Introductory Remarks Concerning Neural Factors in Essential Hypertension

By Edgar A. Kahn, M.D.

The vasoconstrictor effect of neural stimuli from frontal and hypothalamic centers to the blood vessels, including the renal arterioles, is reviewed. Such a theory of the pathogenesis of hypertension is probably deficient, since humoral or atherogenic factors also play a part. Nevertheless, splanchnicectomy may sometimes alleviate superimposed vasoconstriction and help in treatment of the patient.

My interest in hypertension began when I assisted Dr. Max Peet at his first splanchnic section for hypertension in 1933. The patient was an extremely sick 29-year-old man whose blood pressure did not drop below 260/150 mm Hg on bed rest. He was almost blind from hemorrhages and exudates and showed papilledema of 4 diopters. We would now consider the operation on this patient an inadequate sympathectomy although the splanchnic nerves were divided. The patient’s blood pressure, nevertheless, fell to normal and remained there for almost 10 years, his vision becoming perfect.

The theory on the basis of which Dr. Peet performed his first splanchnicectomy was that of shunting the blood from the peripheral circulation into the splanchnic area, thus providing a safety valve. He also believed that splanchnic section would diminish neurogenic adrenal secretion. When the work of Dr. Goldblatt appeared we realized that there might be a third mechanism that the operation relieved when it was successful, that is, renal vasoconstriction. I visited Dr. Goldblatt soon after his first paper appeared, and will never forget his kindness and modesty, nor his reticence in accepting our belief that splanchnic section might relieve essential hypertension by removing the theoretical Goldblatt clamps of renal vasoconstriction.

Dr. Peet believed that so-called essential hypertension was of neurogenic origin, originating probably in the hypothalamus. Actually, this area is not a center of emotions itself, but a center for emotional expression. Stimulation of the hypothalamus produces dilation of the pupils, sweating, pilo-erection, and increase in blood pressure, as well as the emotional expression of fear, rage, and even crying. The actual emotions which give rise to expression by way of the hypothalamus, however, originate, certainly for the most part, in the frontal and temporal lobes.

Some years ago Dr. Piero Foa was studying the preoperative and postoperative renal blood flow on Dr. Peet’s patients. He was doing this procedure on a preoperative labile hypertensive individual when a science reporter entered the laboratory. The latter informed the patient that he was an exceedingly brave man to undergo such a dangerous experiment for the sake of mankind. The renal plasma flow immediately dropped to 10 per cent of the previously recorded level and the blood pressure reached astronomical figures. We have all seen a number of cases of hypertension relieved by the reassurance of the family physician, and it would make an excellent theory that essential hypertension arises from a disturbance of emotion transmitted by way of the hypothalamus ultimately through vasoconstrictor nerves, but it can not be that simple. There must be another factor, an organic or chemical substratum in a fixed hypertension; for though many people are
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emotionally disturbed, only a certain proportion of the same emotional type develop the disease. There is little doubt concerning the deleterious effect of the emotions in hypertension, as in ulcer and in thyroid disease. There is equally little doubt that psychotherapy is not going to stop the progress of a hereditary atherosclerosis. To put it another way, if peripheral blood vessels are in vasoconstriction from an abnormal pressor substance, as Goldblatt showed, surgical excision of vasocostrictor nerve fibers or their inhibition by ganglion blocking agents will have little effect on the caliber of the vessels unless the formation of the pressor substance can be stopped.

We might compare essential hypertension to Raynaud’s disease. Raynaud’s disease is a syndrome which results from abnormal vasoconstriction usually in the upper extremities. In Raynaud’s original paper he stated that the disease was more common in nervous females and realized the effect of the emotions in producing these attacks. We have seen the phenomena of Raynaud’s disease arising from a tumor of the hypothalamus, a tumor of the stellate ganglion, inflammatory lesions of the sympathetic ganglia, lesions of the spinal cord and lesions intrinsic in the blood vessels themselves. Hypertension can persist (just as can Raynaud’s syndrome) after so-called total sympathectomy or after the neurogenic factor has been to all intents and purposes eliminated. The increased tonus of the smooth muscle of the blood vessels which gives rise to the increased peripheral resistance must then be intrinsic or have a chemical basis.

Nevertheless, splanchnicectomy for hypertension has been successful in hundreds of cases. It could be considered a physiologic operation, however, only when a known cause of hypertension is being relieved, as for example, when neurogenic renal vasoconstriction is known to exist. Though I do not doubt that such renal vasoconstriction may be a common cause of essential hypertension in man, the cases where we can establish this at present are few and far between.

We recently evaluated 257 cases operated upon only by Dr. Peet, the dissection being carried bilaterally to or above the sixth dorsal ganglion, the splanchnic nerves and lower dorsal sympathetic chain being excised through the twelfth dorsal ganglion. I used only Dr. Peet’s cases as I do not believe the average surgeon or internist can evaluate his own results impartially. The study would have been fairer still had we used only men for evaluation, since women can so frequently tolerate severe hypertension for years. Seventy-eight per cent of the patients who had been operated upon survived at least 5 to 8 years. Fifty-five per cent of the survivors maintained a drop in the diastolic pressure of 20 or more points, from 5 to 8 years post-operatively.

If we could only pick those patients who would respond to splanchnic section, I am sure that the operation would still be in vogue. When drugs or antirenin are produced which can lower the blood pressure of hypertensive patients without producing severe side effects, the operation will be abandoned. Splanchnic section will then be a phase in the history of the treatment of hypertension, as was subtotal thyroidectomy in the relief of hyperthyroidism. Nevertheless, we have learned a lot about hypertension because of the operation. For example, we knew and published in 1935, after the first 60 splanchnic sections had been performed, that operation was invariably unsuccessful when azotemia was present, that high blood pressure was not a compensatory phenomenon to force the blood through narrowed arteries, that the fundus changes of hemorrhages, exudates, and papilledema could disappear, and that vessel changes in the nature of medial hypertrophy at least could be reversed by operation.2

In recent years, the men in the laboratories have contributed a great deal to our knowledge of hypertension. Certainly as knowledge increases, fewer and fewer cases will be called by that misnomer, essential hypertension. Already many cases of unilateral renal disease,
coarctation of the aorta, and pheochromocytoma have been removed from that category. The main solution of the problem of hypertension still lies ahead, but the work of Goldblatt will remain as one of the great contributions of our time.

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