TYPES OF ARTERIAL HYPERTENSION AND THEIR RECOGNITION

A. CARLTON ERNSTENE, M. D.

Although the most common cause of a consistent elevation in blood pressure is essential hypertension, there are a number of other pathologic states which are regularly attended by increased arterial pressure. Certain of these states are an established part of general medical knowledge; others are decidedly unusual, and one has been set aside as a distinct clinical entity only within recent years. Needless to say, it is of considerable importance that the hypertension of all these conditions be differentiated from the common type of elevated blood pressure. Fortunately, differential diagnosis usually is a simple matter, although in certain instances it may be impossible to arrive at a definite conclusion even after the most detailed clinical and laboratory studies. It will be our purpose in this communication to review the various causes of hypertension with particular reference to those features which are of value in distinguishing one type from another.

ESSENTIAL HYPERTENSION

Essential hypertension is characterized by a continuous, though often variable, elevation of blood pressure in the absence of glomerulonephritis and other conditions known to cause increased arterial tension. The term must still be interpreted to indicate a symptom complex rather than a disease entity, and it is probable that as medical knowledge increases, distinct types of cases will be separated from the main group on the basis of specific etiologic factors. In essential hypertension both the systolic and diastolic blood pressure are increased. Tests of renal function, in the early stages, give normal results, but as time passes, a progressive diminution in function frequently is recorded, and a certain number of patients, probably not more than 10 per cent of the entire group, ultimately die of uremia. Cardiac complications, such as congestive heart failure and coronary artery disease, are of much greater clinical importance and are the cause of death in approximately 60 per cent of all patients, while cerebral vascular accidents constitute the terminal event in 15 or 20 per cent of the cases.

Although the actual cause or causes of essential hypertension remain unknown, there is general agreement that the elevation in arterial pressure is due to increased resistance in the peripheral circulation. Recent studies[1] indicate that both in this type of hypertension and in the hypertension resulting from glomerulonephritis, the augmented resistance is present in all parts of the systemic circulation and is due to an

intrinsic increase in the tonus of the blood vessels and not to organic changes in the vessel walls or excessive vasomotor stimulation.

Essential hypertension may be divided into three types, the benign, intermediate, and malignant, according to differences in the ophthalmoscopic findings. In the benign form, sclerosis of the retinal arteries of variable degree is observed without other abnormal changes. The malignant type, on the other hand, is characterized by the presence of neuroretinitis with edema of the optic disk and surrounding retina. The edema frequently is out of proportion to the other retinal changes, such as hemorrhagic areas, cotton-wool exudates and arteriosclerosis. Differentiation of typical cases of benign and malignant hypertension is comparatively simple, but there is a group of cases which cannot be placed in either of these classes. Ophthalmoscopic examination reveals more extensive changes, in the form of exudates and hemorrhagic areas, than are present in patients with benign hypertension, and yet, because of the absence of papilledema, the condition cannot be classified as malignant hypertension. These cases are therefore placed in the intermediate group. Even in the malignant group, renal function frequently is normal or only slightly reduced at the time the condition is first recognized, and the erythrocyte count and hemoglobin content of the blood are seldom more than slightly diminished until after the development of renal insufficiency. Although the malignant and intermediate forms of essential hypertension usually develop upon earlier hypertension of the benign type, the majority of patients with benign hypertension run the full course of the disease to a fatal termination without progressing to the intermediate or malignant stages. The characteristic pathologic feature of the malignant and intermediate types consists of widespread hypertrophy of the media and proliferation of the intima of the arterioles[2, 3]. These changes have been observed in practically all the organs and tissues of the body, and they are usually absent in the benign form of hypertension. In view of the observations of Prinzmetal and Wilson[1], however, the progression from benign hypertension to the more severe types cannot be attributed to the development of these pathologic changes. There is, on the other hand, considerable evidence to support the view that the change to the malignant type is due to greatly increased, generalized vasoconstriction.

The differentiation between the various forms of essential hypertension is of great importance in prognosis. Whereas the benign type may be present for several years without causing serious complications in patients with malignant hypertension, death usually occurs from congestive heart failure, cerebral hemorrhage, uremia, or a combination

of these causes within two years after the diagnosis is made. All types of essential hypertension have been observed occasionally in young individuals, but they occur most commonly in persons more than 45 years of age. The relationship to age is at times of importance in distinguishing between essential hypertension with renal insufficiency and chronic glomerulonephritis wth hypertension but cannot be taken as an absolute guide.

Hypertension Due to Glomerulonephritis

Arterial hypertension is a common feature of acute diffuse glomerulonephritis and may even precede the appearance of edema and other characteristic signs of the disease. The pressure usually returns to normal as the evidence of active nephritis subsides; when the elevation persists, it may be interpreted as evidence that the disease is progressing to the subacute or chronic phase. The differentiation between essential hypertension and acute glomerulonephritis with hypertension offers no difficulty. The clinical history, the presence of edema, and the urinary findings, particularly hematuria, serve to establish the diagnosis of acute glomerulonephritis with certainty.

Although chronic glomerulonephritis usually is accompanied by hypertension, the blood pressure may remain within the limits of normal for a considerable period of time in the so-called nephrotic type in which edema dominates the clinical picture. In patients in whom nephritic hypertension has been present for some time, ophthalmoscopic examination may reveal findings which are indistinguishable from those observed in any of the three types of essential hypertension. The differentiation between typical cases of chronic glomerulonephritis and essential hypertension usually is easily made. A history of former acute glomerulonephritis is of primary importance but in many patients cannot be obtained either because the illness occurred so many years earlier that it has been forgotten or because the attack was so mild that it passed unnoticed.

Chronic glomerulonephritis occurs in individuals of all ages, but, in contrast to essential hypertension, it is relatively uncommon after the age of 45 years. The most valuable clinical signs of chronic glomerulonephritis are the presence of diminished renal function, albuminuria with urinary casts and microscopic hematuria, edema of renal origin, and anemia. These findings serve to distinguish the disease immediately from those cases of essential hypertension in which renal function has not been impaired. When the same findings are encountered, however, in a patient between 30 and 45 years of age, it may be impossible to determine, in the absence of a history of earlier acute nephritis,

whether the condition is chronic glomerulonephritis or essential hypertension which has progressed to the stage of producing marked impairment of renal function. A similar clinical picture in individuals less than 30 years of age will usually be the result of chronic glomerulonephritis, while in patients past the age of 45 years, essential hypertension is the most probable cause.

The actual height of the blood pressure is of comparatively little value in differential diagnosis, but extremely high pressures are more common in essential hypertension than in chronic glomerulonephritis. Although the heart becomes hypertrophied from nephritic hypertension of long duration, congestive myocardial failure occurs in only a small number of patients, and the common cause of death is, of course, uremia.

Hypertension Due to Urinary Obstruction and Polycystic Disease of the Kidneys

Long continued urinary obstruction, regardless of the cause, may produce a constant elevation in arterial pressure. Among the most common conditions which produce hypertension in this way are hypertrophy of the prostate, bilateral renal calculi, and bilateral ureteral obstruction due to malignant disease of the bladder, uterus or cervix. Essential hypertension may occur in patients who have any of these disturbances, and the decision as to whether the elevation in pressure in a particular case is of this type or due to urinary obstruction frequently must be postponed until the obstruction has been relieved.

In the majority of patients with polycystic disease of the kidneys, the blood pressure is elevated, at times to very high levels. Evaluation of the hypertension depends upon recognition of the underlying disease.

Hypertension in Pregnancy

Elevation of the blood pressure during pregnancy may be due to chronic glomerulonephritis, essential hypertension, or toxemia of pregnancy. In patients with chronic glomerulonephritis, hypertension may have been present for some time before conception or, in individuals in whom nephritis is present in the so-called latent stage, it may not develop until pregnancy has progressed for two or three months or even longer. Recognition that the elevated pressure is the result of glomerulonephritis depends upon the presence of albuminuria with microscopic hematuria and casts early in pregnancy, diminished renal function and a history, when obtainable, of earlier acute glomerulonephritis.

Because essential hypertension is relatively uncommon in individuals less than 45 years of age, elevation of the blood pressure during preg-

nancy is seldom due to this condition. When essential hypertension is the cause, the pressure usually will have been above normal for some time before the onset of pregnancy. In order to establish a diagnosis, it is necessary that the renal function be normal or only slightly impaired, since more than slight diminution in function almost always is to be regarded as evidence of glomerulonephritis in patients in the childbearing age. In addition to the small group of individuals in whom essential hypertension complicates pregnancy, patients who are constitutionally predisposed to this type of elevated blood pressure may first show increased tension during gestation, usually in the later months. Following delivery, the blood pressure generally returns promptly to normal, but after a period of a few months to several years, it again becomes elevated[4].

The most common cause of hypertension during pregnancy is that form of toxemia to which the term "kidney of pregnancy" or "low reserve kidney" is applied. This type of toxemia is characterized by the development, in the later months of pregnancy, of elevated pressure, usually of only slight or moderate degree, albuminuria with urinary casts, and edema. In spite of the edema and urinary findings, renal function is normal. The condition may progress more or less rapidly to pre-eclampsia and eclampsia, but usually it is possible to carry the patient to term or at least to the period of viability. Delivery is followed by a prompt return of the blood pressure to normal and disappearance of the albuminuria and edema. There is no evidence that patients who have experienced this type of toxemia are predisposed to the subsequent development of essential hypertension or impaired renal function.

Hypertension Due to Lead Poisoning

Hypertension with elevation of both the systolic and diastolic pressure is a common accompaniment of acute and chronic lead poisoning. In the acute phase of plumbism, the increased tension is the result of vasoconstriction due to the direct action of lead upon the smooth muscle of the arterioles. If the individual has been exposed to lead for but a short time and is subsequently protected against further exposure, the hypertension is usually transitory and the pressure returns to normal as the symptoms of intoxication subside. Prolonged exposure, on the other hand, frequently results in permanent elevation in pressure even though the patient has experienced no symptoms of acute poisoning. In a certain number of these patients, tests of renal function indicate the presence of kidney damage, and postmortem studies reveal renal lesions similar to those observed in patients with essential hypertension

and secondary vascular nephritis. Whether these pathologic changes are the direct result of the action of lead upon the kidneys and are the principal cause of the elevated pressure of chronic lead poisoning has not been determined. They may be but a secondary effect of the hypertension itself. In other individuals who have been subjected to prolonged exposure, the blood pressure is elevated but renal function is normal and no anatomic change can be demonstrated in the kidneys. In these patients it is probable that the increased tension has resulted from the direct effect of the lead upon the blood vessels.

The diagnosis of hypertension due to lead poisoning is established by the history of exposure, the occurrence of symptoms of lead intoxications, and the presence of signs of lead absorption. Of the latter signs, punctate basophilia of the erythrocytes, the gingival lead line, and the presence of lead in the urine are the most important. Lead is excreted from the body principally through the gastro-intestinal tract, but the presence of lead in the feces is of little diagnostic importance unless the possibility of the patient's having ingested lead can be excluded absolutely. The presence of lead in the urine, on the other hand, is absolute proof of earlier lead absorption.

Hypertension Due to Arteriosclerosis

It is a matter of common knowledge that even extreme degrees of generalized arteriosclerosis are encountered frequently in individuals whose blood pressure tends toward the lower rather than the higher limits of normal. In a certain number of patients with advanced arteriosclerosis, however, a moderate elevation in systolic tension is recorded. The characteristic feature of this hypertension and the feature which differentiates it from essential hypertension is that the increased systolic value is associated with a normal diastolic reading [5].

Hypertension in Coarctation of the Aorta

Coarctation of the aorta consists of a localized stenosis or atresia of the descending arch of the aorta at the point of insertion of the ductus arteriosus. One of the most striking features of the condition is the development of collateral arterial circulation between the branches of the aorta above and below the site of stenosis. This is accomplished mainly by the following routes: (1) by anastomoses between the superior intercostal artery of the subclavian and the first aortic intercostal arising from the aorta below the site of the constriction, (2) by anastomoses between the aortic intercostal arteries and the subscapular artery and its branches, particularly the circumflex scapulae, and (3) by anastomoses between the internal mammary and the deep epigastric arteries. The extent to which each of these vessels takes part in the

collateral circulation varies considerably in different patients. The collateral circulation may manifest itself clinically by the presence of pulsating vessels over the back, epigastrium, and lower anterior part of the thorax, prominent subclavian and subscapular arteries and palpable deep epigastric arteries. In addition, roentgenographic examination of the thorax reveals erosions of the inferior borders of the ribs bilaterally.

Because of the resistance offered to the flow of blood by the stenosed aorta and the collateral arterial pathways, patients with coarctation present hypertension in the vessels of the arms and relatively low blood pressure in the legs. In addition, the femoral pulse wave often is retarded and diminished in amplitude or even absent. This diagnostic sign is seldom absent, and because its presence immediately suggests coarctation of the aorta, the femoral pulse should be palpated in all individuals with elevated brachial blood pressure.

Hypertension Due to Pituitary Basophilism and Tumors of the Adrenal Cortex

Cushing[6], in 1932, described a syndrome due to basophilic adenomata of the pituitary gland in which hypertension is a constant finding. The syndrome usually occurs in young adults and, in addition to the increased blood pressure, is characterized by the rapid development of obesity which is frequently painful and is confined to the face, neck, and trunk, by purplish abdominal striae, and softening of the bones which often leads to marked kyphosis due to collapse of the vertebral bodies. In females, amenorrhea and hypertrichosis of the face and trunk develop early. Hyperglycemia and glycosuria are common findings, and the patients frequently have a dusky or plethoric appearance. The erythrocyte count is often increased, the skin at times is extremely dry, and purpuric manifestations have been described. The patients seem to be very susceptible to infections, particularly of the respiratory tract. The symptoms of the malady appear to result in part from disturbances in the function of the anterior lobe of the pituitary gland, and in part from secondary effects upon the thyroid and parathyroid glands, and the pancreas, and the suprarenals. The suprarenal glands[7] usually are found to be hypertrophied at autopsy, and in a few cases an adenoma of the adrenal cortex has been present.

Although pituitary basophilism is not common, the syndrome is of considerable importance because of its possible bearing upon the entire problem of essential hypertension. The pituitary body has been mentioned frequently in discussions on the etiology of essential hyperten-

sion, and the Cushing syndrome supplies a definite example of a relationship between the pituitary and elevated blood pressure.

It has been recognized for many years that tumors of the adrenal cortex may be responsible for the development of a symptom complex in which hypertension occupies a prominent place. The clinical picture which results from these neoplasms may be indistinguishable from that of pituitary basophilism. It is quite possible that in some of the earlier cases reported as instances of adrenal tumors causing hypertension, Cushing's syndrome was really present but was not recognized because of inadequate examination of the pituitary body. Recently, however, cases have been reported in which all the features of pituitary basophilism have been associated with an adenoma of the adrenal cortex and a perfectly normal hypophysis[8].

SUMMARY

This discussion has been limited to a consideration of those forms of hypertension which are encountered most frequently in clinical practice or are of special interest because of their possible bearing upon the problem of the etiology of essential hypertension. That an elevation in blood pressure is observed either as a transient or sustained phenomenon in several other conditions is well known, but in these states the increased tension either is an unimportant part of the clinical picture or is encountered so uncommonly as to warrant omission from consideration at this time. Differentiation of the various types of hypertension is of fundamental importance with respect to treatment and prognosis, and one is always amply repaid for whatever effort he may expend in attempting to arrive at a proper classification in a particular case.

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