Jefferson's⁴ case was the first published in England, and is worthy of perusal. He draws attention to the causative role of heart disease in the recorded cases. Bull of Oslo estimates that in 80 per cent. of all cases there is a diseased heart, the left side being the usual source of the embolus. These emboli are often multiple; post-operative, more frequently single.

In placing the site of the embolus it is well to remember that it tends to lodge either at the bifurcation of the brachial artery or at the junction of a large branch—for example, the profunda—with the main trunk. The exact site of the initial pain is a very important guide. Occasionally a lump may be felt in the course of the artery at the end of the pulsating area.

The general operative technique follows the usual lines of arterial surgery. The operation is best done under infiltration anaesthesia. The utmost gentleness in manipulation and the careful preservation of any small branches are essential. The wound is kept moist with 1 per cent. citrate solution to prevent coagulation. If the embolism is at the bifurcation, the incision in the artery (which need not be more than one centimetre long), must be made above the embolus; if thought to be at the junction of the profunda branch, the incision would be sited below it, and the embolus and any clot extracted distally. It may come away on releasing the clamp, or it may require to be milked out or extracted with forceps. In a late case, secondary thrombosis may be extensive, and

the radial artery difficult to clear. In one such case the radial at the wrist was opened, a cannula inserted, and the vessel flushed through with a syringe and 2 per cent. citrate solution. A continuous stitch is probably better than interrupted sutures, but, in a small artery like the brachial, more liable to tear, unless great delicacy in handling is observed. Fine waxed thread is the ideal suture.

Prognosis

As regards prognosis, the time element is the crucial factor. Key states that three-quarters of all brachial or axillary emboli do well if operated on within ten hours of onset. Early operation means less secondary thrombosis. In bifurcation emboli, the arm should be immobilized in comfortable extension for a few days, the hand being left free. The further outlook depends on the origin of the embolus or the gravity of the heart disease. Secondary fatal emboli have occurred in cases already successfully operated on for the same condition. A study of the literature shows, as might be expected, that the most favourable cases are those following operation, and without grave heart disease, the patient being still under observation—conditions ideal for early diagnosis and treatment.

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ETIOLOGICAL FACTORS IN HYPERPIESIA *

BY

C. W. C. BAIN, M.B. OXON., M.R.C.P. LOND.

PHYSICIAN, HARROGATE INFIRMARY

Hyperpiesia is a malady in which there is a raising of both the systolic and diastolic pressures. Two points in this definition require emphasis. First, the pressures must be sustained. It is well known that excitement will cause a rise in the blood pressures, especially in the systolic pressure; no blood pressure should be accepted as high until the examiner has satisfied himself that the patient is entirely at his ease. Secondly, in the diagnosis of hyperpiesia the diastolic pressure is more important than the systolic. The systolic pressure is liable to be affected by conditions, such as arterial sclerosis and aortic regurgitation, which have nothing in common with hyperpiesia. A patient with a diastolic pressure of less than 85 mm. of mercury is not suffering from hyperpiesia. As an instance, a patient has had a systolic pressure of 200 mm. or over for eighteen years. Her diastolic pressure has never exceeded 80 mm., however, and now, at the age of 80, she is in good health, without symptoms referable to the circulatory system.

Anyone showing a sustained blood pressure of 150 systolic and 90 diastolic should be regarded as a potential hyperpietic, and should receive treatment aimed at lowering his pressures. Should true hyperpiesia develop, both pressures will tend to rise progressively. At first the increases may not be maintained, and the pressures may vary considerably from time to time. Indeed, hyperpietic crises are liable to occur at any stage of the disease with swift, large elevations of the pressure. The height which the pressure may finally attain in any patient will depend upon the severity of the disease. Usually the systolic reaches a level of 200 to 300, and the diastolic from 110 to 160.

After a time the cardio-vascular system responds to the stress of these continued high pressures by structural

changes, which are permanent. The heart hypertrophies and enlarges to the left, the "cœur de sabot" of French radiologists. Obliterative changes take place in the arterioles; the intima hypertrophies and the lumen may become quite obstructed. This arteriolar hyperplasia is widespread, but it affects particularly the kidney, pancreas, spleen, and suprarenals. The muscular tissue of the arteries hypertrophies, while the elastic tissue degenerates and fibrous tissue takes its place. The thickening of the vessels can best be observed in the retinal fields, since these are the only small arteries that can be seen. When this second stage of hyperpiesia has been reached a raised blood pressure is necessary for the circulation to be maintained through the capillaries. Even if it were possible to lower the pressure permanently, the patient would not feel better for the change. On the other hand, a temporary reduction of the load under which the cardio-vascular system is labouring may afford a needed rest to the heart and vessels, and may postpone a breakdown.

In the third stage of the disease the cardio-vascular system wears out, and death occurs, either by the bursting of a cerebral artery, or from failure of the circulation consequent on the exhaustion of the heart. Occasionally, when the requisite head of blood is no longer forthcoming, elimination through the kidneys fails completely, and the patient dies in coma.

It is generally accepted that hyperpiesia is due to a contraction of the arterioles. The velocity of the pulse wave, or the time taken by the blood stream to travel from the subclavian to the radial at the wrist, may be twice as fast in patients with high blood pressure as in normal subjects. Yet the total velocity of the blood flow, or the time taken by the blood to make a complete circuit, is not increased at all. Using the histamine flush method devised by Weiss, Robb, and Blumgart,¹ we have confirmed their findings that the times are normal, or else prolonged if the heart is beginning to fail; they are never rapid. The acceleration of the flow in the arteries is therefore compensated by the obstruction offered by the contracted arterioles, or rather the raised pressure is itself compensatory to the arteriolar contraction.

* Read to the Section of Hydrology and Climatology at the Annual Meeting of the British Medical Association, Eastbourne, 1931.

What is the cause of this arteriolar contraction? Diathesis, psychic factors, overwork, sedentary occupations, obesity, endocrine changes, circulating pressor bases, have all had their advocates. Their very number is a confession of our ignorance. There is no doubt that high blood pressure is often familial. Lord Dawson found a percentage of school children had pressures above the normal,² but it is as yet too early to say whether any of them will eventually develop true hyperpiesia. Nervous causes and overwork certainly raise the pressure, but many men overwork and never suffer from hyperpiesia, and neurotic people often have low pressures. Sedentary occupations are blamed for many things: hyperpiesia is only one of them. Hyperpiesia and overweight may coexist, but it is doubtful whether the majority of obese persons have high pressures. There is no reason why circulating pressor bases should not be eliminated, since it is known that the kidneys remain normal until their arterioles become affected with those of the rest of the body.

Hyperpiesia will probably be found to be a deficiency disease. As noted by Ismail³ it has something in common with diabetes. In both diseases the usual time of onset is in early middle age; both run a prolonged course, have a certain familial tendency, and are frequently found together. As in diabetes, hyperpiesia is much more severe when it occurs in young people, and its course is more rapid: the so-called malignant hypertension of Keith and Wagener.⁴

A certain tension of the arterioles is necessary for everyone. This tension is unlikely to be maintained by the sympathetic nervous system, which is usually reserved for the rapid changes called for by the external environment. By analogy with other systems of the body,

it is probable that a chemical agent is responsible. It may well be that this substance, or substances, derived from protein, is normally present in excess, and the fine adjustment is made by a hormone, which converts it into something that can be excreted by the kidney. This view would accord with the work of Bain⁵ and Major,⁶ who have both found in normal urine substances with a pressor action; these were absent, or reduced, in the urine of those suffering from high blood pressure. If the hormone is inadequate, the arterioles will be over-stimulated, and the blood pressure will have to be raised to maintain the circulation through the capillaries. According to the extent of the hormone deficiency, various grades of hyperpiesia will result. Overwork and worry are important secondary factors, which will aggravate still further a state which is already abnormal.

Whether this view is correct, time will show. Hyperpiesia is a malady for which we have at present no cure; it will inevitably run its course. The potential hyper-pietic, however, demands active treatment to prevent, if possible, the condition becoming established. In the later stages, also, much can be done to mitigate the disease, both by countering the secondary factors of overwork and worry, and by intensive courses aimed at relieving temporarily the stress upon the overburdened cardio-vascular system.

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TREATMENT OF HYPERPIESIA*

BY

W. EDGECOMBE, M.D., F.R.C.P., F.R.C.S.

PHYSICIAN, HARROGATE INFIRMARY.

The treatment of hyperpiesia must be based on our conception of the cause and the mode of production of the disease. Of the causes, diathesis—the inherent, often familial, tendency to the affection—cannot be removed. Psychical causes, such as overwork, anxiety, mental stress, and worry, are more or less amenable to modification, and must be remedied when possible. Inactive metabolism accompanying obesity and sedentary occupations is susceptible to suitable treatment. Endocrine factors—hyperadrenalism, subthyroidism, and disturbances of ovarian secretion, as at the menopause—can be partially controlled, though not with any great certainty, except in the case of myxoedema. Of the pressor bases concerned in the production of hyperpiesia we have little exact knowledge. Believing them to arise in the alimentary canal and to be derived from protein, we endeavour to control them by reducing the protein intake, and by promoting active elimination of waste products by the bowel, kidneys, and skin. Aperient and diuretic mineral waters and intestinal lavage by the Plombières douches are valuable means to this end. In illustration of the relation between intestinal infection and hyperpiesia the following two cases are of interest.

CASE I

Male, aged 49, when seen in 1913 had a blood pressure of 140/90, and was found on examination to have a large swelling in the region of the ascending colon which was dia-

gnosed as a diverticulitis. In January of the following year he was operated upon for suspected cancer of the bowel; the growth was found inoperable, and the abdomen was closed. In June his blood pressure was 145/95. In 1915 it had risen slightly to 150/95; in 1916 it was 160/100, reduced after a course of baths to 150/95. He was lost sight of until August, 1923, when he reappeared with a blood pressure of 230/130, and symptoms of slight weakness and dragging of the left leg. The swelling in the abdomen had almost disappeared, but was still just palpable. In seven years his blood pressure had risen from 160/100 to 230/130. A course of baths reduced it to 200/124. In 1924 his blood pressure was 220/125, reduced after a course to 200/105, and he died of cerebral haemorrhage in 1925. It is not unreasonable to infer that the chronic infection of the bowel was the cause of the hyperpiesia.

CASE II

Male, aged 48 in 1915, now aged 64. The records of his blood pressure date from 1916, and are seen to have gradually mounted year by year without any cause being discovered. Each year he had a short course of spa treatment. Between 1928 and 1929 there was a sharp rise, for which no explanation could be found. His blood pressure in May, 1929, was 230/110 before and 180/110 after a course of baths. In June, 1929, he had an acute perforation of the bowel, which was found on operation to be from a ruptured diverticulum. He made a good recovery. In December, 1929, his blood pressure was 190/100. An x-ray examination after a barium enema disclosed extensive diverticulitis of the large bowel. Since then he has remained in good health.

Consideration of the mode of production of hyperpiesia points the way to physical methods of treatment. The primary fault lies in the peripheral circulation; it is the narrowing of the arterioles and capillaries, and the consequent diminution of the total channel area available to the blood stream, that determines the high blood pressure; the enlargement of the heart is a secondary resultant phenomenon. Any measures that dilate the vessels and

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