral disease, connected with nerve impulsations from the centers to the periphery and back again.

We undertook the problem of investigating the efferent and afferent impulsation in the renal nerves during the development of experimental neurogenic hypertension.

EXPERIMENTAL METHOD

The experiments were carried out on 47 rabbits. Neurogenic hypertension was caused by bilateral transection of the nerve depressors and the sinocarotid nerves in the neck, with subsequent coating of the region of bifurcation of

common carotid artery with a 10% solution of phenol in ethyl alcohol. The operation was performed in two stages (first on one side, and after 7-10 days, on the other). The arterial pressure was measured in the central artery of the ear, following the method of Grant-Rothschild. After exposing the kidney, the renal nerve in the plexus between the artery and vein was prepared and transected. The potentials of the efferent impulses were conducted away from the central portion of the renal nerves, and the potentials of the afferent impulses, from the peripheral portion, using silver electrodes. The conducted potentials were led into an amplifier, and recorded on a cathode oscillograph. The frequency characteristic of the amplifier was rectilinear, in a diapason of 10-1500 hertz.

EXPERIMENTAL RESULTS

Measurement of the arterial pressure was carried out after every 4-6 days, beginning with the 2nd-4th day after bilateral transection of the nerve depressors and sinocarotid nerves. In the course of 1-3 weeks after transection, the pressure gradually elevated by 10-60 mm Hg. During the next 2-8 weeks it stayed at the level attained or fell to its original level and lower (Fig. 1). The reduction in the neurogenic hypertension which we observed supports the statements of a number of authors [11, 14] on the instability of this type of hypertension. Efferent and afferent impulsation was recorded in the renal nerves of the rabbits before and during the development of hypertension, as well as in the animals with pressures that were decreased to the original level and lower.

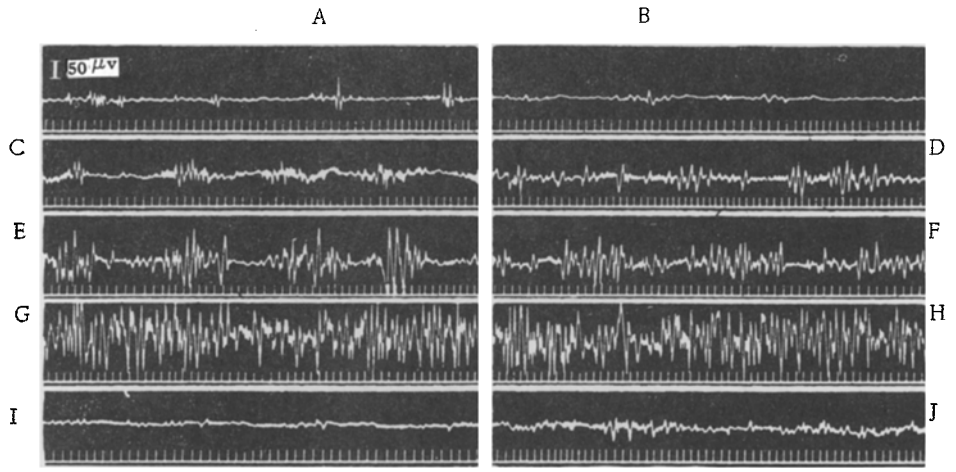

Fig. 2. Changes in the efferent impulsation within the renal nerves with development of experimental neurogenic hypertension. A, B) Without hypertension; C, D) with elevation of the arterial pressure by 10-20 mm Hg; E, F) the same, by 20-40 mm Hg; G, H) the same, by 40-60 mm Hg; I, J) with lowering of the arterial pressure to the original level or below. Time markings (0.02 sec).

Efferent impulsation in the renal nerves of the control animals without hypertension was represented by oscillations grouped into a pulse rhythm, each lasting up to 20 msec (Fig. 2A, B). This impulsation reflects the sympathetic influence on the kidneys [15]. The number of oscillations per pulse group was equal to 2-4, and the amplitude, 40-50 microvolts. In the animals with experimental hypertension, the intensity of the efferent impulsation appeared to be directly dependent upon the level of the arterial pressure; with elevation of the pressure by 10-20 mm Hg, the number of oscillations in the individual group increased to 6-8, and the amplitude, to 60 microvolts (Fig. 2C, D); with elevation of the pressure by 20-40 mm Hg, the number of oscillations in the individual group rose to 12-16, and the amplitude, to 100 microvolts (Fig. 2E, F); with elevation of the pressure by 40-60 mm Hg, the number of oscillations per group increased to 32-36, so that the pulse groups of potentials melded together into continuous oscillatory activity, and the amplitude reached 150 microvolts (Fig. 2G, H); when the pressure was lowered to the original level or below, the number of oscillations per group decreased to 2.6, and the amplitude, to 30-40 microvolts, or the impulsation completely stopped (Fig. 2I, J). In all the experiments where the efferent impulses were recorded, the sensitivity of the oscillographic apparatus was the same. The genesis of efferent impulsation in the renal nerves with the development of hypertension, as seen in our experiments, reflects the presence of a durable excitation of the pressor nerve apparatus belonging to the blood circulation [11, 14].

Afferent impulsation in the renal nerves of animals without hypertension, as a rule, was either absent or manifested by rapid (1-2 milliseconds) and slow (up to 20 milliseconds) oscillations with a frequency of 2-6 per second

and an amplitude of about 10 microvolts (Fig. 3A, 1). (Fig. 3 only shows oscillations of the rapid type). In the animals with hypertension, when the arterial pressure was elevated by 10-20 mm Hg, the frequency of the oscillations rose to 15-20 per second, and the amplitude increased to 15 microvolts; with an elevation in the pressure by 40-60 mm Hg, the amplitude increased to as much as 20 microvolts, with the same frequency. Characteristically, the oscillations in this case grouped into a pulse rhythm (Fig. 3B, 1 and C, 1). With lowering of the pressure to the original level and below, the frequency of the oscillations decreased to 1-2 per second, and the amplitude, to 10 microvolts, or the impulsation completely disappeared (Fig. 3D, 1).

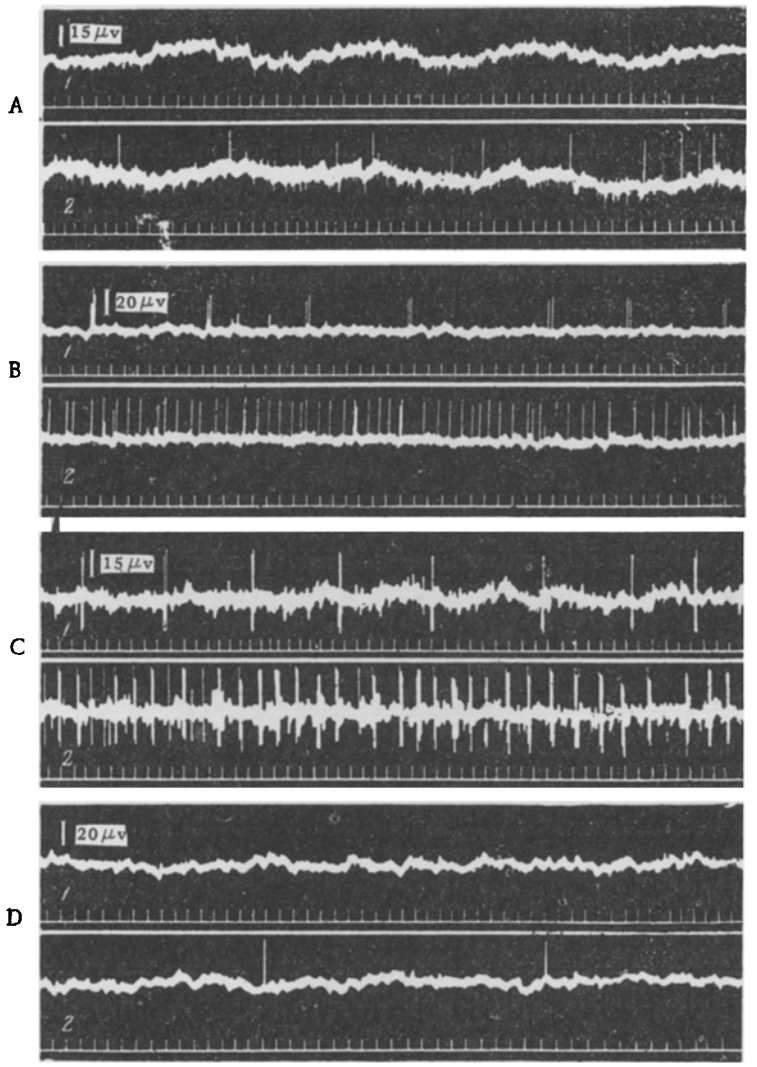

Fig. 3. Changes in the afferent impulsation within the renal nerves with development of experimental neurogenic hypertension. A) Without hypertension; B) with elevation of the arterial pressure by 10-20 mm Hg, grouping of the potentials in a pulse rhythm (300 per minute); C) with elevation of the arterial pressure by 40-60 mm Hg, grouping of the potentials in a pulse rhythm (360 per minute); D) with lowering of the arterial pressure to the original level and below; 1) original impulsation; 2) impulsation to supplementary stimulation of the receptors, caused by pinching of the renal vein. Time markings (0.02 secs).

Supplementary stimulation of the renal receptors, by means of local dilation of the renal vessels (stopping the outflow of blood from the kidneys by compressing the renal vein) also intensified impulsation in direct proportion to the level of the arterial pressure (Fig. 3A, 2B, 2C, 2D, 2); where the animals had elevated pressures, even there we noted grouping of the oscillations in a pulse rhythm.

One of the possible reasons for intensification of the afferent impulsation with development of experimental hypertension appears to be excitation of the renal receptors, caused by dilatation of the glomeruli as a result of predominant spasm in the vessels leading away from the glomeruli [1, 5-8, 11, 13, 19, 20]. It is known that the glomerular apparatus of the kidneys is extremely rich in receptors [21, 26]. Another possible reason for intensification of the afferent impulsation with development of hypertension in this case is excitation, or elevation of excitability, of the renal receptors under the influence of unoxidized metabolic products in the kidney tissue, formed under the conditions of kidney ischemia or under the influence of specific pressor substances [2, 4, 12]. In connection with this, it is pertinent to recall the facts observed by us earlier, in experiments on cats, wherein the afferent impulsation in the renal nerves increased in frequency with disturbance of the oxidative processes in the organ by compression of the renal artery [16].

The well defined grouping of the afferent impulses into a pulse rhythm, observed with experimental hypertension, is apparently also explained by elevation of the excitability of the renal receptors, and the production of their synchronous activity as a result of this. In addition, an essential reason for the pulse grouping of the potentials may be the development of a capillary pulse in the renal glomeruli, as a result of histamine and acetylcholine accumulation in the kidneys during hypertension [9, 10, 23, 24]. Thus, it was established by us earlier, in experiments on cats, that the introduction of histamine or acetylcholine solutions into the general circulatory bed or into the kidney causes the appearance of afferent potentials in the renal nerves, synchronous with the pulse.

SUMMARY

An oscillographic investigation of the efferent and afferent impulsation in the nerves of the renal plexus was performed in rabbits during the development of experimental neurogenic (reflex) hypertension. The efferent and afferent impulsation is increased in frequency and amplitude with an arterial pressure rise; grouping of the afferent impulses in the pulse rhythm appears. The facts, pointed out may be physiologically interpreted demonstrating the neuro-reflex mechanism of involvement of the kidneys into the pathogenesis of hypertensive disease.

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