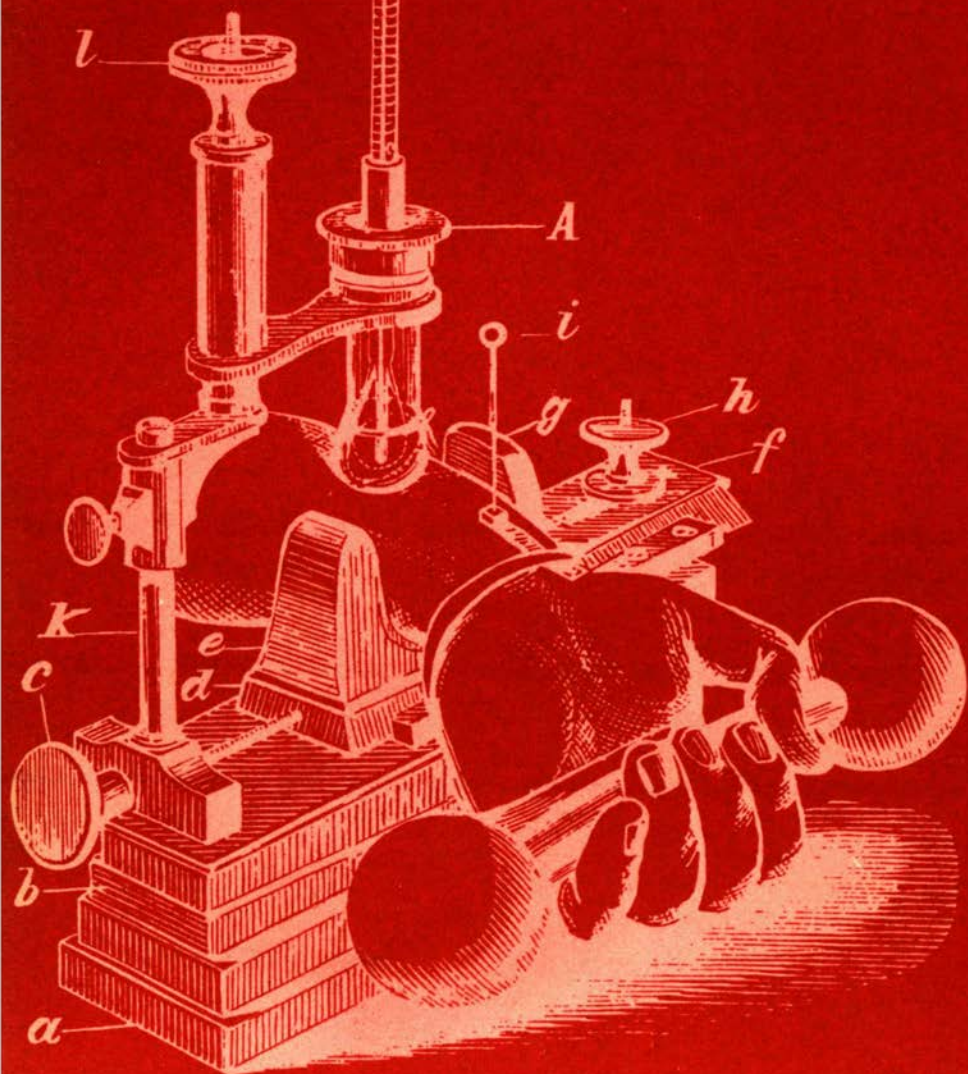


HYPERTENSION

*a suitable case
for treatment?*



No. 39 in a series of papers on current health problems published by the Office of Health Economics. Copies are available at 15p postage free. For previous papers see page 36.

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Printed in England by Hindson Print Group Newcastle upon Tyne.

COVER

Von Basch's sphygmomanometer in use.

From: Von Basch 'Ueber die Messung des Blutdruck am Menschen'.

Zeitschrift für Klinische Medizin.

1881, ii, p. 93.

By courtesy of 'The Wellcome Trustees'.

HYPERTENSION

a suitable case for treatment?



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Glossary

Hypertension

High blood pressure. It is normally shown as the combination of systolic and diastolic pressures, e.g. 180/115 mm Hg may be a typical value for a person with moderate hypertension.

Hypotension

Low blood pressure.

Systolic blood pressure

The arterial pressure at the point when the contraction of the heart forces the pulse wave of blood through the artery from which pressure is being measured.

Diastolic blood pressure

The arterial pressure in between pulse waves when the heart is in 'diastole' or filling with blood ready for the next contraction or 'systole'.

Ganglion blocking drugs

Drugs which prevent the passage of a nerve impulse across the ganglionic synapse. Blockage of the sympathetic ganglia effects a withdrawal of tone and a fall in blood pressure. However blockage of the parasympathetic ganglia causes severe side effects and none of the ganglion blocking drugs are able to discriminate between sympathetic and parasympathetic ganglia.

Adrenergic neurone blocking drugs

These drugs act on the post ganglionic sympathetic adrenergic fibres, thus leaving the parasympathetic nervous system unaffected and obviating many of the resulting side effects from the unselective ganglion blocking drugs.

Diuretics

Drugs which affect the salt and water balance in the body in such a way as to reduce the amount of salt (and thus the amount of water too), thus they provide an alternative means of salt depletion (which reduces blood pressure) without an impracticable low salt diet.

Introduction – the nature and causes of hypertension

Ever since the discovery of the circulation of the blood it has been known that blood is forced around the body under pressure from the contractions of the heart. When the pressure in the arteries is consistently higher than normal (usually through increased resistance from the peripheral vessels), hypertension is said to exist, though a precise and satisfactory definition is impossible because the range of 'normal' pressure can only be arbitrarily defined. Blood pressure not only varies from person to person within the population, but also varies widely within the same person depending for instance on physical exertion, posture, or on mental state at the time of measurement. A person is only considered to be hypertensive if his blood pressure is consistently raised in the absence of any stimuli that may cause a temporary increase in pressure. For this reason it is necessary to measure individuals' blood pressures more than once under standard conditions in order to make any distinction between those who come within an arbitrarily defined range or normality and those who come outside it.

Pickering (1968) classifies hypertension in two ways, by kind and by degree. In the classification by kind, the condition is divided into essential hypertension which is the commonest, where there is no apparent cause, and secondary hypertension. The latter occurs as a manifestation of a known condition such as disease of the kidney, narrowing of the aorta, Cushing's syndrome, toxæmia of pregnancy and various conditions affecting the nervous system. All of these are associated with raised systolic and diastolic pressure levels. The systolic pressure alone may be raised if the stroke output of the heart is increased as in various heart conditions, Paget's disease of the bones, fever and pregnancy. The systolic pressure may also be raised alone through reduced elasticity of the large arteries which often accompanies old age. In the classification by degree, a distinction is drawn between the benign phase where hypertension is generally asymptomatic and the malignant phase where cardiovascular and/or renal complications have developed.

There is some controversy over whether hypertension per se can be considered as a diseased state. Studies of blood pressure levels in the population at large have demonstrated that values are distributed continuously throughout the population. There is

no level at which the population can be divided into those with 'abnormal' blood pressure and 'normal' blood pressure. Nor do life insurance data provide any clear cut dividing line between normality and abnormality (Society of Actuaries 1959) (Dublin *et al* 1949). They indicate that the risk of mortality in all age groups increases with each step in the elevation of either systolic or diastolic pressure. This holds true even at the lower levels of blood pressure, 120/80 which would not be considered 'abnormal' by any clinician. Pickering (1968) wrote 'if we choose to call essential hypertension a disease, it is a disease of a kind hitherto unrecognised by medicine, a disease characterised by a quantitative, not a qualitative, deviation from the norm'. Many doctors are in fact disinclined to consider essential hypertension as a diseased state partly because such a large proportion of the oldest age groups – the majority among women – could be considered to have elevated blood pressure and thus, by implication, to be 'abnormal'. To a considerable extent the issue is a semantic one, incidental to the central point that the higher the blood pressure the greater is the predisposition to morbidity and mortality.

Excess morbidity and mortality among people with high blood pressure comes primarily in the form of cardiovascular and/or renal diseases. High blood pressure itself is not the immediate antecedent of death. However it has been found to be more common among those who have developed coronary heart disease and it is also known to be causally connected with an increased risk of stroke through damage to the vulnerable cerebral vessels. Hypertension leads rapidly to death if the accelerated or malignant phase is reached and no treatment is given. When pressure is sustained at a high level cardiac failure can develop. In addition, very high pressure causes damage to the kidneys which in turn results in further elevation of pressure and starts a cycle which leads eventually to kidney failure and death. Occasionally, patients go spontaneously from a malignant phase to a benign phase though normally, once the cycle of malignancy has started, the prognosis is very poor in the absence of treatment.

Relatively little is known of the causes of essential hypertension and thus little is known of the possibilities of prevention. Studies in developed countries have shown that mean blood pressures in a population rise with increasing age and individuals' pressures become more widely dispersed about the mean (Hamilton *et al* 1954) (Master *et al* 1950) (Miall and Oldham 1963). In early life average male pressures tend to be higher than female pressures. However, from the age of about 45 the mean pressure of the female population becomes higher than the mean pressure among males. To an extent this reflects a selective removal of males with elevated

pressure from the population. They are much more likely than females to develop and die from coronary heart disease. The higher the level of blood pressure the greater is the risk of death from this cause. (Dawber and Kannel 1961.)

Hereditary, dietary, environmental and racial factors have all been found to have a bearing on blood pressure levels but there is not yet any strong evidence identifying one or two crucial factors.¹ In the present state of knowledge, therefore, there is little of practical value that can be done to prevent the development of essential hypertension except perhaps measures to reduce the prevalence of obesity in the population. Blood pressure is known to be positively correlated with obesity (e.g. Miall *et al* 1968) so there may be considerable potential benefit to be derived from reducing the 60 per cent of middle aged people who are ten per cent or more overweight, if not much reason to believe that such a reduction could actually be achieved in the short or medium term.

1 There is some evidence that race may have a bearing on pressure levels and it has also been suggested that high levels of dietary salt intake may lead to hypertension, though no conclusions can be drawn in the present state of knowledge. There is an established correlation between blood pressures of first degree relatives though the comparative importance of hereditary and environmental factors is uncertain.

Treatment of hypertension— past and present

The diagnosis and treatment of hypertension can be traced back to the 1820s when Richard Bright noted, on autopsy, abnormalities in the hearts of some patients who had died with chronic renal disease. He suggested, as one of the possible explanations, that there was an increased resistance to flow in the blood vessels. However, no instrument capable of measuring blood pressure in man² was developed until the middle of the 19th century when Karl Vierordt introduced the principle of measuring the amount of counter pressure necessary to obliterate the pulsations in a peripheral artery in the arm. A number of devices using this principle were developed and in 1896 in Italy, Riva-Rocci described an instrument which was in all its essentials the same as a modern sphygmomanometer. The main difference was the size of the cuff. Riva-Rocci's cuff was only 4.5 cm wide but since this was found to give readings on the high side, 12 cm was eventually accepted as the standard width.

Up to this time only the systolic pressure (the pressure of the pulse of blood) was commonly recorded, but in 1905 a Russian named Korotkoff suggested a method of measuring the different phases of blood pressure which quickly gained acceptance and is now generally recognised as standard. The cuff is tied around the patient's arm and inflated to a pressure well over that necessary to obliterate the pulsations of the peripheral artery. As the cuff is deflated and the column of mercury drops, the first characteristic tapping sound is heard. This begins at the systolic blood pressure level and is due to the return of the pulse wave to the artery hitherto collapsed by the pressure of the cuff. A murmur develops at phase two but disappears at phase three when the initial pulse sound becomes louder. At the fourth phase the sound muffles. In Britain, this is generally taken as the diastolic level, the level of pressure where blood flow can take place through the artery throughout the entire cardiac cycle. The sound then disappears altogether. This is the fifth phase in which there is no obstruction at all to the blood flow in the diastole. It is this point which is generally taken to represent the diastolic pressure in the U.S.A. and some other countries. Thus the measurement of blood pressure

2 In the 1730s, a clergyman named Stephen Hales first recorded a direct measurement of blood pressure by tying a long tube to an artery in a mare's leg and watching the height to which the column of blood rose.

has been a practical possibility throughout the twentieth century, and in addition the main features of diseases associated with large changes in pressure levels were described and defined as early as the beginning of the twentieth century.

However, despite the recognition of the implications of high blood pressure there was little that could be offered as a safe and effective means of lowering pressure. The only drugs available at the time of the first war were the nitrites but their transitory action was of no value for long term medication. In the inter-war years, the thiocyanates were widely used as antihypertensives but they were not very effective, the side effects were severe, and they soon lost favour with doctors. During these years when medical intervention was ineffective, surgical procedures were sometimes successful in lowering blood pressure. One such procedure was the removal of a diseased kidney which was responsible for the raised blood pressure. But the risks were high, the success rate only about a quarter according to one review of published cases, and the procedure was only relevant to a very small minority of hypertensive patients. Great controversy was aroused over another surgical procedure intended to reduce pressure levels, surgical sympathectomy. This procedure developed to the stage where the whole of the thoraco-lumbar sympathetic chain was excised, but results were generally disappointing.

Attempts were also made to manage hypertension through restriction of salt intake. This did have some effect in lowering blood pressure, occasionally by significant amounts. The popularity of this form of treatment reached its peak in the 1940s but few were able to maintain the virtually saltless dietary regime over a long period of time.

In 1949 the ganglion blocking drugs were first introduced and effective treatment of severe hypertension first became possible. Special clinics for treatment of hypertensives were founded in various parts of the world and reports from these indicated that the prognosis of malignant hypertension was much improved with ganglion blocking drugs. However, the severity of side effects and the need for strict control over their use meant that they were only a practicable form of treatment where complications had already set in and where life was immediately threatened. They could not be conceived as a preventive regime for the vast majority of people whose hypertension was as yet asymptomatic.

Two separate pharmacological developments in the late fifties and early sixties had the effect of extending the limit of effective and practical treatment to include asymptomatic hypertensives for the first time. First, in the early 1960s guanethidine and methyldopa, the first of the adrenergic neurone blocking drugs

were introduced. They were followed by others such as bethamidine, guanoxan and debrisoquine. By their selective action they avoid many of the unpleasant side effects associated with the ganglion blocking drugs. The regime is such that lifelong treatment can be contemplated with reasonable equanimity by a hypertensive whose alternative is a markedly more risky, if asymptomatic, existence. However, some important side effects still remain, particularly the tendency to a sudden drop in pressure on standing up. This manifests itself as a feeling of faintness or weakness. This drop in pressure also occurs after exertion.

The second pharmacological development may eventually be even more important in expanding the limits of effective, practicable treatment of asymptomatic hypertension. This was the introduction of the first potent oral diuretic in 1957. Modern diuretics have few side effects, at least in the short term, and they have the great advantage that they do not lead to excessive falls in blood pressure on standing up. They offer the possibility of lowering pressures through salt depletion without an impracticable low salt diet. Over the past ten years a wide range of diuretics has been developed, recently including frusemide and ethacrynic acid. Blood pressure reductions are not generally sufficient to treat patients with very high pressures with a diuretic alone. However, a diuretic alone, or together with reserpine, does have a sufficiently powerful hypotensive effect for the much larger numbers of people with relatively mild or moderate hypertension. Reserpine itself was isolated from crude rauwolfia in 1952 but because it can lead to severe depression its value by itself was very limited. Since the late fifties, however, a combination of a diuretic and a small amount of reserpine has been found to be very potent. The effect of the two together on blood pressure is greater than either alone.

Prevalence of various degrees of hypertension

The reported prevalence of any disease depends on the definition of the condition and on the diagnostic criteria used. In the case of high blood pressure measurement is possible in quantitative terms even if the cut off point of normality is totally arbitrary. A complicating factor which does not cause problems in measuring the prevalence of most chronic conditions is the variability of blood pressure readings at different times and circumstances in the

same person. It has been shown for instance that when pressures are repeatedly taken over time they tend to drop as the patient becomes more accustomed to the procedure. (Pickering *et al* 1961.) This could cause an over-estimate of the amount of hypertension in the community. For this reason more than one casual reading is desirable in order to reduce error. Another source of error is the observer. Wilcox (1961) showed a group of nurses a film of mercury falling in a sphygmomanometer together with a tape of the characteristic sounds. She found wide variations in the nurses' estimations of the single level of pressure shown in the film. Rose *et al* (1964) found similar variations among a group of doctors. There may also be bias from unconscious 'digit preference'. Observers may tend to record a borderline pressure reading on the low side of any arbitrary level of normality, as for instance when success or failure in a life insurance examination is at stake.

Most of these sources of error can be largely overcome with training or with the use of sophisticated apparatus³ but the most potent source of error, the random variability of the patient's blood pressure itself, cannot be overcome except with repeated readings.

There have been two large studies which can be used to provide estimates of the prevalence of hypertension in Britain. Miall and Oldham's (1963) survey used a Welsh population. It is probably the more reliable from the point of view of minimising error. The other survey, (Hamilton *et al* 1954) measured the pressures of

Table 1 *Prevalence of hypertension among adults at different ages*

<i>Sex</i>	<i>Age</i>	<i>Percentage with diastolic pressure of 115 or more</i>	<i>Percentage with diastolic pressure of 95 or more</i>
Male	35-	% .5	% 5.3
	45-	1.2	12.7
	55-	7.1	33.0
	65-74	6.8	30.2
Female	35-	1.7	12.4
	45-	4.9	20.8
	55-	7.7	36.4
	65-74	9.7	66.0

Source Derived from data from Hamilton, Pickering, Roberts and Sowry (1954)

³ Other sources of error include possible over-estimation of pressures in persons with fat arms and errors may be built into the instrument itself if the cuff is the wrong size.

relatives of hospital out-patients in the Greater London Area. It was based on single readings. The results of the two studies are broadly similar to each other and also to a much larger Norwegian study (Humerfelt and Wedervang 1957). The distribution of diastolic blood pressure levels from Hamilton *et al* (1954) survey is shown in Table 1.

The table gives prevalence rates at two arbitrary cut off points. If a diastolic pressure of 95 is accepted as the border between hypertension and normotension then about one-quarter of the 35-74 year old population would be termed hypertensives. If, on the other hand, the critical level was set at a diastolic pressure of 115 then five per cent of the same population would come into that category.

The potential benefits of reducing high blood pressure

The risks of untreated high blood pressure are very well documented in quantitative terms. American Insurance companies have since the inter-war years required a blood pressure reading from their prospective clients, and figures derived from their analysis of mortality rates according to initial blood pressure, based on a single reading, are shown in Table 2.

These figures from the 'Build and Blood Pressure' study show that male mortality ratios (i.e. the ratio of actual mortality over the expected mortality for a healthy man) increase with each step upwards in both systolic and diastolic pressure. At an initial pressure of 160/100, which would not be considered seriously elevated by many clinicians, expectation of death over the period of study was about four times that associated with an initial pressure of 120/80. However, the significance of figures like these seem not to have percolated through the whole profession yet, perhaps because effective control of blood pressure has only recently become feasible. Attitudes formed when no effective treatment was available may take a long time to adapt to new circumstances. Perhaps the next generation of doctors will be more enthusiastic about the potential benefits from controlling mild or moderate hypertension, especially if further research can isolate specific groups of hypertensives who are particularly at risk.

Another perhaps clearer way of expressing hypertensives' excess risk of mortality is by analysis of life expectation. Figure 1 gives data from the experience of 26 American insurance companies over

Table 2 *Mortality according to variations in initial blood pressure 'Build and Blood Pressure' study. Society of Actuaries 1959 Standard and sub standard issues combined. Mortality Ratios.*

a) **Men aged 15-39 at issue**

Systolic BP mm Hg	Diastolic BP mm Hg				
	80	85	90	95	100
120	95	105	115	—	—
132	105	125	150	190	—
142	130	155	185	225	275
152	160	185	225	275	325

b) **Men aged 40-69 at issue**

Systolic BP mm Hg	Diastolic BP mm Hg				
	80	85	90	95	100
120	80	95	110	130	—
132	105	115	130	150	175
142	130	145	165	190	215
152	160	180	200	225	250
162	195	215	235	260	300

Note For all practical purposes, the population represented by insurance company data was an untreated one. This is because people with a pressure over 160/100 were excluded from life insurance altogether and probably no one with pressures below that level would have been given effective treatment over the years to which the figures relate.

Table 3 *Male Mortality ratios for certain causes*

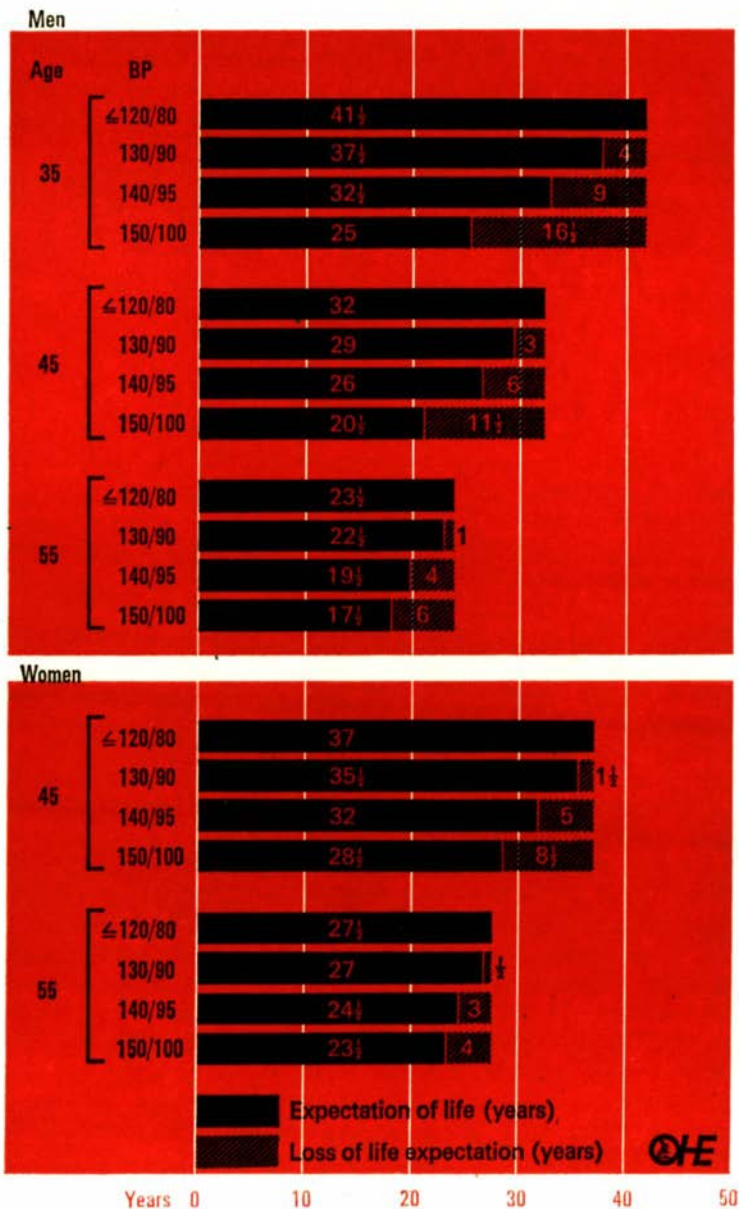
Blood pressure mm Hg	Mortality ratio. (Standard risk—100)				
	Heart and circulatory diseases	Strokes	Nephritis	Diabetes	Digestive disease
138/83 (mildly to elevated) 147/92	180	243	273	158	149
148/93 (moderately to elevated) 177/102	282	616	472	N.A.	193

Source Build and Blood Pressure study. Society of Actuaries 1959

the years 1935-1954. It shows that both men, and to a lesser extent women (with their lower overall mortality rates), suffer a significantly diminished expectation of life. In this experience a man aged 45 with a blood pressure of 150/100 could expect to live 11½ years less than a similar man with a pressure of 120/80 or

Figure 1 *Expectation of life associated with various initial blood pressure levels.*

Source Blood Pressure: Insurance experience and its implications, 1961. New York. Metropolitan Life Insurance Company.



below. This is despite the probability that the vast majority of those to whom life insurance was issued were symptomless at the time of measurement.

Table 3 shows, for all age groups, the conditions which were identified by the 'Build and Blood Pressure' study as contributing to excess mortality among those with elevated pressure.

The two important categories in Table 3 are 'Heart and Circulatory Diseases' and 'Strokes'.⁴ Deaths from stroke are shown to be six times as likely to occur among men with 'moderately elevated' blood pressure than men with normal blood pressure, i.e. 120/80. In terms of absolute numbers, however, diseases of the heart and circulatory system cause more excess deaths because these diseases, particularly coronary heart disease, are much more common than strokes.

The findings are consistent with evidence from other studies which have looked at the excess risk of morbidity associated with high blood pressure as well as mortality. In the Framingham study, (Kannel *et al* 1970) which followed the medical history of 5,000 middle-aged men and women in Framingham, Massachusetts, it was found that morbidity from atherothrombotic brain infarction among initially asymptomatic persons with initial pressures over 160/95 was four times higher than among those with pressures under 140/90. Another publication from the Framingham study (Dawber and Kannel, 1961) demonstrated the increased risk of morbidity from coronary heart disease with each increase in pressure, Table 4. Coronary heart disease is by far the commonest cause of death among middle-aged males in western countries and accounted for the majority of excess deaths in the range of elevations analysed by the build and blood pressure study.

Table 4 *Blood pressure and risk of morbidity from coronary heart disease*

	<i>Systolic blood pressure mm Hg</i>	<i>Relative risk of coronary heart disease</i>
less than	120	28
	120-139	80
	140-159	96
	160-179	166
greater than	179	233

Source Dawber and Kannel 1961

4 Of the others, nephritis is a relatively unimportant cause of death in itself though it can be a cause of hypertension. The excess mortality rates shown against diabetes and digestive disease could have been due to the association of hypertensives with obesity.

Thus there is overwhelming evidence that morbidity and mortality risks increase in a stepwise progression with elevations in blood pressure. But this of itself does not necessarily mean that measures to reduce blood pressure levels before any symptoms develop result in a reduction of the excess morbidity and mortality. What evidence is there that treatment of high blood pressure is of value to the patient?

The benefits of treatment in severe or accelerated hypertension with signs of renal damage, left ventricular failure and retinal damage have not been in dispute since the development of the first effective hypotensives. The prognosis is much improved. Deaths from strokes, renal failure and heart failure are considerably reduced though various studies (e.g. Breckenridge *et al* 1970) have not demonstrated any clear reduction in mortality from coronary heart disease when treated at an advanced stage.

The benefits of treatment of asymptomatic high blood pressure, or hypertension with minimal signs or symptoms, have been somewhat more controversial. However, in the short time since effective antihypertensives have been available, two controlled trials have clearly demonstrated the value of treating high levels of hypertension without severe symptoms, among men at least. The results of the trials are summarised in Table 5. The Veterans' Administration (1967) trial in America measured the results of treatment with thiazide, reserpine and hydrallazine against treatment with a placebo in 143 middle-aged men (without serious symptoms such as haemorrhages or exudates in the optic fundi or dissecting aneurysm) who had diastolic pressures between 115 and 129 (mean 121). Hamilton *et al's* (1964) trial in the U.K. concerned 61 middle-aged persons of both sexes, (similarly without complications of hypertension, and without evidence of arterial disease) who had diastolic pressures consistently over

Table 5 *Results of antihypertensive therapy*

	<i>Hamilton et al (middle-aged persons) 2-6 years follow up</i>		<i>Veterans' administration (middle-aged men) up to 2½ years follow up</i>	
	<i>Control</i>	<i>Treatment</i>	<i>Control</i>	<i>Treatment</i>
No. of patients	31	30	70	73
Mean pre-treatment diastolic pressure	129	136	121	121
Total No. developing severe complications	16	5	27	2
No. developing stroke	7	3	4	1
No. of deaths	2	2	4	0

Source Hamilton *et al* (1964). Veterans' Administration (1967)

110 (mean 136). Various combinations of drugs were used including a diuretic, a ganglion blocking drug and methyldopa when it became available.

Treatment clearly reduces morbidity and mortality among men who are asymptomatic, or who are at least without serious symptoms, with diastolic pressures over 110 or 115. The Veterans' Administration trial gives better results than Hamilton's but this is largely because the former included males only and the latter had a majority of females.⁵ Hamilton's is the only controlled trial which has measured the effect of antihypertensive therapy on women without serious symptoms but with highly elevated pressures. The findings were inconclusive. The results of the treatment group of women were not significantly better than the results of the controls though the data suggested that this was not due to failure to benefit from lower pressure, but failure of therapy adequately to control pressure among women. Hamilton pointed out that when pressure was well controlled then results were good.

Recently the Veterans' Administration (1970) has published the results of another part of its trial which indicates that considerable benefits are also to be derived from treatment among men without serious symptoms who have only relatively mild or moderately elevated blood pressures (Table 6). The trial covered 380 middle-aged men with diastolic pressures of between 90 and 114. In the untreated control group there were 19 deaths and a total of 56 assessable morbid events. In the treatment group the corresponding numbers were 8 and 22. These results suggest that the threshold at which significant benefit is derived from antihypertensive therapy is very much lower than has normally been accepted by clinicians as grounds for routine treatment. Benefits tended to increase over time of follow up and among those observed for 5 years the cumulative incidence of morbid events was 55 per cent among the controls and 18 per cent among the treatment group. Thus the 'effectiveness' of treatment could be calculated at 67 per cent in that the treatment group had 67 per cent fewer morbid episodes than the control group. The higher the initial blood pressure level the greater was the 'effectiveness' of treatment. Thus 75 per cent of morbid episodes were prevented by treatment among those with initial diastolic pressures of 105-114 as compared with 35 per cent among those with initial diastolic pressures of between 90 and 104 mm Hg. Even in the lower range of pressure levels the benefits were quite large though less confidence can be attached to the latter figure because of the

5 In Hamilton's male group there were no complications or strokes among the ten treated and eight complications among the twelve on placebos. All of the complications and strokes in the treatment group were among females.

Table 6 Results of antihypertensive therapy among men with initial diastolic blood pressure levels of 90–114 mm Hg

	<i>Veterans' Administration (middle-aged men) mean duration of follow up—3.3 years</i>	
	<i>Control</i>	<i>Treatment</i>
No. of patients	194	186
Mean pre-treatment diastolic pressure	101	100
Total morbid events*	56	22
Deaths from cerebral haemorrhage, subarachnoid haemorrhage and dissecting aneurysm	5	0
Other related deaths	14	8
Total related deaths†	19	8

Source Veterans' Administration 1970

* 20 men from the control group (none from the treatment group) were taken out of the trial before morbid events had developed because their diastolic pressures went over 115 mm Hg.

† There were two deaths each in the control group and treatment group from causes unrelated to hypertension.

relatively small number of morbid events upon which the calculation was based in the 90–104 range. This reservation applies with even greater force to the lower end of the 90–104 range itself.

The trial confirmed the view that benefits were mainly derived from prevention of such complications as cerebral and subarachnoid haemorrhage. The incidence of morbid events from atherosclerotic conditions such as coronary heart disease did not appear to be significantly different between the treatment and control groups though the authors pointed out that atherosclerosis develops over a long period of time and there is the possibility that reduction of blood pressure at a much earlier stage might reduce the incidence of this sort of disease.

However, some reservations must be made about the trials. The populations of the Veterans' Administration trials may not be typical either in susceptibility to complications or tolerance of treatment. They were men who were already in contact with the health services, and the results for men picked up through any random screening programme may not be the same. Also, the subjects of the experiments were certainly better motivated than most to accept the undesirable side effects of treatment. 'Unreliable' and 'unco-operative' patients were excluded from the trial before it began.

Furthermore, beneficial results cannot be inferred for all sex and age groups from the results among middle-aged men. It has been noted that Hamilton's results in his female group were equivocal and there is no information from controlled trials of the benefits of treatment in the older age groups and in young men and women. This is an important set of priority areas for further epidemiological work. A controlled trial is at present taking place at St. Thomas's Hospital and Cardiff, looking at patients of both sexes with diastolic pressures of 100 or more.

Mortality rates in England and Wales

Given the effectiveness of antihypertensive therapy it might be expected that national mortality rates would reflect the breakthrough in treatment of severe hypertension in the early 1950s and the extension of effective and acceptable therapy to less severe hypertensives in the sixties. Figures 2 to 4 show mortality rates in England and Wales for the major conditions associated with hypertension over the years 1940 to 1968. National recorded mortality rates are notoriously susceptible to changes in diagnostic criteria and changes in classification which come with the development of medicine. Time series must therefore be treated with great care, but there are some points which stand out quite clearly from the graphs.

Figure 2 shows that mortality rates for hypertensive disease, which had been rising rapidly before 1950, (and this was probably a reflection of increasing recognition of the condition) started to fall away rapidly after 1950. The pattern is complicated by the sixth revision of the International Classification of Diseases and Deaths which became effective in 1950. However, it conforms to the expectation that these deaths, mainly from advanced malignant hypertension, would be reduced immediately by the availability of therapy capable of reversing the malignant phase.

Figure 3 shows mortality rates from cerebrovascular disease (strokes) between 1940 and 1968. Reductions might be expected to coincide with the introduction of the first effective therapy for severe hypertension around 1950 and further reductions in mortality might be expected as more and more people benefited from the extension of effective and acceptable therapy. Actual mortality rates do not in fact precisely coincide with pharmacological advances, but a gradual reduction is evident from the mid nineteen fifties onwards. Dramatic reductions in death rates

Figure 2 Hypertensive diseases, including hypertensive heart disease. (Pre 1950 'Essential Hypertension'). Death rates per million living by age and sex. England and Wales 1940-1968.

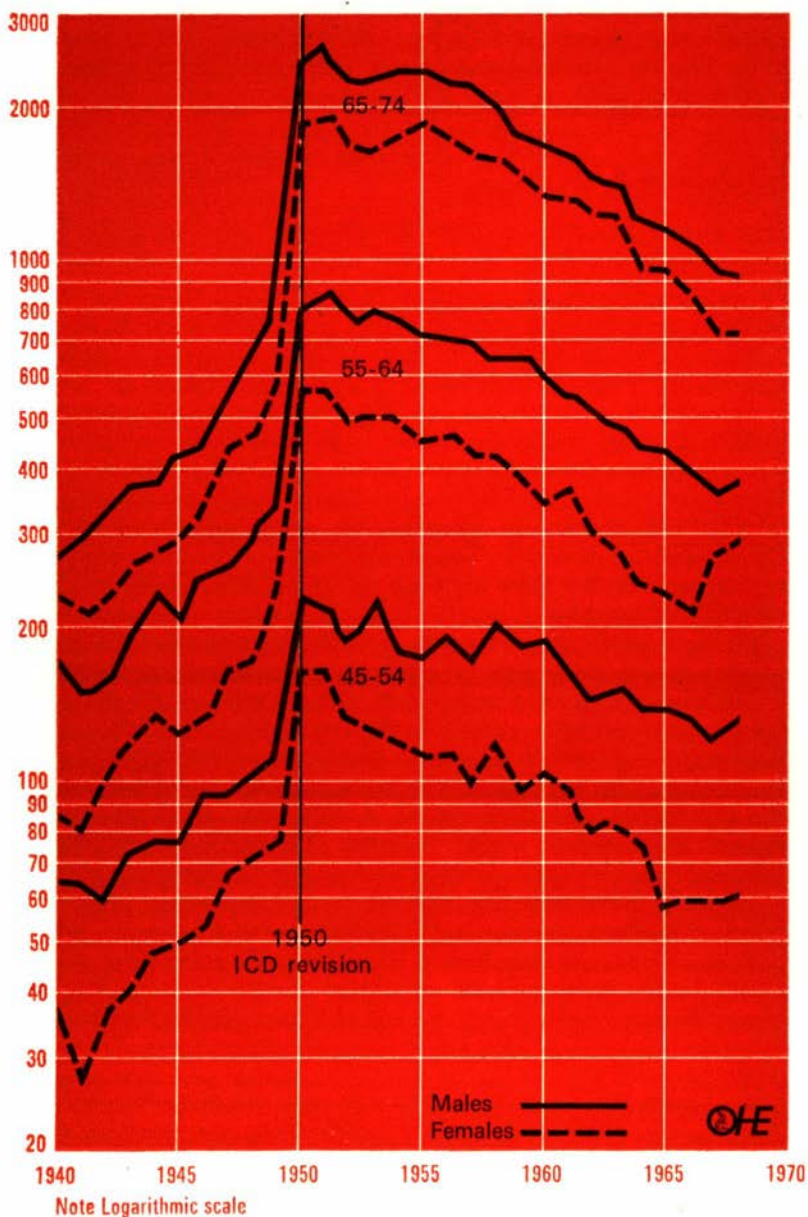


Figure 3 Cerebrovascular disease. Death rates per million living by age and sex. England and Wales 1960-1968.



over the past 30 years have followed new means of prevention and treatment in a number of infectious diseases, but it is only in the case of high blood pressure and its consequences that benefits of new forms of treatment have as yet been clearly reflected in national mortality rates for a degenerative disease associated with the process of ageing.

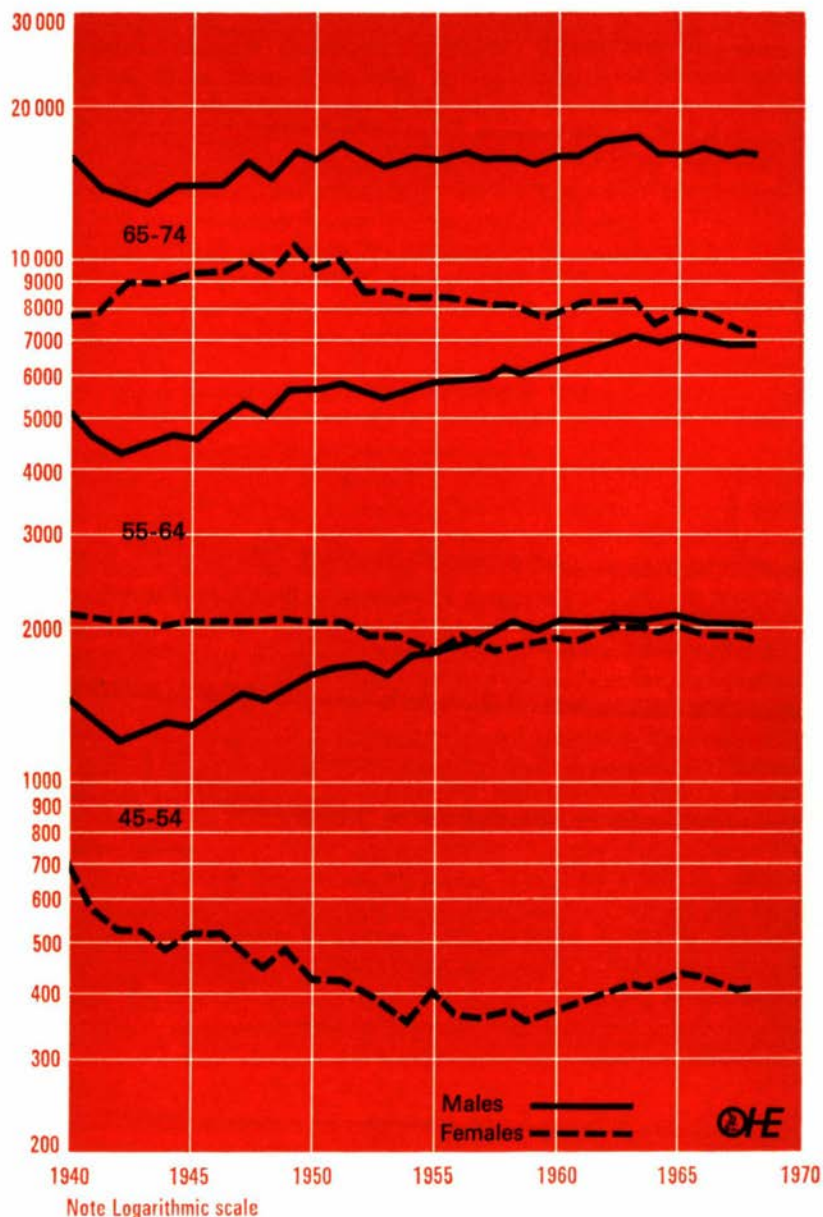
There may appear, at a superficial level, to be some inconsistency between national mortality rates and the results of the controlled trials. The controlled trials, and the insurance companies' mortality data as well, suggested that males would benefit more than females from blood pressure reduction. In contrast, national female death rates from stroke in the 55-74 age range have declined more rapidly than male death rates, Figure 3. The apparent inconsistency can be explained in two ways. First, middle-aged and elderly women consult their doctors for essential benign hypertension about twice as much as middle-aged men. There is the presumption, therefore (since this approximately reflects differential rates of hypertension) that more women than men are on hypertensive therapy. The second point relates to the poor response of women in Hamilton *et al* (1964) trial. These women had very highly elevated blood pressures. If antihypertensive therapy is better able to control relatively moderate or mild hypertension among women then they may benefit as much as men, as the national mortality data suggest. The answer to this will only be found through more controlled trials among women with mild or moderate hypertension.

Little can be deduced from the pattern of mortality rates from ischaemic heart disease. It is known that blood pressure is only one factor, albeit an important one, in the causation of ischaemic heart disease. It has been noted that blood pressure reduction may not reduce mortality from coronary heart disease and the pattern of Figure 4 provides no evidence to the contrary.

The cost of hypertension

Table 6 gives estimates of the NHS resource cost in 1969 of various major diseases associated with hypertension. No figure representing the total cost of high blood pressure and its complications can be given since the amount of morbidity attributable to high blood pressure (or the amount which could be prevented by blood pressure reduction) cannot be disentangled from the total.

Figure 4 *Ischaemic heart disease. Death rates per million living by age and sex. England and Wales 1940-1968.*



Hypertensive disease itself cost the NHS resources worth £20.6 million or 1.6 per cent of those sectors of the NHS where costs could be broken down by disease group. Over half of this, £11.7 million, was spent on antihypertensive drugs. This accounts for about 6 per cent of the total cost of the pharmaceutical service, a proportion which has doubled over 10 years. In comparison the cost of hospital in-patient treatment for hypertensive disease was estimated to be £5.4 million in 1969. In contrast to the other sectors of the health service, hospital resources devoted to hypertension have been decreasing. Between 1958 and 1967 there was a fall in the age and sex adjusted admission rate to hospital in England and Wales and the total number of bed days attributable to hypertensive disease fell by 38 per cent as compared with the 1 per cent fall in bed days for all causes of admission to hospital. This probably reflects better control of the small proportion of hypertensives entering the accelerated or malignant phase.

The cost of strokes in NHS resources was estimated at £41.9 million in 1969 or 3.2 per cent of sectors analysed. Nearly all of it was spent in hospital in-patient treatment. Only a proportion, though probably a large one, is attributable to high blood pressure in the sense that each step up in blood pressure increases the risk of morbidity from strokes. For strokes, however, there has been no reduction in bed usage despite reductions in mortality. In-patient bed days rose by 46 per cent between 1958 and 1967, largely because average duration of stay per case has remained constant instead of dropping steadily as it has for most other causes of admission.

In the case of ischaemic heart disease too, only a proportion of costs are attributable to high blood pressure which is only one of a number of predisposing factors. Ischaemic heart disease consumed NHS resources estimated at £27.5 million or 2.1 per cent of the cost of the sectors analysed in 1969 despite being commoner than strokes and despite accounting for rather more hospital admissions too. In-patient bed days which accounted for most of the cost rose by 53 per cent between 1958 and 1967.

Though the data are crude the figures suggest that the cost of drug therapy for an increasing number of hypertensives in the past may have been partly compensated by a release of hospital resources due to a reduction in the work load imposed by severe hypertensive disease on in-patient departments. There is not yet, however, any evidence of a massive payoff internal to the NHS similar for instance to the payoff from tuberculosis chemotherapy in the immediate post war years when long and ineffective treatment in sanatoria was rendered unnecessary and large amounts of health service resources were released.

Table 7 *The cost of various conditions associated with hypertension to the National Health Service, UK 1969*

Health Service Sector	Cost attributed to			
	Hypertensive disease ICD 400-404 £ million	Ischaemic heart disease ICD 410-414 £ million	Cerebrovascular disease ICD 430-438 (strokes) £ million	All diseases ICD 000-999 £ million
Hospital Service in-patient cost (current)	5.4	21.9	40.4	842
General Practice	3.5	2.4	0.8	147
Pharmaceutical Service	11.7	3.2	0.7	189
Dental and Ophthalmic Services	nil	nil	nil	115
Sub-Total	20.6	27.5	41.9	1293
Other services, including local authority health services, hospital out-patient costs, hospital capital expenditure and miscellaneous. (No basis for allocation of cost).	NA	NA	NA	587

Source OHE estimates

Indirect costs

The second group of costs are the indirect ones, including sickness absence costs. In 1968/69, a total of 7.5 million days of certified sickness absence were attributed to hypertension, 2.3 per cent of all days of absence. The estimated cost of this absence to the individuals concerned and to the payers of sickness benefits was £34 million⁶ in 1969 terms. The economic cost of absence attributed to strokes and ischaemic heart disease was £16 million and £88 million respectively though in both cases only a proportion is attributable to high blood pressure, relatively small in the case of ischaemic heart disease and larger in the case of strokes.

⁶ The conceptual difficulty of placing a realistic economic value on each working day lost has been discussed elsewhere (OHE 1971). In short, to the extent that absence from high blood pressure and associated diseases tends towards the long term, chronic end of the spectrum it is not unrealistic to value each day lost at the national average gross pay level, i.e. £5 per man day and £3 per woman day in 1969.

Figure 5 Days of certified sickness absence expressed as a rate per 1000 insured persons. Various causes. 1954/5 to 1960/61 age standardised to 1951 population. 1962/3 to 1966/7 age standardised to 1962/3 population.

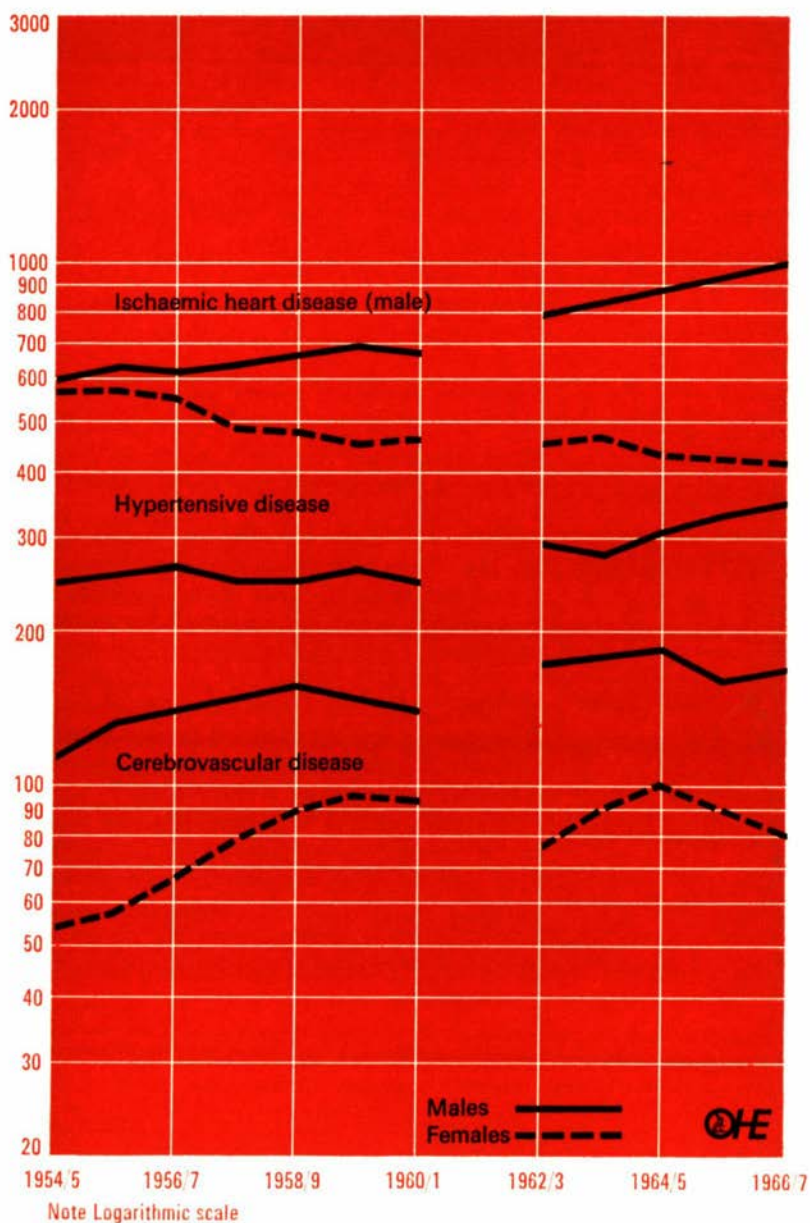


Figure 5 shows changes in age adjusted rates of working days lost through sickness absence attributed to hypertension and major associated diseases. Time series of absence certificates are extremely difficult to interpret. A great many variables are important in determining recorded levels of sickness absence. However, one point is worth commenting upon. For hypertensive disease both mortality rates and usage of hospital beds have declined following the advent of effective antihypertensive therapy, but no such reduction has been evident in days of sickness absence attributed to hypertension among men. In fact, in recent years male rates have been increasing more rapidly than corresponding rates for all causes of sickness absence. It is possible that this may be a result, in part at least, of the extension of antihypertensive treatment to a larger number of asymptomatic men. More likely, since only about one to two per cent of the middle-aged male population is actually on antihypertensive therapy, increased absence could result from an increased awareness of the dangers of high blood pressure among both the medical profession and their patients. This could have the effect of altering the threshold level at which morbidity attributed to treated or untreated high blood pressure is translated into spells of sickness absence. High blood pressure may be a useful tag for certifying absence due to various primary causes, social or medical. In this case any expectation of an economic payoff through reducing the amount of morbidity in the community caused by high blood pressure is unlikely to be realised. An assault on asymptomatic hypertension through a widespread screening programme may well create more certified absence than it prevents.

Personal costs

The third and final group of costs are the intangible ones of personal hardship and disability to the sick person himself. In the case of high blood pressure most of the people who might benefit from treatment do not have serious symptoms and therefore the cost of hardship and disability is largely composed of the risk of later morbidity and mortality which have been described already. An additional cost which has also been alluded to is the exclusion of hypertensives with a pressure of about 160/115 from any form of life insurance. This affects about 1 per cent of men between the ages of 45 and 54 and about 7 per cent of men between 55 and 64 (Table 1), but a weighted premium is likely in a much larger proportion.

An indicator of the personal as well as the financial costs of severe health impairment can be derived from national sickness

absence statistics. They analyse the amount of long term disability in the working population. Apart from essential benign hypertension itself most of the certified absence due to the major complications is for persons off sick for 6 months or more. Very few of these persons will have retained their jobs after that period of time and many are unlikely to work again. In June 1967, 7,200 men and 3,700 women in Britain had been absent from work for over 6 months due to hypertensive disease. For cerebrovascular disease the figures were 6,400 and 900 respectively and for ischaemic heart disease they were 31,200 and 4,400. The long term sick and their families are an important group among the 2 million people at present living on resources below the level which the Supplementary Benefits Commission considers necessary for reasonable subsistence. If the primary objective of the health services is the extension of life which is both healthy and socially active then the prevention of some of this long term incapacity, which is disastrous for the individual and his family in both personal and financial terms, would be likely to represent very much better value for money than most of the activities of the National Health Service.

Another aspect of the personal costs of hypertension is the effect on the family of the premature death of the husband. Using the American insurance data (Figure 1) as a guide, a man of 45 with a blood pressure of 150/100 could only expect to live for $20\frac{1}{2}$ years as compared with the 32 years of life expectancy of a similar man with a pressure of 120/80 or below. This means an expectation of $11\frac{1}{2}$ more years of widowhood for the wife, with perhaps disastrous consequences if the husband dies well before retirement without making adequate provision for his family.

Screening for hypertension

At present, hypertensives are not sought out and treated on a systematic basis. There are wide variations in the action that different doctors take in similar cases. Most general practitioners would prefer not to treat asymptomatic hypertensives in the general population as a matter of routine unless their pressures were very high. What, therefore, are the merits of seeking out and treating hypertensives in the general population as a matter of routine, and more critically, what is the level of blood pressure which should be taken as a guideline to the necessarily arbitrary division between hypertension and normotension?

In view of the foregoing discussion on the costs of hypertension and associated conditions it would not be possible to justify a national or regional screening programme on the grounds that treatment, though expensive, would result in the release of compensating resources in other sectors of the NHS. The cost of a programme can best be considered and evaluated in the light of its contribution to the reduction of mortality and incapacitating morbidity alone.

Costs

The cost of a screening programme in National Health Service resources would be composed of an increased work-load on doctors and ancillary staff, increased data handling facilities, some increase in equipment and facilities, and drugs. Most of the extra cost would be generated by therapy which at present accounts for the larger part of the cost of hypertensive disease. The order of magnitude of increased costs can be gauged from figures given in Table 1. Assuming these to be representative of the country as a whole, the extension of medical treatment to all persons with a diastolic pressure of 115 or above would raise the proportion of 35-74 year olds actually under treatment from the present figure of about 2 per cent to about 5 per cent and would probably more than double the £11.7 million spent on anti-hypertensive drug therapy in 1969. If treatment were extended to persons with a diastolic pressure of 95 or above, then the figures in Table 1 indicate that this would cover as many as another 20 per cent of the 35-74 year old population. Even bearing in mind the lower cost of antihypertensive therapy based on diuretics in the 95-115 range, the cost would be in the order of £50 million a year, a figure which is of the same order as the real increase in total NHS resources in any one year. Clearly, expenditure of this magnitude which would entail the postponement of many alternative projects within the NHS could only be justified by very convincing evidence of the benefits to be derived from mass screening and treatment.

Also on the debit side are the possible harmful effects and risks of antihypertensive therapy. Present day hypertensive drugs can still have some unpleasant side effects which are clearly apparent to the patient, including postural hypotension and impotence, though the diuretics which may be used for mild and moderate levels of high blood pressure are more acceptable in this respect. However, there are in addition the unknown risks of long term therapy with drugs which have only been known for a relatively short period of time. A person who is placed on antihypertensive therapy is likely to remain so for life. Clearly the risk of long term

toxicity is a vitally important unknown factor which demands that any routine screening and treatment programme should maintain close supervision of the population in order to detect serious side effects.

Benefits

Against these costs can be set the gains from treatment following a screening programme. Here, there is still disagreement as to whether the prognosis of cases of high blood pressure likely to be discovered through screening is significantly improved through treatment. Whereas it is generally agreed that men and women who come to the attention of the health services through symptoms associated with hypertension should be given treatment to lower their blood pressure, there is doubt as to whether treatment at the asymptomatic stage confers any benefits over and above those from starting treatment only when symptoms are apparent.

Behind these reservations is an unwillingness to commit large scale resources to a programme and place people on therapy until its effectiveness is demonstrated beyond reasonable doubt. Once any screening or preventive programme has been started both patients and doctors are likely to find it extremely difficult to run the programme down even if new evidence demonstrates its ineffectiveness. Procedures, once established, tend to have a momentum of their own. The key question is the extent to which benefits of treatment for various levels of asymptomatic hypertension have been demonstrated beyond reasonable doubt and here the critical issue is the extent to which the results of the controlled trials of antihypertensive therapy can be generalised to apply to the whole population which would be subject to screening.

The first point to arise from the results of the trials so far is that no clear benefits have been demonstrated from the treatment of women without severe symptoms. The trial of Hamilton *et al* (1964) was the only one to include women (under 60). All of them were 'asymptomatic' in the sense that they showed 'no complications such as papilloedema, exudates or haemorrhages on retinal examination . . . no signs of cardiac enlargement, and no albuminuria . . .' All had diastolic pressures over 110. The treated group of women did not fare better *en bloc* than the untreated women, though it is very important to emphasise that less than half of them had their blood pressure well controlled (initially ganglion blocking agents with or without thiazide diuretics were used and later methyl dopa was substituted in some cases). Those whose pressure was well controlled did fare significantly better. It may be that the women represented relatively difficult cases (perhaps

more susceptible to complications and less responsive to therapy) since they had been referred to hospital for 'essential benign hypertension'. Unfortunately there is no clearer evidence to indicate whether women with diastolic pressures of 110 or more who are discovered through screening would (a) be as subject to complications as Hamilton's control group and (b) be as responsive or more responsive to therapy. Controlled trials to measure the effectiveness of treatment and blood pressure control on women of various ages, symptoms and levels of pressure are very urgently required. They may well demonstrate benefits from screening, though until then a screening programme for asymptomatic women would not be justified.

For middle-aged men, both controlled trials, Veterans' Administration (1967) and Hamilton *et al* (1964), have shown good control of pressure and significant benefits from treatment when diastolic pressures are over 110 or 115. However, the proponents of the view that screening is not yet justified can point out that the characteristics of both Hamilton's and the Veterans' subjects were probably dissimilar from the characteristics of a population subject to screening. The Veterans' subjects were certainly very well motivated to continue with therapy and it is unlikely that hypertensives discovered through routine screening would be so co-operative. There is evidence from Wales that asymptomatic middle-aged men who have not gone through any process of referral through the health services are only likely to remain on therapy for three years after being picked up and treated at random. But the really crucial issue is the extent to which the subjects of the trials were 'asymptomatic' or 'symptomatic'. Neither controlled trial is completely clear on this point. Neither of them included persons who were displaying signs of complications of hypertension, though the very fact that Hamilton's subjects were referred to hospital for 'essential benign hypertension' may indicate that some signs or symptoms of hypertension, if as yet only minor, had appeared and the number of morbid incidents in ensuing years was perhaps greater than might be expected among 'asymptomatic' hypertensives. Similarly among the Veterans' subjects, the method of selection was not made clear and although those with signs of serious complications were excluded there may have been a bias towards the selection of those men who were, if not as yet 'symptomatic', at least on the verge of developing serious symptoms and thus much more likely than randomly discovered asymptomatic men with the same level of blood pressure to show benefits from treatment.

On the other hand it can be argued that people are notoriously unpredictable in their usage of health services. It would be totally

unrealistic to expect hypertensives who were about to develop severe symptoms to select themselves from the wholly asymptomatic hypertensives and initiate the process of examination and treatment themselves. It is probable that a large proportion of those men with the same level of symptomatology as those in the trials are unaware of any pressing reason for consulting a doctor and are unlikely to be discovered except by screening or the development of a severe complication. Where the onset of the complication is gradual and the process reversible there may still be a case for waiting until hypertension becomes symptomatic, but where the onset is sudden and often leads to irreversible damage or death then there is a very strong case for providing treatment before any signs have become apparent. Stroke and dissecting aneurysm are sudden occurrences which have been shown to increase in incidence with each increase in pressure level and which have been shown to be largely preventable by treatment in the two trials.

Such evidence as exists suggests that it is the level of pressure *per se* (and not the development of associated signs and symptoms) which is the primary predictor of excess morbidity and mortality from stroke. There is therefore very strong (though not yet conclusive) evidence that successful lowering of blood pressure among the sort of men who would be discovered by screening for diastolic pressures over 110 or 115 would confer benefits over and above those to be derived by haphazard treatment of persons who consult their doctors.

Perhaps the final factor in the balance is the point that no new evidence from controlled trials can be expected for five years or more, since it would take at least that time to find and report significant results from hypertensives picked up through screening. On balance a screening programme to discover and offer treatment to middle-aged men with diastolic pressures of over 110 or 115 mm Hg can be almost certainly justified on present evidence. The extra cost of drug therapy for a nationwide programme for males alone would probably be in the order of £5 million to £7 million⁷ as compared with £11.7 million already spent on antihypertensive therapy in 1969. Perhaps an alternative might be a programme restricted to one of the new administrative areas to come into existence in 1974. A pilot programme under operational conditions could provide information and experience on the important question of how regularly and for how long randomly discovered asymptomatic men can be expected to remain on their therapy. It could also provide experience on the

7 An exact figure could only be predicted with precise data on the average cost of treatment at various levels of pressure and such data does not exist.

actual costs of screening and treatment. Hart (1970) has described a programme carried out in Glamorganshire within the normal framework of NHS general practice without any overall increase in workload, and thus presumably with few extra manpower costs. The general practitioner may be in the best position to perform the initial blood pressure measurements since he sees an average of 60 per cent of his middle-aged male list in the course of a year (HMSO 1958), but all sectors of the health services can have overlapping roles to play and a programme initially restricted to one area could provide useful guidance here.

Below the diastolic pressure level of 115 there is only one controlled trial upon which to base conclusions, the Veterans' Administration's (1970) trial of middle-aged men with diastolic pressures above 90 and below 115. Benefits were demonstrated within this range but here again all of the reservations and counter arguments that were cited above apply to the results of this trial too. On the crucial question of the degree of symptoms displayed by the subjects, the report of the trial is not sufficiently clear, but at this range of pressure levels only relatively minor symptoms of hypertension would be expected. Thus men with similar levels of symptomatology and blood pressure could not realistically be expected to initiate the process of examination and treatment themselves.

However, in this trial most of the benefits were concentrated in the upper end of the range, from 105-114. 'Effectiveness' of treatment, it was noted, was only half as great over the 90-104 range and the small number of morbid events reduces confidence in the significance that can be attached to the findings. The conclusion must therefore be that although all the results are in the same direction, the evidence is not yet strong enough to warrant extension of screening down to these mild to moderate levels of asymptomatic hypertension at a cost of another £30-40 million a year in drugs if both men and women were covered. It is within this range that large trials are necessary to establish the pressure and symptomatology at which benefits start. The necessary results may eventually come from the trial at St. Thomas's Hospital and Cardiff though when treatment of pressure levels of around 100 are being evaluated a very large number of persons are needed if significant results are to be obtained and the problems involved in conducting this sort of trial are considerable.

If benefits were in fact eventually proven for treatment of asymptomatic persons of both sexes down to a diastolic pressure of around 95 then a programme (in the order of £50 million a year) might face a serious financial constraint within a National Health Service working on a limited budget.

Payment by the patient himself, it could be argued,⁸ might be one method of obtaining funds specifically for this purpose. However, apart from arguments of equity this would lead to the situation where effective treatment was limited by price while a large amount of ineffective treatment within the bulk of the NHS was not so limited. Where there are clear benefits of antihypertensive treatment the objective of policy ought to be to make sufficient resources available to meet the need at a defined level and the price mechanism would have little relevance to this process. Either new sources of funds which do not limit demand by a price barrier would be necessary, or more ideally a shift of resources away from the many areas of ineffective medical care. This would have the effect of both reducing waste and increasing the final 'output' of the NHS in terms of reduced morbidity and mortality. At least, there is a good case for ensuring that the new resources which become available to the NHS each year are earmarked for the treatment of the levels of hypertension for which benefits are clear or any other programme that offers a similar return on investment.

The immediate problem is not pressing if a policy of screening middle-aged men to pick out diastolic pressures of over 115 were adopted. The extra cost of £5-7 million a year would not be critical, but even here perhaps there is room for a novel approach to doctor/patient relationships which might be extended if screening for lower levels were found to be justified. This approach would, incidentally, reduce the financial strain that would be imposed by universal application of a detection and treatment programme. Normally, a doctor determines what is best for his patient according to his own judgement. Since only he comprehends the nature of the condition for which the patient presents for relief, there is rarely an opportunity to discuss the merits and demerits of treatment with the patient with a view to reaching a joint decision. In the case of high blood pressure picked up through screening, however, the patient is likely to be symptomless for practical purposes. He does not present for the relief of symptoms. Furthermore, the doctor is unable to tell the patient that he is suffering from a disease per se. All that he can normally do is weigh up the risks associated with the patient's level of blood pressure, and if the patient is told the risks then there is no reason why he should not make his own judgement for himself.

8 It could, for instance, be calculated on the basis of the American insurance figures (Fig. 1) that a man of 45 whose pressure of 150/100 was reduced to 120/80 or below would expect to gain $11\frac{1}{2}$ years of life. If he were to remain on therapy for the rest of his life at (say) £10 per year in drugs, each extra year of life would have cost him about £30 allowing for inflation and discounting of future values. This could form part of the basis for his decision on treatment.

What is suggested is that the general practitioner might tackle the problem by explaining precisely and quantitatively to the patient the risks attached to his level of blood pressure and the benefits and demerits of treatment. The patient could then come to a rational decision of his own either before or after trying the treatment to find whether the side effects are acceptable. This would operate within the constraint that patients would only be enabled to opt for or against effective treatment. They should not be given the opportunity of satisfying a preference for ineffective therapy. Clearly this would require a good deal of educational effort for both the general practitioners and their patients, including the provision of a clear and reliable summary of the statistics (and in this case fairly good ones do exist) for all general practitioners. It would also be vitally important to take account of the possible mental damage that might be inflicted on a person who is told that he has a life threatening condition yet who for some reason does not accept treatment. Clearly doctors must be selective in deciding who should or should not be given the facts, but it may be at least one partial solution to the problem of reconciling scientific rationality with consumer choice. The treatment of high blood pressure could be just the sort of field in which a doctor/patient relationship could be encouraged which relied less on mystique and more on the provision of effective treatment jointly chosen by the doctor and the patient.

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