REVIEW ARTICLE

High blood pressure—ancient, modern and natural

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II

NATURAL HISTORY AND PROGNOSIS

Natural history

The study of the natural history of hypertension is difficult. It would be of great value to take a large sample of people at the age of 30 years and follow them until they are 60 years or more, though this is not the type of research which appeals to young men who want quick results and neither does it appeal to old men who may not live to see the results. Probably, an enormous number of people would have to be studied to yield results of statistical significance. Life insurance companies can give important information but their information is subject to three disadvantages. First, they exclude all candidates with significantly raised blood pressures; secondly, the number of women is always small and, thirdly, there is a tendency amongst examining doctors to record pressures which are just below the accepted maximum of the company. Perhaps general practice holds the key to a study which could be of value, particularly if it was carried out in an area where the population was relatively static. Such a study would need the co-operation of many separate practices and probably four generations of family doctors.

The natural history of hypertension is extremely variable as is well summed up by Fishberg (1954):

> The clinical duration of the condition varies between wide limits. There are instances where an individual who has never sought medical advice suddenly dies on the street. At necropsy a coronary occlusion or cerebral haemorrhage is found, the origin of which in hypertension is revealed by the presence of cardiac hypertrophy and renal arteriosclerosis. On the other hand, the hypertension may last for three or four decades and even do the subject no harm until he finally dies of some disease having no relation to the hypertension. These pictures constitute the extremes, and the vast majority of instances of essential hypertension fill all conceivable gaps between them.

Early essential hypertension is not usually marked by any characteristic symptoms, and signs are minimal. The onset of the disease is seldom observed except in patients who happen to have a medical examination for some unrelated condition, who are having an examination for insurance purposes, or as part of antenatal care. The clinical picture of the middle stages of essential hypertension is also extremely variable. The later course consists largely of the manifestations of cardiac and vascular complications. In the benign phase those complications that kill are either quantitatively related to the arterial pressure, as for example cardiac failure, or are manifestations of atheroma, which seems to be more frequent and severe at high pressures, but occurs at low. A specific clinical picture is presented by the malignant phase, produced by the effect of the very high blood pressure.

One of the results of Volhard and Fahr's (1914) classification was that for the first time it was thought that essential hypertension might end in two ways either benign or malignant. The end stage of the malignant phase was almost identical with that of chronic Bright's disease. The patient in the early malignant phase usually presents a distinct clinical picture and the peak age is about 40 years. A patient may present

with disturbances of vision, with severe headache, or less commonly, another complaint or the presentation may be sudden with a vascular catastrophe. Examination discloses a high diastolic pressure usually over 130 mms Hg. The urine is usually normal and renal function shows no gross upset. The fundus shows the early changes of hypertensive neuroretinopathy, with early papilloedema and ill-defined exudates in both eyes. Soon, blood and protein appear in the urine and the renal function begins to decline. The retinopathy increases in severity, haemoglobin falls, cardiac function fails, and death usually occurs within a year of one or a combination of uraemia, cerebral haemorrhage or left ventricular failure. The malignant phase also occurs in most cases of secondary hypertension and this was first pointed out by Derow and Altschule in 1935. In the benign phase the patient tends to present with symptoms at a later age, around 50 years and the condition can remain stable for years. A quarter of them die of some intercurrent illness, about a half die of cardiac failure and a quarter from cerebrovascular disease.

The effect of the increasing hypertension on the cardiovascular system is now well understood. The cardiac output is equivalent to the pulse rate times the stroke volume. In uncomplicated cases of hypertension, tachycardia is rarely a feature of the clinical picture and the heart steps up its output by increasing the stroke volume. Consequently, the myocardial fibres have to stretch further and are subjected to greater tension. In this way the heart manages to expel the increased volume of blood with a greater degree of mechanical efficiency. In short, the heart at least initially, works more economically. In absolute terms, however, its oxygen consumption is increased. The myocardium is able to adapt itself to this higher level of performance without requiring the assistance of neural stimuli, as described in Starling's law.

The increased peripheral resistance prevents the ventricle from emptying completely during systole and the residual blood is thus added to the blood which flows from the atrium into the ventricle during the next diastole, the result being once again, an increase in the volume of blood filling the ventricle during diastole. The heart cannot cope indefinitely with this added strain by working more economically. Once the strain exceeds a certain critical level, the mechanical efficiency of the heart muscle decreases, its oxygen requirement becomes steadily greater, more and more residual blood accumulates in the ventricle owing to incomplete systolic emptying, and the ventricles begin to dilate. When this stage has been reached, the heart can no longer maintain the required output simply by stepping up its stroke volume and, instead, it now has to beat faster and becomes increasingly dependent on stimuli provided by the sympathetic nervous system. Initially, this process can be delayed by a compensatory increase in the muscle mass of the myocardium but once the weight of the heart exceeds the threshold level of approximately 500G, its reserves become fully exhausted. Dilatation of the ventricles now proceeds at a rapid pace, while at the same time the performance of the heart continues to deteriorate. After a while the atria are no longer able to cope with the volume of inflowing blood, with the result that the blood pressure in the veins rises. Finally, typical signs of congestion in the pulmonary and systemic circulation (cardiac asthma, pleural oedema, leg oedema, hepatic congestion, renal congestion etc) provide an urgent warning that the end is nigh and at this stage only energetic treatment is likely to save the patient.

When this degree of decompensation has been reached, the arterial blood pressure may, owing to a reduction in cardiac output, decline to almost normal levels. In such circumstances, the reason for the heart failure may not be immediately apparent and is, in fact, often not discovered until the performance of the heart has been improved by treatment with digitalis and diuretics. In addition to the typical chronically progressive form of haemodynamic heart failure as outlined above, there is also an acute form resulting from a sudden rise in arterial pressure. Particularly dangerous in this

connection is the type of abrupt increase in peripheral resistance within the systemic circulation which occurs, for example, in acute glomerulonephritis or in hypertensive crises due to other causes. Here, the myocardium has no time to hypertrophy and thus to meet the challenge. Whereas the left ventricle, confronted with a sudden increase in resistance, can no longer empty properly during systole, the right ventricle carries on functioning as if nothing had happened (because resistance in the pulmonary circulation remains normal) and continues to pump the usual amount of blood into the left atrium. Consequently, too much blood accumulates in the pulmonary veins, fluid seeps out of them and floods the alveoli, causing pulmonary oedema and dyspnoea of such severity that the patient is liable to die within only a few hours.

The coronary arteries are also included in the natural history of chronic hypertensive cardiac failure. They have a significant bearing on the question as to why the myocardium cannot continue to hypertrophy without its efficiency being impaired because if it could, then presumably decompensation would never occur. In compensating cardiac hypertrophy, not only the thickness but also the number of the muscle fibres increase, and there may have to be an increase in the number of capillaries supplying them with blood. What does not, and what cannot increase, is the diameter of the coronary arteries where they branch off from the aorta as at that point a rigid non-elastic structure composed of connective tissues prevents an increase in diameter. This means that as the oxygen requirement rises in response to progressive hypertrophy, a stage is soon reached when not even the maximum quantity of blood which the coronary arteries are capable of conveying is sufficient to satisfy the heart's needs. Once this has happened, not even an increase in the velocity of the coronary blood flow will serve any purpose, because, to achieve this, the myocardium would have to work even harder and would require correspondingly more oxygen.

Owing to the inadequate oxygen supply, individual muscle fibres perish and are replaced by connective tissue. The muscle fibres in the immediate vicinity of such necrotic tissue hypertrophy in an attempt to make good these losses, but the overall result is nevertheless a progressive deterioration in the condition of the myocardium, with all that this implies. Coronary insufficiency may also give rise to angina pectoris, thereby aggravating the symptoms with which the hypertensive patient already has to contend. If the coronary arteries are also affected by atheroma then the resultant coronary insufficiency will be worse and the result of this may be that in chronic coronary insufficiency the patient may sustain small areas of muscle necrosis or he may suffer a dramatic acute myocardial infarction resulting in the death of a large mass of myocardial cells. It is possible that sustained high blood pressure may be a traumatic factor that could damage the vascular wall from inside the lumen of the blood vessel, which might encourage a thrombus to form.

There are three common modes of death from cerebral vascular disease in essential hypertension. The commonest is a thrombotic occlusion of an atheromatous intracranial artery. Less common is a thrombotic occlusion of an atheromatous extracranial artery, carotid or vertebral. The third is cerebral haemorrhage.

The most characteristic lesion of the benign phase of hypertension is to be found in the small arteries and arterioles, particularly in the kidneys. In the small arteries such as the interlobular, the elastica fragments and thickens the intima. In the arterioles a fatty hyaline deposit is found in the intima under the endothelium. Controversy exists as to the relationship between these changes and the elevated pressure. Goldblatt (1947) takes the view that these changes are the cause of the raised pressure, the arteriola thickening reducing the blood flow through the afferent glomerular arterioles. The majority of authorities however, consider these changes to be the consequence of hypertension and cite three arguments for this belief. First, these changes may be absent in early essential hypertension (Bell 1951); secondly, similar changes are found in secondary

hypertension; and, thirdly, the changes are less intense in the affected kidney if its renal artery is narrowed than in its intact fellow. Whatever is the truth, there is no doubt that these lesions are related quantitatively to the raised arterial pressure, and as the condition progresses, blood and protein appear in the urine, the blood urea rises and renal function steadily declines.

Prognosis

The chief causes of death in western Europe and the United States of America are now cancer and cardiovascular disease, and figures of death certification suggest that vascular disease is now the commonest cause of death in this country. Many of these patients with vascular disease have high arterial blood pressure. The experience of insurance companies has shown that mortality is related to arterial pressure, and that the increased mortality of those with high blood pressure is largely due to cardiovascular disease often in association with renal disease. The incidence of hypertension in the population has increased as more people now reach the age when it is more common, and the result is that, in countries such as our own where the expectation of life is high and infectious diseases that are common in other parts of the world are rare, arterial hypertension has become one of the major medical conditions seen by doctors. The number of patients with this condition seen in general practice is now high. It is the family doctor who sees the patient in the early stages, he has the best opportunity to notice the appearance of complications and almost invariably it is he who treats the patient in the final stages of the illness.

The realization that arterial pressure could prove useful in assessing life expectancy was borne out by the figures of insurance companies which began to accumulate from 1910. In 1950, Master, Dublin and Marks did a big study on 74,000 men and women in the United States of America, and produced a table of mean readings and standard deviations for age groups. Wood (1960) states that, "if a blood pressure 150/100 mms Hg or above means hypertension, then the prevalence of this disease is five per cent in young adults, 10–20 per cent in the fifth decade and 35–40 per cent in those between the ages of 60 and 65 years". McMichael (1961) referring to a survey carried out by Hamilton and Pickering *et al.* in 1954, stated that, if this survey was correct, about 13 per cent of the adult population have systolic blood pressures over 160 mms Hg.

Much of the variation in the published causes of death from hypertension is due to the method of selection. In Janeway's series there was a high incidence of uraemia, which may have been due to the inclusion of cases of chronic nephritis. Probably the most representative series are those of Hunter and Rogers (1923), which shows the data of insurance candidates, and of Bechgaard (1946), which shows the causes of death in 1,038 patients followed for four to 11 years at the Rigs Hospital, Copenhagen. About a third to a half died of heart disease, about a sixth of cerebral vascular disease, a tenth from renal failure and the remainder from a disease not directly attributable to hypertension.

The sex of the person seems to be important. In a study of untreated hypertension Leishman (1959) showed that in hypertension the male sex had an unfavourable prognosis and in his series which were watched over the period of time from 1946 to 1959, not a single male survived whose diastolic blood pressure was initially found to be over 130 mms Hg. The greater tolerance of women for high blood pressure was evident, and even in cases of malignant hypertension, the average duration of survival of the female was twice as long as that of the male. It is not known why women withstand hypertension better than men. Newman and Robertson (1959) in a study of treated hypertension also found a comparative immunity of women to the complications of hypertension.

Our understanding of the different courses exhibited by patients with essential hypertension is derived from the work of Volhard and Fahr (1914). They showed that

essential hypertension might follow one of two courses. In most patients, the course was long with little change from year to year and death, when at last it came, was due to heart failure, cardiovascular disease or intercurrent disease. These patients did not show albuminuric retinitis, nor did they develop more than a mild renal insufficiency. The kidneys obtained after death from such patients showed splitting of the elastica of the arteries and the arterioles showed a fatty hyaline change. Apart from areas of sclerosis due to these changes the nephrons were well preserved. In some patients, however, mostly younger and with typically a high diastolic pressure, the onset of a different course was heralded by the appearance in the fundus oculi of the changes characteristic of albuminuric retinitis; then, or soon afterwards, protein, red blood cells, and casts appeared in the urine; renal function began to deteriorate rapidly and death would occur usually within a year of the onset of retinitis, from uraemia, cerebral haemorrhage and heart failure. In these patients the kidneys after death showed not only the changes in the arteries and arterioles just described, but also severe endo-arteritis and changes in the renal substance resembling nephritis. In 1919 Fahr described the two types of arterial lesion which were always present in this condition which he named malignant nephrosclerosis, namely acute arteriolar necrosis and cellular intimal thickening. Subsequent work by others, notably Keith, Wagener and Kernohan (1928), Ellis (1938) and Fishberg (1939) have fully confirmed these early clinical and pathological studies of Volhard and Fahr.

It is to be noted that the term malignant hypertension refers to a clinical diagnosis, made on the finding of hypertensive neuroretinopathy in a patient with high blood pressure, a supporting fact being rapid deterioration in kidney function. The term malignant nephrosclerosis, is strictly speaking, a pathological diagnosis based on the histological findings in the kidney and other organs, and particularly on the presence of arteriolar necrosis. Though there is no reason to doubt that both series refer to the same set of disease processes, they are not quite synonymous, for there is not a precise correspondence in time between the onset of hypertensive neuroretinopathy and renal arteriolar necrosis.

Prognosing is a very difficult thing to do with most illnesses and this is exceptionally so with hypertension. Other diseases may be present which exert as great, or even a greater effect on the outcome, as the raised blood pressure. One of the biggest difficulties is that vascular degenerations on which prognosis largely depends, often give no sign of their presence until a cardiovascular catastrophe has occurred. There are certain signs and symptoms which carry a bad prognosis, such as bilateral papilloedema, pulsus alternans, gallop rhythm and nocturnal dyspnoea. However, the unexpected often happens, particularly a cerebral vascular accident.

The statistics supplied by insurance companies (Dublin *et al.* 1949) and Bechgaard's (1946) follow-up study of over 1,000 patients with hypertension have shown clearly that life expectancy diminishes with even small rises in the blood pressure and that excess mortality is due mainly to cardiovascular and renal disease. Leishman (1959) studied 211 patients with diastolic pressures initially more than 100 mms Hg over a period of about 13 years, or until their death. They were not given any treatment and one half (106) of these patients died from a cause which could be attributed to their hypertension. This represented 66 per cent of the men and 39 per cent of the women, a further indication of the resilience of the female sex in this condition. Also with the exception of 14 of these patients all died before they were 60-years old.

In reporting upon average weights of men, the interesting comment is made from insurance statistics (Dublin *et al.* 1949) that whereas the average weight of women has fallen from the previous (1909–27) study, the weight of men has increased by about five pounds. The lowest mortality is shown to occur amongst people well below average weight. The study confirmed the generally accepted fact that blood pressure tends to

increase with increase in weight. The actuarial data presented suggest that pressures over 140/90 mms Hg are associated with an appreciable extra mortality. A moderate degree of hypertension in an overweight male would seem to provide him with an increased mortality rate of about 60 per cent over the overweight normotensive male, or a rate of 70 per cent over the average weight, normotensive male.

Similarly, Gubner and Ungerleider (1959) state that when two of those factors; obesity, hypertension and hypercholesterolaemia are associated, the chance of developing coronary artery disease is approximately five times greater than for persons who have none of these abnormalities. Dahl and Love (1957) also presented evidence that the combination of overweight with high salt intake predisposed to an appreciable grade of hypertension. In contrast it is a common clinical experience that the prognosis in hypertensive overweight women is appreciably better than for their male counterparts. It would appear that the overweight male hypertensive deserves special dietary consideration.

The prognosis in essential hypertension is related more to the effect of hypertension on vital organs, than to the height of the blood pressure itself. If the function of the heart and kidneys in particular, is significantly impaired, then the prognosis is not good. Because the prognosis is influenced by many factors, consequently the effects of blood pressure reduction are dissimilar in individual patients. Perera (1955) found in 500 patients with hypertension that the average age at onset was 32 years and at death 52 years and the commonest cause of death was cardiac failure. Once papilloedema developed the outlook was poor. Leishman (1959) described the progress of 211 untreated patients with hypertension, regularly followed since 1946. He compared them with the progress of 73 patients treated by lumbodorsal sympathectomy and 118 patients treated with ganglion-blocking drugs. Half of the untreated cases died and the number of deaths in the treated cases was only one third of the deaths in untreated cases. Many other doctors have reported improved prognosis with the use of ganglion-blocking drugs, either by themselves or in combination with other drugs, such as serpasil. These reports include those of Dustan *et al.* (1958), Kincaid-Smith, McMichael and Murphy (1958).

The most convincing evidence of benefit is to be seen in the regression of pathological changes in the retinal arteries and arterioles and even the most severe vascular changes seen in the young patients can improve. Kincaid-Smith, McMichael and Murphy (1958) and Harrington *et al.* (1959) reported on a series of 83 patients where hypertension was severe enough to justify the use of ganglion-blocking drugs, during the period 1951–58. The results observed were compared with a control series previously reported. The expectation of life of the treated patients was increased by a factor of six to eight times over that of the untreated control series and prolonged survival was particularly seen in those patients where renal function was normal or only slightly impaired at the start of treatment. Death was most frequently due to uraemia, and heart failure as a cause of death was considerably diminished in the treated group.

Widespread evidence exists that modern medical treatment greatly improves the prognosis of patients with hypertension but there is as yet no actual evidence that in patients whose only abnormality is a moderately raised blood pressure, treatment will prevent complications in later life. Because of the long course of benign essential hypertension such evidence cannot in any case be available for many years. Two important papers have described the results of treatment in malignant hypertension. First, Dustan, Scheckloth, Corcoran and Page (1958) reported on the course of 85 patients who had been under treatment with potent hypotensive drugs. Thirty-three per cent survived for five years and 26 per cent for six years and these figures were a gain over previous figures for untreated cases. Renal failure was an important cause of death and cerebral haemorrhage was the most common vascular complication and was

found in patients who received treatment before there were extensive vascular changes. Benefits of treatment were most pronounced in retinitis and in heart failure. Improvement in the electrocardiogram was seen in one third of the cases, but reduction in the cardiac size occurred less frequently. Renal function was not influenced and patients with neurological complications did not derive much benefit from treatment. The benefits of treatment therefore seem to be most pronounced on retinitis and heart failure. Secondly, Kincaid-Smith, McMichael and Murphy (1958) reported that the most florid vascular lesions of the young patient under treatment for malignant hypertension regress to a state comparable to that seen in elderly subjects. Accordingly, arteriosclerosis and atheroma were limiting factors in the success of treatment, particularly in older subjects.

Conclusions on the benefits of hypotensive treatment can only be drawn by following up two series of hypertensives broadly comprising treated and untreated cases. To ensure closely matching standards this is best carried out by the same observer over a sufficient period of time. A five-year follow up was made by Leishman (1963) of two such groups; 104 untreated cases over five years and 183 patients maintained for three to five years on hypotensive therapy, 79 on guanethidine for three years and 104 on ganglion blockers for five years. The two groups were studied consecutively since hypotensive treatment cannot ethically be withheld for long periods. All cases exhibited a diastolic pressure over 120 mm on initial examination and were grouped into three severity grades: 120–129 mm Hg, 130–149 mm Hg, and 150+ mm Hg. Although the age and sex distribution of the groups was even, each regime category was only adequately represented in the intermediate severity range i.e. patients with a blood pressure exceeding 130 mm but not showing papilloedema. In the lowest blood pressure grade, distribution of patients was uneven and the total numbers of malignant cases were considered too few to be of any comparative value.

In all three groups, probabilities of death within one, three and five years from all causes, were calculated and a striking feature was the high mortality rate among the two high-severity grades in the first years. These deaths were due mainly to uraemia and strokes and the former caused more deaths among the males, virtually accounting for the differences between death rates of the sexes. There was a spectacular contrast between the treated and untreated cases, and the probable risk of the treated cases dying within five years was found to be within a third and a sixth of the untreated patients. In the lowest range, 120–129 mm Hg diastolic no successfully-treated patient has died in three years of study. Some protection from uraemia and stroke is therefore afforded by hypotensive treatment.

Successful control of blood pressure can prevent serious impairment of renal function and this would strengthen the opinion that some reversal in the progress of arteriolar fibrinoid necrosis protects such patients. Contrary to this, a number of patients died from strokes despite blood pressure reduction, suggesting a different underlying pathology. These reflect a tendency to cerebral arteriosclerosis and atheroma and any blood pressure reduction is unlikely to afford these patients similar protection. The protective effect of blood pressure control in respect of heart failure was also evident since 11 of the untreated group died of heart failure in contrast to only two of the controlled group. These reports suggest that life can be extended and health improved by suitable treatment in selected patients.

Conclusions

The heart and blood vessels have always fascinated man. From ancient times to the present day they have excited the curiosity and interest of medical men. Hypertension is a particularly interesting condition for several reasons. It is a condition which is readily measured. It affects both sexes, all classes of people and all age groups.

It is now even being studied in children (Kass and Zinner 1969).

It can be present through most of a person's life and the treatments available are numerous, sometimes complex and often unnecessary.

It still defies accurate definition and its aetiology and management are open to controversy (Fry 1969).

It is a condition which by its very prevalence in the community must stimulate the interest of family doctors. They, in turn, are particularly well placed to observe and comment upon this interesting condition.

NATURAL HISTORY AND PROGNOSIS

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CONCLUSIONS

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CONCLUDED

Joules

Nutritionists have conventionally used the kilocalorie as the unit of energy. It has recently been proposed that all measurements should be made according to the standard metric system (S.I. or *Systeme Internationale*) and energy is accordingly measured in joules. 1,000 kcal is equivalent to 4.186 million joules or 4.186 megajoules, designated M.J.

Several examining bodies in the U.K. have stated their intention of adopting the S.I. system but, at the time of writing, no authoritative body has made a pronouncement on the change to M.J.

Review of nutrition and food science.
October 1969. No. 17. 3.