The management of high blood pressure in general practice

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HIGH blood pressure is a measurement, a quantified physical sign. Using only a crude sphygmograph to estimate arterial tension, Mahomed of Guy's understood its relation to renal and cardiac disease remarkably well by 1881 (Batty Shaw, 1952). Riva-Rocci's sphygmomanometer in 1896, and Korotkoff's interpretation of the sounds it produced in 1905, provided the technical basis for the measurement we use today; yet as late as 1939 a British Army medical officer was refused a sphygmomanometer as a normal piece of general practitioner's diagnostic equipment, and it is only since the second world war that failure even to possess one became an exceptional crime in primary care.

The lethal associations of hypertension were recognised very soon after sphygmomanometry became simple and easy. Large series of figures were accumulated from 1910 onwards, particularly in the United States, and though their basis of selection cast doubt on some of the early conclusions about the shortened expectation of life associated with higher pressures, they have been broadly confirmed by the massive build and blood pressure study of the Society of Actuaries (1959) and the study of the Metropolitan Life Insurance Company (1961) covering over five million lives insured, over follow-up periods of 20 years.

Up to the 1950s, when the first satisfactory treatments arrived, there were two groups of people really interested in blood pressure; the insurance companies, because they won or lost money on its predictive value for expectation of life, and academic physicians. The latter had highly selected cases, with gross over-representation of renal and retinal damage, and a prognosis generally so bad that one could forgive general practitioners trained in teaching hospitals for generally discounting the seriousness of high blood pressure in the absence of gross signs of organ damage.

There is no great urgency in diagnosing an asymptomatic condition that one can do nothing about, and every reason to play down the risks. At the same time a mythology of symptoms was built up by doctors and transmitted with authority to their patients, associating headache, giddiness, flushing, sweating, and other symptoms related to emotion through visceral nervous pathways, with high blood pressure; patients with these symptoms expected their blood pressures to be taken—and they were. Thus the myth of hypertensive symptoms, as a pointer to diagnosis, became self-replicating.

The arrival of effective drugs in the 1950s, and of tolerable drugs in the late 1960s, with good evidence of the effectiveness of reducing pressure in preventing stroke, renal damage, retinal damage, and heart failure, imposed a duty on doctors to make this treatment available to those who needed it. Since at the optimum time for treatment, these people do not have symptoms, diagnosis must be a planned operation applied to whole populations at risk, an almost completely new situation for the general practitioner (not quite, because antenatal care is already a model for organised anticipation of symptoms).

It is management in this strategic as well as tactical sense that will be discussed and described here. As a condition with a quantified definition, a quantified relation to

Journal of the Royal College of General Practitioners, 1975, **25**, 160—192

outcome in mortality and morbidity, a quantified relation to various risk-groups divided by sex and age, quantifiable targets in treatment, and at the same time of real importance in terms of death and gross handicap, the rational management of high blood pressure could be a critical pathway along which much else may later follow.

An operational definition of high blood pressure

Hypertension falsely appears to be a more precise term than high blood pressure, for the plain fact is that the definition of high blood pressure depends on the definition of high. Many data now show that apart from a tiny minority of cases of secondary hypertension, levels of blood pressure are continuously distributed, and continuously related to mortality, through their entire range, without any natural breaks to suggest a valid qualitative definition (Pickering, 1968). To create a separate category of hypertensive disease, involving the presence of causally related symptoms or evidence of organ damage, is to dodge the whole issue; these are the very outcomes we seek to avoid by diagnosis and treatment. High blood pressure is a sign, not a disease; a sign we must define, and then identify and if possible change, before it becomes a disease. Secondary prevention is nine-tenths of the content of the management of hypertension—if it is properly organised.

It is true that a case can be made for regarding as normal only those distributions of pressures found in a few neolithic or nomadic populations, where pressures are not only very low, but remain stable or even fall with age (Lovell, 1967); but this is not true of all such populations, and is not consistently related to any racial or environmental factor so far identified (Shaper, 1972).

The precise definition of a condition causally related to premature death or serious disability is useful because it assists action; diagnosis implies prognosis and a plan to change that prognosis. Since prognosis and effective action are both historical variables, so is the definition within which diagnosis operates. It may at some future time (not necessarily remote) be reasonable to aim at a low, age-stable distribution of pressures similar to those of the vegetable and fish-eaters of Polynesia, or the carnivorous Masai nomads of Kenya; but our present strategy must be directed at the most pressing situation we have, in which four percent of the population aged 20–64 have blood pressures in a range requiring investigation and probably treatment (on present evidence), and about half of them are not ascertained (Hart, 1970).

The definition on which that figure is based, and adopted throughout this paper, is that proposed by Pickering (1968) for consideration of treatment on grounds of pressure alone. He chose these cutting points as those which, in Bechgaard's 20-year follow-up of more than 1,000 untreated hypertensives, were associated with a mortality $2\frac{1}{2}$ times that expected in the whole population, standardising for age and sex (Bechgaard *et al.*, 1956):

DIASTOLIC PRESSURES WARRANTING TREATMENT ON GROUNDS OF PRESSURE ALONE

age	*men*	*women*
20–39	100+	110+
40–64	105+	115+

In practice, other indications (renal damage, retinal damage, cerebrovascular damage, and signs of myocardial strain) are rare below these levels. At ages over 65, we really do not have the evidence for a definition in this sense. The question of diagnosis and treatment in the elderly is fully discussed later on, but in general there is little evidence to support the initiation of treatment in this age group unless there is a specific indication

for it, for instance left ventricular failure or retinal damage; its continuation after 65, having been initiated some years earlier, is a different matter.

Sex differences

This operational definition applies different scales in men and in women, in different age-groups, and it ignores pressures below these cutting points that are also associated with raised mortality.

The difference between men and women in their capacity to tolerate high pressures is great. In Bechgaard's series, whereas men under 50 with diastolic pressures over 120 had a 750 per cent excess mortality, women in this range had only a 286 per cent excess (Bechgaard *et al.*, 1956). Whereas at all ages under 40 mean pressures are a little higher in male population samples than female, the number of female hypertensives exceeds the male in middle age, and exceeds it very greatly in the elderly; this is almost certainly an effect of selection, with women tolerating high pressures much better than men and therefore surviving to old age (Miall and Oldham, 1963: Record and Whitfield, 1964). As for age, numerous studies have shown that for any given pressure, associated mortality is higher in the young than the old, simply because it is more deviant from the mean for that age-group. Division into more than two age-groups would be cumbersome though this may well become necessary.

Diastolic pressures above 85mm

Finally, can it be right to ignore diastolic pressures in the ranges above 85 or 90 diastolic, but below these cutting points? Pressures in this range are associated with substantial excess mortality. The Metropolitan Life Insurance study (1961) showed a loss of three years of expected life for men with diastolic pressures of 90 at age 45, and 1½ years for women.

However, we have no convincing evidence that mortality is reduced by treatment at these levels, and it would be wrong to assume that lifelong treatment with antihypertensive drugs is necessarily or probably without danger; thiazides, which are widely used in this range of pressure, can precipitate diabetes, itself a substantial additional risk factor for ischaemic heart disease, which is the principal cause of death associated with hypertension; they are also a cause of hyperuricaemia, which may also be a risk factor.

Nor do we yet have convincing evidence that the risk of myocardial infarction can be reduced by reduction of blood pressure. For the time being the importance of this group is as a source of recruitment of new defined hypertensives, as their pressures rise. Such an operational definition must be revised as new evidence becomes available. In 1968 they provided a good working frame. The apparently continuous distribution of blood pressure in populations may well, as Platt (1967) suggests, contain and conceal two or more overlapping populations, with substantially different risks at the same pressure. We may in future be able to define hypertension on criteria other than pressure, particularly when we have accurate medical records for two or three generations. However, there is no means of doing this now, and the operational definition suggested corresponded with the level of knowledge when whole-population care first became feasible.

Causes and associations

The extra-renal causes of secondary hypertension are extremely rare. In 20 years of general practice, with two successive populations of 2,300 and then 2,100, I have had no cases of co-arctation of the aorta, Cushing's disease, phaeochromocytoma, or primary hyperaldosteronism; they have all been suspected and looked for on the usual indications.

I have found one case secondary to a meningioma, an extremely rare cause, and one severe hypertension probably caused by an oral contraceptive. The contribution of all but the last of these to the total hypertensive population, on any definition, must be small indeed.

Association with renal disease is fairly common. Of 49 hypertensives (as defined) identified by screening the whole of my own population semi-continuously over five years, one had a congenitally absent or non-functioning kidney, two had gouty nephropathy, and there was one diabetic with renal damage. Many of the women had, of course, a recorded history of bacteriologically confirmed renal tract infection, and Kass (1967) and others have produced some evidence that the roughly four per cent prevalence of bacteriuria in women in Jamaica may be associated with higher pressures. Even if this is confirmed here, it will not account for much male hypertension, and gross unilateral infective renal disease is now rare in this country.

On a quantitative basis, the overwhelming majority of cases are of unknown cause, so-called essential hypertension. It is unlikely that this group will ever disintegrate into a number of specific secondary hypertensions of known cause, and it is almost certainly a multifactorial condition. This parallels the situation in ischaemic heart disease, except that there is no evidence of any recent dramatic change in the distribution of blood pressures to compare with the modern epidemic of myocardial infarction.

Regarding the cause of essential hypertension, Pickering's team concluded in 1954 that

> ". . . what has been called essential hypertension is a purely arbitrary segregation of those having arterial pressures in the higher ranges and having no disease to which these high pressures can be attributed. The factors concerned in the pathogenesis of so-called essential hypertension are thus those concerned in determining the arterial pressure in the population at large." (Hamilton *et al.*, 1954).

Genetic factors

What are these factors? The largest single one is certainly genetic; the tendency of children to resemble their parents seems to account for about one third of the variance in the distribution of blood pressure in populations (Pickering, 1968; Miall and Oldham, 1963), though it could be as much as two thirds. This inheritance is almost certainly not due to the effect of a single gene, a view now also discarded by Platt (1967); it is therefore not likely to be susceptible to change by enzyme substitution or any form of genetic tinkering.

The large part of the variance in blood pressure that is environmentally determined, in the widest sense, seems to be even greater in those with diastolic pressures 60 mm or more above the mean for their age-group (Miall, 1967a). The most severe cases show the least concordance with their relatives; this is a most important observation.

Obesity factor

Apart from ageing and the classical causes of secondary hypertension, there are at present only two unequivocally established post-conceptional factors influencing sustained blood pressure: obesity and family size. The effect of obesity is well known, but complex; it is still present after allowing for the mechanical effect of arm girth on the technique of measurement (Bøe *et al.*, 1957), and blood pressure can fall after successful weight reduction by amounts that cannot be accounted for by the reduction in arm girth alone (Terry, 1923; Preble, 1923). Bøe and his colleagues, who examined nearly 68,000 people, found a mean rise of 2 mm systolic and 3 mm diastolic for every 10 Kg increase in weight; not a very large effect.

Per contra, starvation and thin arms produce low pressures; in fact my clinical impression is that the effect of a really skinny arm is even greater than that of a fat one. The effect of smoking, which is generally associated with lower pressures, is probably through its effect on body weight; it is small, and reversed in male ex-smokers.

Family size factor

The effect of family size on blood pressure is one of the mysteries of medicine. A tendency for both husbands and wives to have lower pressure if they have children was clearly established and repeatedly confirmed by Miall (1959) on a Welsh valley population, and elsewhere in Britain by Lowe (1961), and in another Welsh population by me (unpublished). Miall found a similar but smaller effect in Jamaica, but the effect was not found in Germany, India, or the Gilbert Islands (Pflantz and Lovell, 1967).

The differences in men aged 25 to 54 in Miall's data were from 4·3 to 6·2 mm of systolic pressure, and in my own data, from 8 to 10 mm of systolic pressure, using a random-zero sphygmomanometer to eliminate bias. Both these sources showed a disappearance of this effect in the fifth decade, with the parous catching up with the nulliparous, which seems to argue against a theory of causation based on domestic contentment.

Another of my studies, also unpublished, showed no relation between coital frequency (as stated by 94 per cent of a geographically defined population of 350 men aged 30–64) and blood pressure, nor between coital frequency and family size.

This relation to family size is of real importance, because it is the only consistent evidence we have of a link between blood pressure and some measurable aspect of human behaviour; it is also less open to the facile interpretations that have nullified so much of the vast literature on psychosomatic aspects of hypertension.

Blood pressure is transiently raised by alarm or by vigorous physical activity. Continuous direct arterial pressure recordings made over 24-hour periods in 22 unrestricted subjects (Bevan *et al.*, 1969) showed frequent swings of up to 25 mm of diastolic and 45 mm of systolic pressures during the day, and the ranges differed little between normal subjects, those with benign hypertension, and those with hypertension in the malignant phase.

The abnormality in hypertension appears to lie not in an unusual degree of reactivity expressed by wider deviations from a common mean, but in a new setting of that mean; there is as yet no conclusive evidence that repeated stimuli of the same nature that provoke transient deviations, can cause a persistent shift in the setting of the mean. In a cautious review of the vast English language literature on the subject (mostly of poor quality), Cochrane (1971) concluded rather optimistically that it yielded

> "... general support to the idea of a link between perceived stress in the environment, a personality over-reactive to stress and high blood pressure."

A more critical review by Ostfeld and Shekelle (1967), with which I sympathise, concluded:

> "(1) It is established beyond a reasonable doubt that acute psychological stress may initiate sudden and transient elevations of blood pressure in some persons.
>
> (2) It is not established that repetitive or continuous psychological stress leads to sustained elevation of blood pressure in anyone."

Hypertension is not consistently related to neurotic symptoms. This is more fully discussed below, but it is probably in psychosocial sensory input that most of the so far unspecified causation will be found. The finding will be further delayed, so long as facile and simplistic prior assumptions continue to dominate the research field to an extent that makes it easy to miss the few really useful studies published. The present stresses of

living under the threat of nuclear war and 15 per cent mortgages are unlikely to be greater than those of famine, massive childhood mortality, and forced mass-emigration.

Many current assumptions have yet to be tested, and when they are, the results can be surprising; continuous recording of direct arterial pressures during car driving, for instance, showed only small and transient rises while overtaking, and otherwise little difference from sitting down at home (Littler *et al.*, 1973). Automatic semicontinuous recording in free subjects seems to be the only way to liberate us from the orienting reflexes of the normal test situation, which are quite likely to differ for mainly cultural reasons between populations and between subjects. Even using such techniques, we need hypotheses that take full account of the complexity of social behaviour, of cerebral cortical activity, and of its relation to autonomic vascular control.

Oral contraceptive factor

The contraceptive pill, at least the oestrogen-progestogen combination, certainly raises blood pressure in some cases (*British Medical Journal*, 1973; Weir *et al.*, 1974). The rise is usually small and reversible three months after stopping the Pill. This tendency is greatest in women over 40 who are also high-risks for thrombo-embolism, and are least suitable for oral contraception with oestrogen-progestogen preparations. Another association in women is with uterine fibroids (Miall, 1967b); the effect is probably hormonal, and could be connected with the low-fertility factor already discussed. There is also a small independent association of raised pressures with anaemia in both sexes (Elwood *et al.*, 1970).

Some factors not associated with hypertension

Blood pressure is *not* associated either positively or negatively with serum cholesterol, at least over the age of 40 (Epstein and Eckoff, 1967). It is probably not associated with skin colour (Miall and Cochrane, 1961); a great deal of work in the United States has ignored the effect of orientating reflexes that may operate differently in white and black populations being examined by white doctors in variously threatening test situations.

The high mortality of black Americans from cerebrovascular disease may be an independent effect related to social class; in England and Wales in 1959–63 social class 5 showed a 35 per cent excess mortality from cerebrovascular disease, and social class 1 showed a 14 per cent deficiency. Social class 5 showed almost the same excess for hypertension mortality, 38 per cent, and social class 1 a 15 per cent deficiency (Registrar General, 1971). It is possible that both these differences may in part reflect differences in the quality of medical care, which may also apply to blacks in the United States.

Blood pressure is also probably not associated with salt intake, at least in Britain (Miall, 1959), and is not consistently related to occupational physical activity (Lowe, 1964).

There may be a positive association with living in soft-water areas (Stitt *et al.*, 1973). There seems to be no association with sleep patterns (Winkelstein, 1967). Although many studies abroad have shown higher pressures in urban than rural populations, and a few have shown the reverse, Miall's (Miall and Oldham, 1958; Miall, 1959) careful study of Welsh mining and rural populations showed no difference. He has also shown that the level of attained pressure at any one time is the chief determinant of the size of the next increment with age; high pressures rise with age more rapidly than low ones (Miall and Lovell, 1967). There seems also to be a smaller, independent effect of ageing, unrelated to previously attained pressure (Miall and Chinn, 1973). This carries the important clinical implication that sustained reduction of blood pressure could have a lasting effect on the underlying tendency for high blood pressure to accelerate with age.

Important role of primary physicians

I hope this lengthy review of causation will help primary physicians to see that although much is known (little of which seems to be effectively taught to undergraduates) what we do know is dwarfed by what we do not know. Many of the necessary advances will have to be made in primary care where prolonged studies of cohorts with a known basis of selection from a defined population, will be possible. As we shall see, the organisation needed for this is much the same as that needed for the planned delivery of diagnosis and treatment, and the primary physicians of the future may play as great a part in clinical and causal research as hospital physicians do now.

Diagnosis: planned and transactional

High blood pressure is not related to symptoms, short of the stage of organ damage, to avoid which is the purpose of diagnosis and treatment. There is no relation between headache and level of blood pressure in the diastolic range below 130 (Waters, 1971). There is sometimes an association above that level (Al Badran *et al.*, 1970), which may have prognostic importance as an indicator of eventual cerebrovascular accident (Fry, 1966). It is certainly possible to have diastolic pressures over 170 without headache or any other symptom, as I have seen two such patients in the practice.

Of the other traditional symptoms of giddiness, dizziness, breathlessness, fatigue, palpitation, anxiety and depression, only breathlessness has been shown to be associated with level of blood pressure in a properly controlled study, and even then the association was small (Robinson, 1969). Similar population-based studies showed no association with scores for neuroticism among those not consulting their doctor, but there was an association in those who did, which was even greater in those referred to hospital (Robinson, 1962). Cochrane (1969), in a well-controlled study, found no association at all with neuroticism scores, and attributed Robinson's findings to the depressing effects of medication.

Doctor-initiated diagnosis

If high blood pressure is unrelated to symptoms, it cannot be efficiently diagnosed by the normal transactional process in general practice, in which patients with symptoms go to a doctor for relief. Data on the proportions of hypertensives receiving treatment in the population at large confirm this. Miall studied the proportion of men and women in a whole population with diastolic pressures over 110, who had received treatment at any time from their doctor (Miall and Chinn, 1974). Of men aged 45–54 about 38 per cent had had treatment, and of men aged 55–64, about 32 per cent. The proportions of women in the same age groups were about 58 and 57 per cent. In those over 65, for whom antihypertensive treatment is generally ineffective in preventing stroke, and carries serious risks of unwanted effects that can be dangerous, about 57 per cent of the men and 63 per cent of the women had been treated.

Further confirmation comes from market research. Two thousand general practitioners randomly sampled and stratified for year of qualification and area of practice are observed for prescribing habits for one week in each quarter. Table 1 gives the results for one quarter in 1968 and another in 1973 (Intercontinental Medical Statistics Ltd., 1974). Nearly 50 per cent more women than men were under treatment with antihypertensive drugs under 65, although prevalence in men is slightly greater before the menopause, and associated mortality is very much greater. About half those under treatment were aged 65 or more.

The number receiving treatment is staggering. Intercontinental Medical Statistics estimated a total of over seven million prescriptions for antihypertensive drugs at 8·75 million consultations in 1968, rising to over nine million prescriptions at 11¼ million

TABLE 1

DISTRIBUTION OF TREATED HYPERTENSION BY AGE AND SEX IN A RANDOM SAMPLE OF 2,000 GENERAL PRACTITIONERS, 1968 AND 1973 (INTERCONTINENTAL MEDICAL STATISTICS LTD. 1973)

Year	*Number of observations*	*F:M under 65*	*F:M all ages*	*Per cent 65+*	*Estimated national consultations prescriptions*
1968	2,051	1·49	2·13	*50*	8,760,000 7,014,000
1973	2,766	1·47	1·87	*57*	11,233,000 9,284,000

consultations in 1973. Too many people are taking antihypertensive drugs on slipshod indications, with perfunctory follow-up and control, and too few of those with hypertension at levels where treatment is now mandatory on good evidence have been diagnosed and brought into programmes of effective treatment.

Discussion of this subject with hospital physicians is usually difficult. They say, " We always take our patients' blood pressures "; which, being translated, means that their housemen do. They feel it is little to ask that general practitioners do the same. Such arguments do not merit serious discussion between gentlemen.

Always taking blood pressures presents serious logistic problems. Twenty-two representative Scottish general practitioners were observed in a beautiful study by Buchan and Richardson (1973). They found a median consultation time face-to-face with the patient of 4·2 minutes, with most consultations falling within plus or minus two minutes of this time. The mean time taken for a sphygmomanometer reading was 1·2 minutes. Blood pressures were measured in about ten per cent of all consultations with patients over 15 years old, ranging from 8·8 per cent of those aged 25–44 to 14·6 per cent in those over 65.

Realistic discussion of more complete blood pressure measurements in primary care must start from these data, which do at least represent a system of care designed to give equal access to all. The American pattern of care, with a much more complete routine programme of investigation usually related to sickness episodes, but increasingly including a " health check " examination, does not result in much better delivery once we look at whole populations at risk. In a " small, moderately prosperous " town in Georgia, with a good standard of medical care, 41 per cent of hypertensives found in a large random sample did not know they were hypertensive, and only 30 per cent had been under treatment. Only 19 per cent of white women were unaware of their hypertension, compared with 42 per cent of white men, confirming the same trend as in Britain (Wilber, 1967).

British system of care

The British system, with defined populations and a minimum of economic barriers to treatment, offers the possibility of planned diagnosis of important asymptomatic conditions, but it is a possibility that can be realised only through struggle against powerful traditions. British general practice, particularly in industrial areas, stems from the tradition of frequent, brief transactions for episodes of mostly minor illness, in which treatment was usually illusory, and diagnosis an exercise in prophecy rather than the first stage of an effective plan for action.

Only now are we slowly, painfully, and incompletely adapting to the effective care of the chronic illness that makes up most of our really disabling morbidity and premature mortality. We have hardly begun to adapt to the tasks of secondary prevention, which is the real nature of the treatment of hypertension short of its gross end stage. For the time being, it is the patient with symptoms notionally associated with hypertension who is most likely to have her blood pressure measured—the patient with

headaches, giddiness, tinnitus, flushing, palpitation, and other left submammary sensations. These symptoms present more commonly in women than in men, and in the old more than the young.

Doctors in general practice learn quickly to anticipate the expections of patients, and know well that a simple mechanistic explanation for vague and difficult but medically unthreatening symptoms is acceptable, however unrelated to outcome it may ultimately prove. If fat elderly and middle-aged women fear hypertension and associate their minor symptoms with it, their blood pressures will be measured more often and more hypertensives will be found among them. From this situation springs the present irrational pattern of diagnosis and treatment (Hart, 1974).

The diagnosis of hypertension can and should be a planned procedure related to populations rather than to individual consultations. Decisions on treatment should follow the evidence we have on its outcome in terms of improved longevity and reduced stroke morbidity, and on the other hand, of iatrogenic misery and disaster.

Organisation of planned diagnosis

Finding all hypertensives in a population requires a sorting procedure that will identify all cases that may be considered for treatment and further investigation, and also that intermediate group of borderline pressures from whom new cases are most likely to appear in the next few years. This procedure must be applied more or less continuously over time, within spans related to the natural history of hypertension.

Though the progression of the mean pressure of a whole population with age is a smooth, predictable process, this is not true of individuals. Rapid progression can occur into the defined range, particularly if attention has been directed only at diastolic pressures; if Miall and Lovell's hypothesis is correct, we should expect those with " high normal " pressures to show larger annual increments with age than those with lower pressures, and these runners-up probably need following up at least once in every two-year span; in my own practice we have been trying to follow them up at least once a year. The rest, with diastolic pressures more than 15 mm below the defined cutting points, are being followed up at least once in each five-year span.

The first stage in such a sorting procedure must be to define the age-group at risk. The case yield under 30 is probably too small to justify screening on present evidence, and finding asymptomatic hypertension over 65 probably does not influence outcome positively. The proportion of the registered population of all ages who consult at least once during one year was 67 per cent in the Royal College of General Practitioners' morbidity survey of 1970–71 (Office of Population Censuses and Surveys, 1974), and over five years this probably reaches about 90 per cent. All but a few of these contacts can be used to obtain blood pressure readings, and if 100 per cent coverage is aimed at this can be secured by invitation letters and ultimately by home visiting to the residual ten per cent.

The workload involved in this can only be coped with by training ancillary staff (not necessarily nurses) to adhere to a strict but simple protocol in measurement and recording of blood pressure. Because most doctors have been carelessly trained and have acquired intractable bad habits, others with specific training and supported by a subsequent critical interest in their work, will usually do better.

Standardising technique

A mercury manometer should be used (the scale is too small on the aneroid instrument and there is more to go wrong), with a full 300 mm column. None of the makers of the standard instruments make cuffs the inflatable parts of which will fully encircle a stout arm. An outsize cuff must be used for very fat arms, and at present the only ones available are thigh cuffs—long enough, but much too wide, and very hard work to inflate.

The effect of arm girth in all but the very obese is quite small, and does not justify routine correction (Holland and Humerfelt, 1964). There is an effect of obesity independent of the effect of arm girth. The glass tube must be cleaned with a pipe cleaner three or four times a year, and the meniscus should be checked at zero from time to time. A diaphragm stethoscope is easier to use than a bell, and less likely to give false readings by pressing too hard on the brachial artery.

Some clothing must usually be removed from the patient. The commonest source of error is a tightly rolled sleeve impairing flow through the brachial artery, and forcing the cuff down onto the crease of the elbow where the arterial sounds cannot be clearly heard.

A standard procedure should be followed so that all readings are comparable, using either the right or the left arm (but consistently, one or the other) with the patient seated and the semiflexed arm resting comfortably on a table. The cuff should be inflated rapidly to 200 mm, or to a point just above the disappearance of a palpable pulse at the wrist, and then deflated slowly and steadily at about 2 mm to each pulse beat; a very rapid descent gives false low readings, and is a common error.

The stethoscope should be applied lightly above the elbow crease, over the brachial pulsation. There is no advantage in recording standing and lying pressures, unless patients on ganglion-blocking drugs are being followed up. Pressures should be recorded to the nearest 2 mm below the sound changes; first regular tap for systolic, disappearance of sound for diastolic (phase 5). Muffling is in my experience more difficult to recognise and to teach. When sounds can be heard right down to zero, a tight sleeve should be sought.

Importance of several readings

The diagnosis of uncomplicated hypertension should never be made on a single reading, however high; even a diastolic pressure of 170 can be repeated three or four times at five-minute intervals, giving useful information as a baseline for future pressure control. Either replicate readings on prolonged single occasions, or single readings on at least three occasions, should precede other action in every case. Single readings on multiple occasions may fit in better with the usual style of National Health Service practice, and has the advantage of being less frightening to the patient, with more opportunities for effective patient education; but without good organisation it can also lead to serious ommissions from the essential checklist of investigations needed before treatment begins. Though I have not done this myself, replicate readings at five minute intervals is a possible initial sorting procedure (Apostolides *et al.*, 1974).

Other data collected by ancillary staff

When blood pressure measurements are made by ancillary staff, they must be encouraged and continually reminded to record and report relevant observations about the apparent mood of the patient and other factors that may produce an alarm reaction, and a pressure that is not truly casual. Some patients have to be seen several times before valid readings can be taken.

At the same time height and weight should be recorded, as well as current smoking, oral contraception and occupation, so that all this information is already before the doctor at first interview.

Logistics

The logistics of this sorting procedure, as applied in my own practice, are shown in figure 1. The big fall in numbers of defined hypertensives between first and second

readings, and the much smaller fall between second and third readings, suggest that three readings do define a fairly stable group. These proportions will probably be reproduced in other populations where the same procedure is followed, yielding an initial defined group of hypertensives about four per cent of those at risk between 20 and 64, with rather more men than women, particularly in the younger groups. The practice organisation required for this work has been well described by Coope (1974).

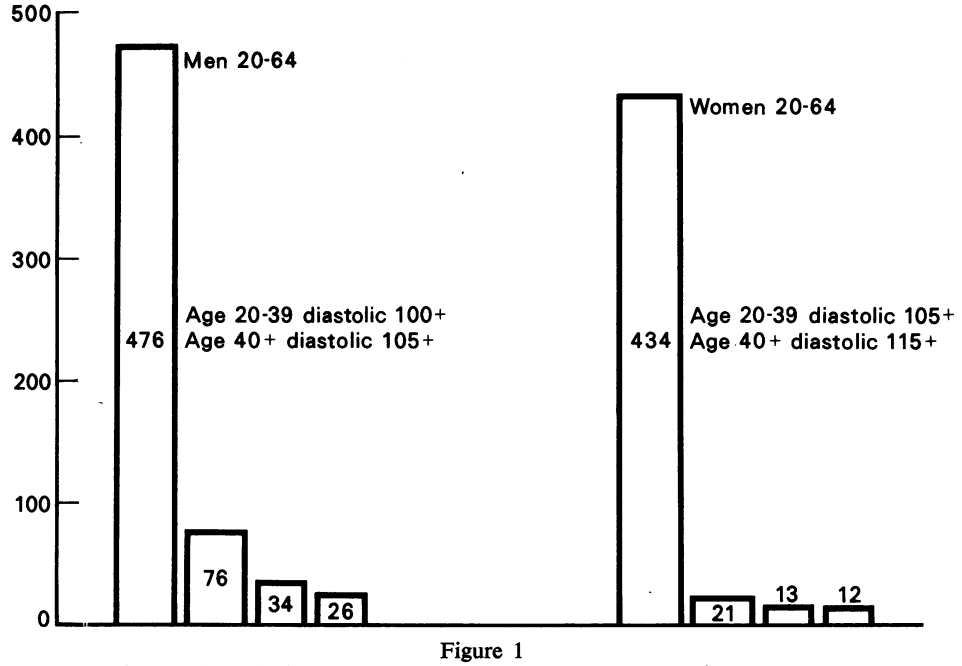

Figure 1
Persistence through three readings of diastolic pressures warranting treatment

The workload involved in this diagnostic stage is not excessive. In my own practice we found that annual consultation rates were remaining about the same, before and since total screening began, and are now falling slightly. It seems to be true of most well-organised practices that there is an annual reduction in patient-initiated consultations (Royal College of General Practitioners, 1973), and the time gained in this way can be used to increase doctor-initiated consultations without any net increase in workload The really heavy extra commitment lies in the follow-up of treated hypertension, and if treatment at diastolic pressures under 100 becomes validated this may become a serious limiting factor.

Investigations before treatment

The initial investigation of hypertension should serve three aims: to identify reversible primary causes when they exist; to identify high-risk subgroups independent of the level of pressure; and to provide baselines for subsequent control and evaluation of complications. The display of knowledge and diagnostic defensive cover is not relevant to the effective care of patients.

Secondary hypertension in apparently well people should for the most part be detected by running through the following checklist of simple tests, providing that this possibility continues to be borne in mind throughout the many subsequent years of treatment and follow-up—particularly in those cases that are unexpectedly difficult to control:

Co-arctation of the aorta	Augmented neck and wrist pulses Absent or delayed femoral and ankle pulses
Primary aldosteronism	Low plasma potassium Hypokalaemic T-wave depression sometimes Muscle weakness sometimes
Phaeochromocytoma	Urine vanillylmandelic acid (VMA) No response, or anomalous response, to treatment with antihypertensive drugs
Renal disease	
Polycystic kidneys	Family history (simple dominant)
Other congenital deformities and chronic pyelonephritis	Recurrent renal tract infections
Gout nephropathy	Arthritis and hyperuricaemia, stone
Hypercalcaemia	Stone, hypercalcaemia
Renal arterial stenosis	Occasionally, para-umbilical mumur, asymetrical pyelogram, and in all, impaired renal function (creatinine) and bacteriuria.

Co-arctation is easily diagnosed and treatment relatively straightforward. For the rest, hyperaldosteronism should be suspected where routine ECG shows T-wave depression (more commonly, of course, associated with left ventricular strain), and plasma potassium should be estimated before the pitch has been queered by thiazide diuretics. A simple screening technique by measurement of rectal electrical potential difference has been described (Edmonds and Richards, 1970), but so far as I know has not been extensively applied.

Renal artery stenosis is difficult, expensive, and sometimes dangerous to diagnose accurately, is usually associated with extensive and serious atheromatous obstruction elsewhere, and in nearly all cases can be more safely and effectively treated with antihypertensive drugs than by surgery (*The Lancet*, 1969); so the world won't come to an end if we miss it.

The much commoner renal lesions bear a complex relationship with hypertension and rarely deserve surgical correction on this indication alone (*The Lancet*, 1968). Obviously renal lesions may need treatment in their own right, and hypertension is itself a serious danger to the failing kidney.

In general this is a field in which much unrealistic advice has been given, impossible to apply on a mass scale. It is wrong that the effective treatment of the relatively enormous numbers of cases of primary hypertension, or of secondary hypertension with irreversible prime causes, should be impeded by extravagant and often poorly discriminatory tests for very rare causes.

A more serious question is the possibility of segregating high-risk subgroups within the hypertensive group as a whole, independent of levels of pressure. This would be particularly desirable in the case of women, so many of whom reach healthy old age with long histories of diastolic pressures sustained well above 120 or even 130; it would be wonderful to be able to pick out the high-risk cases, and concentrate treatment on them alone.

So far this possibility is largely unrealised, and the great bulk of the evidence is that it is the intraluminal pressure itself that is the cause of organ damage, particularly in the case of Charcot aneurysms and stroke; overwhelmingly the best index of risk is

mean blood pressure, its accuracy depending largely on the number of readings contributing to the estimation of that mean. However, there are other variables of real prognostic value, independent of level of pressure (but in practice, in almost every case, associated with high pressures, diastolic 105 at least). Retinal changes up to and including arterial narrowing but without other retinopathy are associated with about half the subsequent five-year mortality found in those with retinal exudates, haemorrhages or papilloedema, in untreated hypertension (Keith, Wagener and Barker, 1939). This difference in mortality seems to be associated with renal damage rather than stroke, cardiac failure, or infarction (Smith, Odel, and Kernohan, 1950), and such cases are rare in primary care.

Although headache is probably no commoner in moderate hypertension (below diastolic 120) than in the general population, Fry found that initial complaint of headache in the presence of hypertension was associated with a threefold increase in stroke mortality in the subsequent ten years, compared with those not so complaining (Fry, 1966). Although his numbers were small, this is an important observation and should probably influence the decision to treat in borderline cases. There seem to be no other prognostic indicators of future stroke, but a history of even the most transient paresis or dysphasia makes treatment mandatory; specific enquiry should always be made about previous facial weakness or dysphasia, however transient.

ECG changes of definite left ventricular hypertrophy (R> 12mm aVL, or >20 mm I, II, III, aVF, or >26 mm V5 & 6 plus ST segment depression and inverted or flattened T I, II, aVL and V2-6 (Rose and Blackburn, 1968), even when not associated with coronary disease, congestive failure or rheumatic valvular disease, carry a mortality of 59 per cent over 12 years. In men over 45 with these changes, half die within eight years of developing this ECG pattern (Kannel, Gordon and Offutt, 1969). This pattern of left ventricular strain has a prevalence of about three per cent, is closely related to level of blood pressure, and is present in over half those with systolic pressures over 200. It correlates poorly with radiological enlargement of the heart and with left ventricular mass at necropsy (*The Lancet*, 1970) and probably indicates an altered sequence of electrical conduction associated with sudden death, rather than true muscle hypertrophy.

Mortality is doubled in the untreated disease by the presence of proteinuria, after standardising for age, sex, and arterial pressure (Bechgaard, Kopp, and Nielsen, 1956), but this is mainly associated with the malignant phase, which should nowadays be very rare. Impaired renal function roughly trebles mortality, but again it is rare in the lower ranges of pressure (Sokolow and Perloff, 1961).

There is some evidence that raised serum IgG levels in hypertension may reflect vascular damage directly, and could be of prognostic value (Ebringer and Doyle, 1970): this is so far only an interesting possibility. Plasma renin activity is not related to heart attacks or strokes in hypertension (Mroczek, Finnerty and Catt, 1973).

It is difficult to overstress the importance of securing reliable measurements of all important variables before treatment begins: retinopathy, ECG changes, radiological heart size, and renal function tests may all revert to normal with treatment, plasma potassium may fall with thiazide diuretics, and the degree of hypertension itself can be estimated only by withdrawing treatment, if the number of readings before treatment is insufficient.

A baseline ECG is absolutely essential, as quite minor changes in the limb leads may later be easily detectable if there is a previous tracing to compare them with, and one must assume that most of these cases will eventually have episodes of myocardial infarction. In every way, full assessment is easier before treatment has started, and the temptation to rush in with drugs before investigation is complete must be resisted.

The necessary investigations are time consuming, and my own practice has been to

divide them up into three, weekly follow-up sessions after the first three ascertainment blood pressure readings. During these three weeks placebo treatment (ascorbic acid 50 mg twice a day) is used to retain co-operation and to evaluate minor symptoms that may later be wrongly attributed to the side-effects of effective drugs; this has been instructive. This time is also used to try to persuade cigarette smokers to stop, since this will by itself probably have a greater effect on mortality than any anti-hypertensive drugs. Not only is the patient at his most receptive at this time, but it is also very difficult to impose the discomfort of nicotine withdrawal concurrently with the side-effects of treatment.

Intravenous pyelography as a routine investigation may be useful, but difficult to arrange when so few British general practitioners have direct access to this procedure. Referral of all cases to hospital physicians merely for this to be done is wasteful and absurd, and easily leads to confusion of responsibility in subsequent management. In this situation it seems best to do IVPs on all men with bacteriuria (dipslide colony count $\gtrsim 10^5$), and women with persistent or recurrent bacteriuria, all those with frank gout or hyperuricaemia, and all those with a history suggestive of stone or other renal disease.

The following checklist summarises the standard initial investigations suggested:

History

Headache
Facial paresis and/or dysphasia, however transient
Breathlessness in terms of specific local function, e.g. " How many times do you have to stop when going up Market Hill ".
Anginal pain, in terms of specific local function.
Claudication, in terms of specific local function.
Muscle weakness.
Nocturia x?
Coital frequency (baseline for subsequent evaluation of side-effects).
Antidepressant drugs (for drug interaction).
Oral contraception.
Specific current occupation.

Physical examination

BP seated; at least three preferably six times before any treatment started.
Retinoscopy, under dilatation
Corneal arcus, under 40
Both wrist pulses, pulsus alternans
Carotid, femoral, posterior tibial and dorsalis pedis pulses
Auscultation for triple rhythm
Auscultation for lung crackles
Body weight for height

Urine

Protein and glucose
VMA (24-hour urine preserved with 10 ml concentrated HCl)
Dipslide for bacteriuria

ECG

For LV strain
T waves for hypokalaemia
Baseline for future infarction

Blood

Plasma electrolytes
Urea and/or creatinine
Cholesterol (not fasting) and lipid profile
Uric acid

X-rays

PA chest for heart shadow
IVP in selected cases

Nearly all of these can be initiated by a nurse with enough specific training.

Aims of treatment

Short of organ damage, hypertension is symptomless, so in the great majority of cases, managed in general practice, the relief of symptoms is not a rational aim of treatment. In fact most treated cases will have symptoms caused by treatment itself, and they must from the outset be warned that this may occur.

To accept fortuitously presenting symptoms of headache, giddiness and so on as caused by high blood pressure, and relieved by its fall, is an opportunist course on which lasting mutual confidence between doctor and patient cannot be built, and patient education cannot begin: both of these are essential and should not be sacrificed for spurious short-term gains.

The aim of treatment is to prevent organ damage by sustained reduction of mean systolic and diastolic pressure for the rest of the patient's life. The nature of this organ damage is different in treated hypertension from the untreated condition. The latter was shown well by Smith and others' necropsy series of essential hypertension without retinopathy. Of 200 cases, 47·5 percent died of causes unrelated to hypertension, 23·5 per cent of cardiac failure, 18·5 per cent of coronary disease, 13 per cent of stroke, and 2·5 per cent of renal failure (Smith, Odel and Kernohan, 1950).

There is now no doubt that treatment of men with diastolic pressures of 90 and over, sustained over five days in hospital, reduces both mortality and morbidity very substantially (Veterans Administration Co-operative Study Group, 1970). In 380 hypertensives with diastolic blood pressures averaging 90 to 114 randomly allocated to treated and placebo groups, hypertension-related morbid events were four times as frequent, and related deaths were 2½ times as frequent in the placebo as in the treated group. The reductions were in deaths from congestive heart failure and stroke, not in coronary heart disease, and the degree of benefit was related to the initial level of blood pressure.

The dramatic reduction in deaths from stroke, cardiac and renal failure, and the equally striking lack of effect on deaths from ischaemic heart disease, have been confirmed in all major hospital series (Hamilton, 1966: Breckenridge, Dollery and Parry, 1970), but the very high morbidity in the untreated group makes it clear that the Veterans Administration patients had more severe hypertension than a screened group would have, with an apparently similar diastolic definition.

The possibility remains that treatment in young men of 30–45 may have some preventive effect on ischaemic heart disease (Werkö, 1971) and that β-adrenergic blocking drugs may prevent deaths from post-infarction arrhythmias, without increasing deaths from pump failure: so far there is no conclusive evidence of either of these possibilities, but there have not been enough young men in most series to reach any firm conclusions.

On women we have little evidence. The really big difference between the sexes in the natural history of hypertension lies in the male excess of ischaemic heart disease;

the male excess of stroke under 64 is small. One might then expect that women would differ little from men in their need for and response to treatment. The results of Hamilton's relatively small controlled series (Hamilton, 1966) are consistent with this, but there is an urgent need for more data on women before firm conclusions can be reached.

In terms of outcome then, our aim is to prevent stroke, retinal damage, renal damage, and ventricular failure and to ensure that any treatment we use shall not increase the risks of a fatal outcome in the likely event of myocardial infarction. Much more evidence is urgently needed on the positive or negative effects of β-adrenergic blockers in this last respect.

Cerebrovascular accidents

Fears of precipitating cerebrovascular accidents by reduction of blood pressure seem to be unfounded, though it should not be too rapid (Strandgaard *et al.*, 1973). The reduction of stroke seems to be because of the elimination of haemorrhage from the Charcot micro-aneurysms that are so common in those with high pressures (Russell, 1963; Cole and Yates, 1967), probably produced directly by the effects of high pressure in the arterial lumen. The eventual disappearance of these after sustained fall in pressure may account for the low incidence of stroke in patients treated for more than four years (Breckenridge, Dollery and Parry, 1970).

The incidence of cerebral thrombosis is probably not reduced, but Barham Carter (1970) and Beevers and others (1973) showed that stroke-survivors are much less likely to suffer a recurrence if blood pressure is controlled, and Meyer and others (1968) showed a small *increase* in cerebral bloodflow after reduction of pressure in stroke-survivors. However, Barham Carter found that those with poor or intermittent control did even worse than those who were not treated at all.

Although blood pressure reduction probably does not reduce the incidence of mortality of myocardial infarction, hypertensives are such a high risk group that other preventive measures of proved effectiveness should be applied. Unfortunately very few measures *are* of proved effectiveness.

To stop patients smoking is of real value and deserves designation as a specific aim in treatment. Reduction of serum cholesterol may reduce infarction rates, though the small reduction in mortality in patients with angina by the use of clofibrate (Group, 1971; Research Committee, 1971) was not associated with falls in cholesterol. Substantially altered diets certainly do no harm in the small minority of patients prepared to adhere to them, and are effective in reducing cholesterol and triglyceride (Evans, Turner and Ghosh, 1972) but the evidence of their effectiveness is not, in my opinion, sufficient to justify much pressure on the more casual majority. Weight reduction (a marginal risk factor for coronary heart disease) is discussed below. There is so far very little evidence that regular physical exercise short of exhaustion significantly influences either coronary heart disease or hypertension.

These aims of outcome must be translated into aims of process—the measures of treatment in real terms. They are simple: a vast weight of evidence supports the view that the harmful effects of hypertension derive almost wholly from sustained raised pressure in the arterial lumen, and are reversed by its sustained reduction, so the question resolves itself into how much reduction, and how often it is verified.

Degree of control achieved

A rational approach to the first of these must relate to the reductions achieved in the controlled trials on which our planning should so far as possible be based. Hamilton (1966) defined good control as a diastolic pressure consistently below 100, fair control

below 110, and poor control consistently over 110, in cases with minimum pre-treatment pressures of 110 recorded at least three times.

The first Veterans Administration Study (1967) achieved average reductions of 43 mm systolic and 30 mm diastolic pressure in cases with pre-treatment mean diastolic pressures of 115–129, implying a mean treated diastolic pressure of 85–100.

The second Veterans Administration Study (1970) achieved average reductions of 27 mm systolic and 17 mm diastolic pressure in cases with pre-treatment mean diastolic pressures of 90-114, implying a mean treated diastolic pressure of 73–97 mm. Reductions short of these targets were proportionally less effective but still worthwhile (Taguchi and Freis, 1974). Zacharias (1972) agreed with Hamilton in aiming at treatment levels of diastolic pressure consistently below 100 mm; this has been my own practice, and it seems generally acceptable. It should be borne in mind that this is a good deal less stringent than the Veterans Administration studies on which so much of our policy depends, but it is unrealistic to expect patients to tolerate the substantial side-effects of reduction to the 70s and 80s, that occur in most cases.

Verification of treatment levels must be made at least once every three months for the rest of the patient's life when the treated pressure is satisfactory and stable. Where pressures are unstable or not satisfactory, patients should be seen weekly until control is achieved.

Selection for treatment

If we accept that self-selection by presented symptom does not identify cases of hypertension effectively, the selection of cases must depend on the doctor, even more than in other diseases. His selection should be based on a policy based on evidence, and cases not selected for treatment should still be followed up at least once a year in case this decision needs to be revised.

Treating the elderly

The choice for or against treatment in each case should be the result of balancing the risks and disabilities of treatment against the risks of inactivity. This balance rarely favours starting treatment in the elderly, unless there are acute symptoms such as left ventricular failure indicating an immediate need for reduction of blood pressure. Systolic hypertension in the elderly (systolic pressure over 160 and diastolic pressure *under* 90 sustained over three readings) is relatively rare (2·7 per cent). Colandrea and others (1970) found a 50 per cent of excess of deaths compared with controls with systolic pressures under 140 and diastolic pressures under 90. Systolic hypertension is generally thought to arise from reduced vascular elasticity from atheroma, and this of course carries risks of its own that will not be reversed by any treatment. Evans (1965) in a post-mortem series of 29 mental hospital patients over 65, found a mean fall in blood pressure of 20 mm during the decade preceding death. There was a closer correlation of degree of atheroma, particularly in the cerebral arteries, with the initial pressures than with those recorded during the two years before death. Barham Carter (1970) found that the reduced mortality in younger stroke-survivors associated with effective antihypertensive treatment was absent in those over 65, but the number of elderly patients was too small to reach a conclusion one way or another. Although evidence is incomplete on treatment of diastolic hypertension initiated over 65 (W.H.O., 1971) there is so far little in its favour despite the large number of cases apparently being treated. It seems likely that the many women and the small number of men who can survive beyond 65 with diastolic pressures over 120 are already a selected group of exceptional toughness, for most of whom antihypertensive drugs represent pseudo-scientific meddling rather than effective concern for their health. Where treatment and control are already

well-established there seems no reason to stop just because the patient reaches his 65th birthday, but even then a cautious reduction of dosage should be attempted and the need for treatment reviewed annually.

Other exclusions from treatment

The other exclusions are those with a complicating concurrent illness or disability, and those with intervening social problems or personal attitudes to treatment. Patients who suffer concurrently from several serious disorders, each requiring permanent drug treatment, present exceptional difficulties. Combinations of diabetes, gout, asthma, and rheumatoid arthritis are common examples. Although treatment of marginal hypertension should certainly be avoided for these people, because of the large additional burden of iatrogenic risk, in the main these difficulties simply have to be faced and overcome. Some of the most difficult cases of this sort emerge from teaching hospitals, where a wealth of polypharmacy may be accompanied by total failure to educate the patient in the nature of any of his diseases or treatments.

Social and subjective problems are the commonest difficulties, but anticipation of them rarely justifies exclusion before even trying. Alchoholism makes treatment very difficult indeed, but there seems no reason why it should not be attempted. I have excluded cases of severe mental defect and of schizophrenia, but only because of problems in educating these patients which I lacked the time or imagination to face. We are fortunate in our freedom from the constraints of prescribing costs; in an American series 33 per cent of dropouts from treatment were because of financial hardship (Caldwell *et al.*, 1970). Finally there is the patient who does not want treatment. Of my 34 screened hypertensives, four refused any treatment at all, and of course this view should be respected with a friendliness sufficient to keep the door open should they change their minds later on.

Those who will be grossly undependable in drug-taking even after prolonged effort are difficult to forecast. Zacharias (1972) found that 16–20 percent of his patients persistently failed to take drugs as prescribed, although he was running " a special department where great trouble is taken to simplify the regime and explain its purpose ". I have found about the same proportion of defaulters in those who accepted treatment, but many of these were patients whom I expected to be easy, and some of the easy ones I thought would be difficult. Persistently intermittent drug-takers should be lapsed from treatment, because of the evidence of higher risks in intermittent than sustained hypertension.

It should be remembered that the Veterans Administration trials (1967,1970) were highly selective; they excluded nearly half the patients who originally met their criteria for inclusion in the study, because of unreliability in either attending for follow-up or in taking their treatment.

Treatment other than antihypertensive drugs

These fall under two headings, the control of obesity and the control of psychosocial factors: both present exceptional difficulties and few rewards.

The association of hypertension with obesity has already been discussed. It begins very early: Christakis and others (1968) found a mean diastolic pressure of 85 or more in 20 per cent of obese schoolchildren aged 10–13, compared with nine per cent of the non-obese.

Fletcher (1954) studied pressures in a group of obese women over a period, and found that of the hypertensives (mean pressure 196/116), those who lost a stone or more in weight had a 17 per cent fall in systolic pressure, and those who lost less than a stone stayed the same. Normotensive obese women who lost a stone or more in weight showed

a four per cent systolic fall. The serious literature on control of blood pressure by weight reduction is very sparse, and probably reflects both the relative neglect of this method, and its great difficulty in most cases.

This neglect has been shared by me, exhausted by many years of relatively unsuccessful attempts to help patients to lose weight. Though highly motivated doctors can certainly achieve worthwhile results, they are not dramatic. Craddock's (1973) beautiful study of the management of obesity should be known and used by us all, and gives a thorough account of the weapons available in this usually disappointing struggle. Mutual-aid groups on the lines of weight watchers may be more successful, at least in women: such a group has functioned at our health centre for the past two years and has shown better results than individual personal advice.

In the presence of hypertension advice on diet may be more effective, particularly if the patient can see it as a possible alternative to lifelong drugs, as will often be the case with diastolic pressures under 110. Craddock found that weight reduction achieved in the first three months was a fair guide to subsequent success, and serious attempts at dieting should precede rather than coincide with starting antihypertensive drugs. Obesity can be treated by drugs, exercise, and diet.

Drugs used for obesity

In general drugs are only transiently effective if they are not preceded by a period of effective dieting with increased exercise, during the initial phase of high motivation: drugs can be useful as this wanes (Craddock, 1973). The choice lies between diethylpropion ' Apisate ', ' Tenuate ', or the more commonly used sustained-action ' Tenuate Dospan,' and fenfluramine (' Ponderax.') Diethylpropion has a very small CNS-stimulating action, but there have been cases of addiction. Fenfluramine tends to have a depressing effect and many patients feel unwell on it: combined with antihypertensive drugs this could lead to drug defaulting. In other respects it offers some advantages; it slightly increases the antihypertensive effects of methyldopa, guanethidine, bethanidine and reserpine (but not debrisoquine), independent of weight loss. It does not antagonise β-adrenergic blocking drugs. It should be withdrawn slowly, as abrupt withdrawal can provoke severe depression. It is noteworthy that of 832 overweight doctors who had treated themselves for obesity, only seven per cent had used drugs at all (Yudkin, 1968).

There is much evidence that obesity is inversely related to exercise and energy expenditure, but some doubt about the relations of cause and effect. Regular exercise can reduce weight more effectively and certainly more safely than total fasting in hospital, and though there is no conclusive evidence of a beneficial effect on hypertension or in preventing myocardial infarction, there is certainly no evidence of any harm. This aspect more than any other is one that must be tailored imaginatively to the individual patient. It has been neglected not only by doctors, but by government and local authorities, who must be ultimately responsible for expanded and more attractive facilities for mature and middle-aged sport.

Psychosocial factors

The control of obesity, however, is simplicity itself compared with the control of psychosocial factors. In the first place we do not know what those factors are with any useful precision: and if we did, there is little reason to think anything effective could be done about them. There can be no greater cause of misery and ill-health in Britain than the shortage of housing and the plight of homeless families, and no one can claim to be unaware of it. Despite this the number of houses available to rent has fallen steadily for the past ten years. The situation may or may not cause hypertension, but certainly produces suicide, divorce, and every sort of bitter unhappiness: homeless families are not helped by gratuitous advice to obtain a home.

The other possible factors are similar. Excessive overtime and shiftwork, and works line-speeds in particular, must be prime suspects as possible causes of hypertension, as well as other kinds of exhaustion and damage that may be more immediately important. When we get serious research and specific results in this field, the battle can begin to get primary prevention implemented: we must hope that doctors will by then be prepared to fight harder than they have in the past for a humane social environment.

The most intimate knowledge possible of the patients' working, family, and social life is needed for effective delivery of care: a little of this knowledge may also be useful to indicate areas of possible causation that might be changed. However, there are very few serious sources of stress that are presently removed on the initiative of doctors. This situation could change if we had an effective personal industrial medical service independent of managements, and a more precise knowledge of causal psychosocial pathways: both of these seem equally remote for the present, but in the long run such primary prevention is likely to make more medical and economic sense than the secondary prevention that inevitably dominates present discussion.

Antihypertensive drugs

Like most fields of treatment, hypertension both suffers and benefits from the rapid introduction of a large number of drugs in a short time. No general practitioner can be proficient in more than a few of them, and those hospital specialists who are, do not have experience of the problems of primary care. They are discussed here in the order of preference in which I used them during the past five years: the sequence of groups was probably agreed by many if not most specialists in the treatment of hypertension, but the choice of individual drugs is necessarily personal. The groups are: thiazide, diuretics, β-adrenergic blocking drugs, clonidine, methyldopa, ganglion blocking drugs, and has an adjuvant only, reserpine.

With the exception of the thiazide diuretics, all of these must be titrated against blood pressure, a relatively unstable variable to be interpreted with caution: the increments of dosage must be small, appropriate to the poor discrimination of our observations. Changes in drugs should be made singly, and the number of drugs kept to a minimum, even at the cost of less-than-ideal control. The most wonderfully devised drug combinations cannot be effective if the drugs are not taken.

Some understanding of the mode of action of the different groups is essential, if only because of the risks of drug-interaction in lifelong therapy, and effects on other chronic diseases, but none of the drugs now in use were anywhere near completely understood when they were introduced, and few of them are now. Any drug that lowers blood pressure and does not kill many people will be marketed, and much of its initial pharmacology will consist of informed guesses as to how it works. Since sudden death is in any case common in the group under treatment, serious risks to a minority of patients could go undetected for a very long time, particularly if they are associated with coronary heart disease or its precursors.

Thiazide diuretics

These are often effective in reducing mean diastolic pressure by 10 or 15 mm by themselves, and can be combined with any of the other drugs to reduce their dosage. Chronic overdosage can certainly produce potassium depletion, but this seldom occurs at optimum doses. The possibility of potassium depletion must always be borne in mind, particularly as its symptoms resemble the side-effects of most anti-hypertensive drugs.

It is important to understand that the hypotensive effect of thiazides is independent of their diuretic effect; it is probably an effect on peripheral vascular resistance rather than blood volume or sodium, and does not increase above a very low threshold dose

(Cranston *et al.*, 1963). There is little to choose between the different thiazides: bendrofluazide 2·5 mg twice a day is a good standard dose, and cheap. Frusemide has little hypotensive effect and is quite unsuitable because of its rapid diuretic action.

Thiazides reduce insulin secretion, and can in this way upset the control of diabetics and probably precipitate diabetes in susceptible subjects. However, maturity-onset diabetes is usually associated with peripheral insulin resistance rather than islet-cell failure, and the danger of adding to the already high risk of ischaemic heart disease is probably small (though so far as I know, as yet unmeasured). A common but neglected side-effect is hyperuricaemia: this can precipitate podagra, and may cause renal damage.

The propriety combination with potassium and reserpine 'Salupres' has never been validated by controlled trial, but in my limited experience substitution of thiazide alone for this combination often leads to loss of control, and it was probably useful. The potassium in the formulation is useless, and it is presumably the reserpine that is effective.

β-adrenergic blocking drugs

These drugs are well tolerated, causing occasional tiredness but fewer symptoms than any of their precursors. They are contra-indicated in asthma (reversible airways obstruction), but not in obstructive chronic bronchitis and emphysema. Where there is doubt about this a flow-meter must be used for control. The whole group—propranolol, oxprenolol, pindolol, alprenolol, practolol—has been succinctly, comprehensively and accurately reviewed in the *Drugs and Therapeutics Bulletin* of 26 April, 1974. The group is multiplying in the normal manner of profitable and fashionable drugs, and may become as incomprehensible as the antidepressants and tranquillisers.

There is more experience in Britain with propranolol than any other of these drugs. It seems to differ little from oxprenolol. Practolol has a more selective effect on β-receptors in the heart and can be considered for use in asthmatics, but it has less antihypertensive effect than propranolol (it is not promoted as an antihypertensive drug) and has recently been found rarely to cause reversible psoriasis-like lesions on the palms, soles and knees, occasionally associated with impairment of tear flow leading to keratitis.

The whole group reduces cardiac output and slows the heart: in fact the slow pulse is a valuable independent measure of consistent dosage. Low output failure is not an important risk in asymptomatic hypertensives under 64, unless there is some other cause of heart failure, and even then β-blockade can be used cautiously after digitalisation. There is a serious risk of low output failure after myocardial infarction (Multicentre trial, 1966). Propranolol may increase levels of transaminase without implying myocardial damage (Wilkinson, Luetscher, and Goldman, 1971). It can be used effectively in twice daily dosage because though its plasma half-life is short, its metabolites are pharmacologically active and have a much longer half-life (Hansson *et al.*, 1971). There is a great variability in response: it is essential to start with a very small dose (40 mg twice a day), to add daily increments of 40 mg in the early weeks according to response, and not abandon the drug until limited by side-effects (usually bradycardia less than 50 a minute, or excessive tiredness) or the absence of useful response after three months of treatment. Some patients respond very late, but very well.

In Zacharias' (1971) series of 309 patients observed over five years, more than 30 per cent needed over 600 mg a day, more than 15 per cent needed over 1,000 mg and a few required up to 2,000 mg (this is very cumbersome with the present maximum tablet strength of 80 mg, and there is an urgent need for 250 mg tablets). On strict criteria, 219 patients had good control, and 19 fair control, on propranolol and a thiazide diuretic, and a further five had good control, and 17 fair control, on propranolol, a diuretic, and some other antihypertensive drug. Including all these methods of treatment, only four

(two per cent) had poor control. Sixty-six people reported tolerable side-effects, and 35 severe side-effects, and in 26 of these treatment was stopped. The commonest complaint was cold hands and feet, the most important bronchospasm and impotence—but this was much less common than with any of the other antihypertensive drugs.

β-blockade has the additional advantage of being an effective treatment for angina pectoris, and there is some evidence that it may reduce its mortality (Amsterdam, Wolfson and Gorlin, 1968). There is suggestive but incomplete evidence of a reduction in the mortality of myocardial infarction in hypertensives treated by β-blockade (Shell and Sobell, 1974).

There are theoretical reasons for preferring β-blockade to most alternatives. With much greater precision than ganglion-blockade, it isolates the cardiovascular system from the pressor response to intense emotional stress in terms not only of hypertension, but of plasma-free-fatty-acids and blood glucose (Taggart and Carruthers, 1972). If convincing evidence can be found of a sustained effect on blood pressure by psychosocial factors, this would seem one sensible way to treat it. However, we seldom improve much on nature, and transient pressor responses to catecholamine release are clearly of physiological value in normal people—who can only be arbitrarily divided from hypertensives.

Clonidine

Clonidine differs from all other antihypertensive drugs in its mode of action, which is still poorly understood. Heart-rate and output fall as with β-blockade, but both this and its hypotensive effect are probably the result of a direct action on the vasomotor centre in the brain stem. With long-term use there may also be reduced peripheral vascular responsiveness to catecholamines and angiotensin. The usual starting dose is 200 micrograms, most cases are controlled by doses of not more than one mg a day, and the highest doses used have been about five mg a day.

It tends to make patients very tired, also causes a dry mouth and constipation, and on the whole I have found it much less well tolerated than β-blockade, but it is useful in the presence of asthma. It is also effective in preventing migraine, probably more consistently than propranolol. It is contraindicated in depressed patients and psychotic reactions have been reported (Ng *et al.*, 1967). Long-term tolerance with loss of control seems fairly common, and there can be sudden dangerous rises of pressure on withdrawal due to accelerated catecholamine release; it must always be tapered off slowly. Its future place is likely to be small.

Methyldopa

As the first really successful antihypertensive drug to supersede ganglion-blockade, methyldopa has been very popular, there are still many patients well stablilised on it, and there is little need for them to change. It is no longer a first-line drug, because at least three-quarters of patients feel so tired on effective doses. In my experience (mainly with quite heavy manual workers) dosage in excess of two grams is always intolerable, and over 1,250 mg usually so. Response is fairly rapid and dosage titration is easy. It seldom causes impotence, but more frequently a lethargic indifference that is almost as bad. Between ten and 20 per cent of patients eventually develop a positive Coombs' test and a few of these develop haemolytic anaemia: both are reversed by withdrawal. Liver damage occurs rarely, but can be fatal. It usually occurs during the first three months of treatment, and is not related to dosage (Toghill *et al.*, 1974). Between one third and one half of patients show no useful hypotensive response.

Its mode of action is still not understood: it may disturb the metabolism of noradrenaline precursors to produce the less potent metabolite methylnoradrenaline. It has to be given three or four times a day.

Ganglion blockade

The ganglion-blockers include guanethidine, bethanidine, and debrisoquine. All of them have the considerable disadvantage of producing marked postural hypotension, which can be quite dangerous (by falling about) on rising in the morning, even if pressure remains high later in the day. They produce further extreme falls whenever any large vascular bed dilates—the splanchnic area after a meal, muscle vasculature in exercise, the skin in hot weather—to produce faintness and syncope that can be very disabling. Venous sympathetic innervation is affected as well as arterial, producing pooling in the legs and abdomen, reduced venous return, and an exaggerated Valsalva response when coughing or straining on the toilet. They all cause failure of ejaculation, but not, as a rule. of orgasm.

Despite all these serious disadvantages, they are still useful in severe hypertensives who are for some reason refractory to the newer drugs, and whose level of pressure or symptoms makes treatment mandatory. They make heavy demands both on the patient and the care team.

I have only used guanethidine. This must be started at ten mg a day, with increments added at not less than weekly intervals, as the full effect of each dose is only reached in seven days. It can be given in one dose a day. The main problems are postural hypotension and diarrhoea (about two-thirds of patients) which can be controlled with codeine phosphate tablets. Bethanidine produces less diarrhoea, more postural hypotension, and has a short action. Debrisoquine seems to lie between the other two in all these respects; it also depresses urinary vanillomandelic acid output and can thus conceal evidence of phaeochromocytoma.

The antihypertensive effect of all the ganglion-blocking drugs is antagonised by tricyclic antidepressant drugs and by some other psychotropic drugs, including chlorpromazine. This interaction can lead to very severe and unsuspected hypertension. Patients on ganglion-blocking drugs must have standing and lying pressures recorded routinely, as well as sitting pressures.

Reserpine

For a long time reserpine has seldom been used except as an adjuvant. In this role it was useful, producing good control in patients who previously appeared completely refractory to treatment. The serious suspicion of a carcinogenic risk will probably eliminate it from use in this country at least (Boston program, 1974). Depression is rare in the doses of 0·1 to 0·5 mg daily that are now normally used. The proprietary combination ' Salupres ' contains 0·625 mg and in the usual dosage of two to four tablets a day quite commonly causes depression.

It seems likely that β-blockade is likely to dominate the antihypertensive drug field more or less completely for the next few years, with or without thiazide as an adjuvant. Because of the enormous differences in dosage required with nearly all these drugs, a wider range of tablet strengths is needed to encourage a greater ultimate boldness (preceded by prolonged and cautious incremental titration) before switching from one drug to another.

It is also important to remember the more mundane causes of treatment failure considered in the next section.

Management of follow-up

The follow-up of a whole population is far more difficult than its initial screening. It falls naturally into two parts: diagnostic follow-up, and treatment follow-up.

Diagnostic follow-up consists of blood pressure measurement of:

(1) All in-migrants to the defined population,

(2) All those within the population who cross the lower limit of the defined age-group (30–64),

(3) All those diagnosed as hypertensive in the original cohort, who are not under treatment (for whatever reason), annually.

(4) All those within the original cohort with diastolic* pressures sustained within 15 mm of the defined cutting points over three readings *once in every two-year span.*

(5) All the rest of the original cohort, *once in every five-years span.*

Organisation for this task depends on a suitable flagging system showing those outstanding for these procedures, so that they can be picked out by ancillary staff during ordinary consultation visits. We have tried using variously coloured cards inserted into the patients' records, sticking up about two inches above the top of the record envelope: these are not durable enough and soon get bent over during record sorting. The best material is resin laminate in strips about three inches wide, available in many colours, very durable, and which will accept felt pen writing (or tacky plastic labels to accept ball-point pen). When the flagged procedure has been completed, the flag is removed. An alternative organisational method for accumulating screening data has been described by Coope (1974), with a good design for a follow-up clinic.

Treatment follow-up

Treatment follow-up involves the collection of a standard minimum set of data in all treated patients at maximum intervals of three months. A list of all treated hypertensives should be maintained and reviewed once every quarter, and those who have not consulted should be followed-up by the attached health visitor or other ancillary staff.

It is essential that hypertensives should not be given repeat prescriptions by lay staff without medical control. This can and does go on for years in some cases. A colleague found a woman with a diastolic pressure of 160 who had been on repeat prescriptions for reserpine for the previous six years, without any clinical supervision. Prescriptions for antihypertensive drugs should be exactly calculated to give a week's treatment in hand at the time of the next appointment, which may result either in very large or very small prescribed quantities. This can lead to difficulties because of fixed-rate prescription charges, and can be avoided by encouraging all hypertensives to obtain the Department of Health's annual season ticket for prescription charges.

Intervals between appointments depend on the stability and level of blood pressure readings. Difficulties can arise when patients have to attend week after week for the incremental titration of dosage, when, as in so many cases, the effective dose proves to be very high—possibly 15 times the initial dose, implying 15 visits over more than three months. One solution to this problem, which I have not yet used but seems quite rational and practical, is to lend the patient an electronic sphygmomanometer to make and record his own readings, and within certain limits, his own dosage adjustments.

At each visit, there is a checklist of minimum data that should be collected on every patient: systolic and diastolic pressures seated pulse rate, body weight, current smoking, symptom checklist—administered or self-administered, current drugs, both related and unrelated to hypertension.

*Categorisation in terms of diastolic pressure alone has little to commend it other than tradition, as prognosis is more closely linked to systolic pressure at all ages, according to the Framingham data, and systolic pressures are easier to measure. However, traditions are extremely powerful, and there are many others more harmful than this. When we act rationally in terms of diastolic pressure, it will be time to shift to the use of systolic pressures. Calculation of mean pressures is not practical politics. (Kannel, 1970; Stuart *et al.*, 1974).

In addition there must be a review of variables peculiar to that patient, arising from symptoms or signs found on previous visits or the nature of medication (e.g. standing or lying pressures in those on ganglion-blockade). The marker flag inserted in the record envelope should have these items entered on it, so that both the minimum set and any such extra data are recorded in the notes by the ancillary staff, *before* the patient sees the doctor.

It is a good practice to ask the patient to bring all his tablets, in their containers, at every visit. I had one whose poor response to treatment was the result of taking ten tablets of bendrofluazide daily with one tablet of methyldopa: this situation was recognised because of his severe nocturia.

The accumulation of masses of data over many years presents serious problems of recording and retrieval. These are for the time being aggravated by the small size of the present National Health Service record envelopes, and the absence of standard flow-sheets: the A4 folder (eventually to become standard but still unpardonably delayed by the Department of Health and Social Security) contains a graph-ruled sheet suitable for graphic representation of flow variables. This is particularly valuable in ensuring systolic as well as diastolic control, and permitting rapid review of the entire course of treatment.

The aim of treatment follow-up is not merely to measure and respond to changes in the main variables related to outcome—blood pressure, weight and smoking: no less than this is the task of retaining compliance with supervised treatment for a lifetime.

It is absurd to imagine that hypertension in relatively large numbers of people can be safely supervised without planning, organisation, delegation of work, and structured collection of data: but it is no less absurd to think that the creation of such machinery will of itself create a caring environment for the symptomless patient, that will augment and consolidate his initial motivation to improve the chances of a long and healthy life by submitting to a life-long discipline. That discipline is essential, but it must be made tolerable in every possible way, particularly by encouraging friendliness, frankness, mutual understanding and continuity in care.

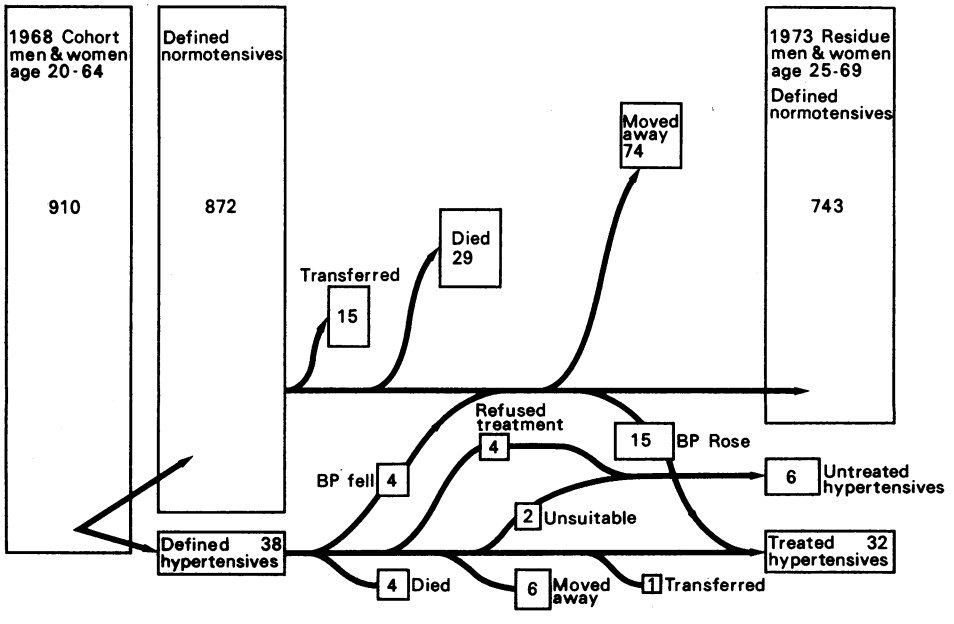

Figure 2
Progress of a whole population continuously screened for hypertension 1968–73.

A study of the hypertension division of the Henry Ford Hospital at Detroit (Caldwell *et al.*, 1970) has shown the disastrous effects of neglecting this. A cohort of 76 patients first treated in 1961 was followed: by 11 months 50 per cent had lapsed from treatment. By five years 74 per cent had lapsed, nine per cent had died, and only 17 per cent remained on treatment. This may be compared with the flow diagram in figure 2, in Glyncorrwg: of a cohort of 34 treated hypertensives followed for five years, 12 per cent lapsed, 12 per cent died, 22 per cent moved or transferred, and 53 per cent remained on treatment. Figure 3 shows that this continued supervision was with one exception successful in reducing both systolic and diastolic pressures.

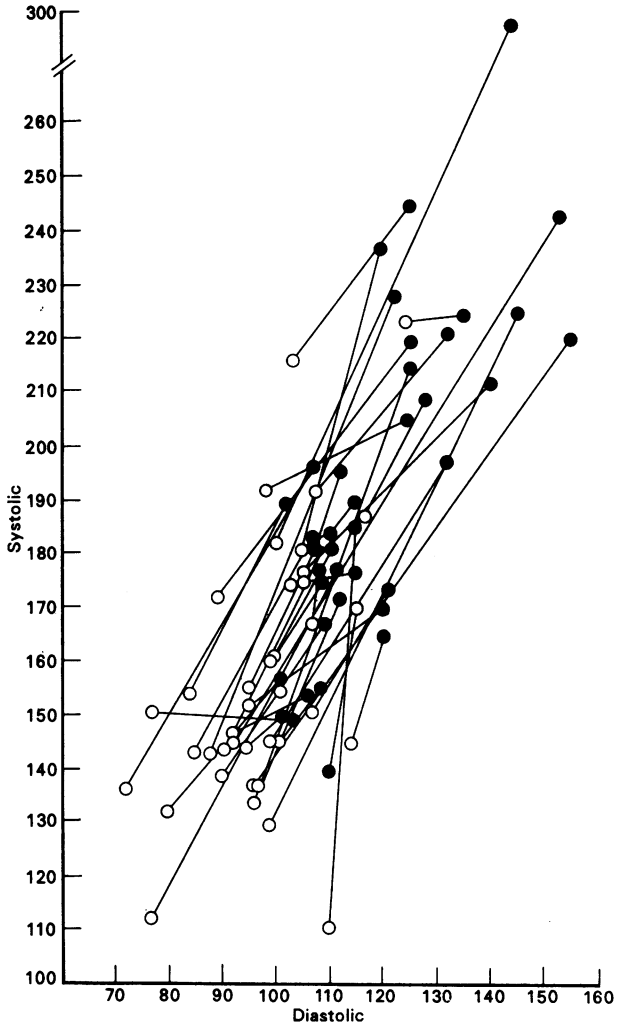

Figure 3
Means of highest and lowest blood pressure recorded in year preceding treatment and in second year following treatment ●, in 26 male and 14 female hypertensives—1968 screened cohort.

With all regimens there remains a variable proportion of unresponsive patients. These include:

(1) Those refusing treatment in the absence of symptoms (or occasionally in their presence), either overtly, or covertly (by non-compliance).

(2) Those exposed to an intractable background of social or psychological stress; these are not easily predictable, and can usually manage with sufficient imaginative support and a flexible programme of treatment.

(3) Those with multiple symptoms rightly or wrongly attributed to drugs; this group can only be clearly defined by using a standard initial period of placebo treatment.

(4) Patients who cannot take any drug regularly; this can be minimised by twice daily dosage regimens wherever possible.

(5) Patients with serious occupational problems—usually exceptionally heavy manual work.

(6) Patients with an apparently primary drug resistance, who need large doses of multiple drugs.

The problem has been well discussed by Zacharias (1972). He found 16–20 percent of his patients were persistent drug defaulters. He found true drug tolerance (rising effective dosage) to be rare; more frequent was excessive prescribing to compensate for the poor response to treatment of a patient who is simply not taking the tablets prescribed.

All of these problems must be explained to and understood by the ancillary staff who deal with the patients, helping them to the view that a lapsed patient represents a treatment failure second only to cerebrovascular accident, for which we, rather than the patient, must accept responsibility. A system that cannot command 80 per cent compliance from patients is clearly not serving their interests, and should be changed.

The future

Speculations on the future are useless, except in so far as they modify our present actions.

It seems certain that all future developments will depend on a framework of data collection and recall that can define various grades of hypertensives as a risk group within whole populations, and probably risk-subgroups defined on variables other than sustained blood pressure alone. Development of this social and organisational framework is an urgent task, if new and validated methods of treatment are to be applied as they become available to all those who can benefit from them. Habitual use of systolic rather than diastolic pressures may give us more effective definitions; this may be as difficult to bring about as the still incomplete recognition of blood pressure as a continuously distributed variable.

The framework for delivering care described in this paper could, with relatively small modifications, accept the advances in care that can presently be conceived, providing the definition of high blood pressure (in the sense of a disorder to be rectified by treatment) does not extend so widely that logistics alone would defeat any system of total care. The Baltimore study (Apostolides *et al.*, 1974) found 37 per cent of the black city-centre population eligible for treatment on a definition of diastolic pressure sustained at 95 plus (40 per cent for men, 33 per cent for women), at ages 30–69. Have we the right to extend universal treatment at lower risk levels of pressure, when we have nowhere suceeded in delivering effective care to all of those, or even nearly all of those, at high-risk levels? The first step should precede the second, because otherwise that second step is likely to be misconceived. This point may be more difficult to understand in a system of care where treatment at low-risk levels has long been mandatory in privileged minorities of the population, to the financial advantage of both doctors and pharmaceutical manufacturers. The assumption that privileged treatment is necessarily better treatment is likely to be wrong. It is doubtful that any condition with a 37 per cent prevalence will ever justify lifelong drug dependence.

The appropriate operational definition for hypertension in the diastolic range 90–109 in otherwise healthy people should emerge from the large Medical Research Council multi-centre controlled trial of treatment being directed by W. E. Miall and now in its initial phase. Evidence from this should precede any general move to extend treatment in the diastolic range below 105 (fifth phase) or 110 (fourth phase). At present the evidence from the U.S. Veterans Administration study is being misused to support projections for mass treatment that go far beyond its evidence or the intentions of its authors, and which are unlikely, if not impossible, to be carried through to effective long-term care and follow-up.

It is more likely that other risk variables will be found, particularly in relation to cerebrovascular disease, or that the measurement and characterisation of blood pressure behaviour will become more sophisticated and depend on more measurements, before lifelong treatment is begun.

Figure 4 shows continuous five-minute plots of blood pressure throughout 24 hours in two normotensive patients studied at Pickering's unit in Oxford (Bevan *et al.*, 1969). Such measurements, using non-invasive techniques, may well become possible at primary care level, now that computers and micro-miniaturisation offer us the means to record and analyse vast amounts of data in a short time.

It is also possible that when more is known of the environmental causes of hypertension, forms of physiological re-education may become possible, that do not depend on drugs at all. Attempts at re-education by bio-feedback or other means, have so far been inconclusive, but quite encouraging, (Benson *et al.*, 1974; Brener and Kleinman, 1970; Patel, 1973; Miller, 1969; Katkin and Murray, 1968). Those who sneered at the projections of the Royal Commission on Medical Manpower, suggesting one doctor per 100 population by the year 2,000, should consider how advances in treatment of this nature can be delivered with fewer physiological engineers than this.

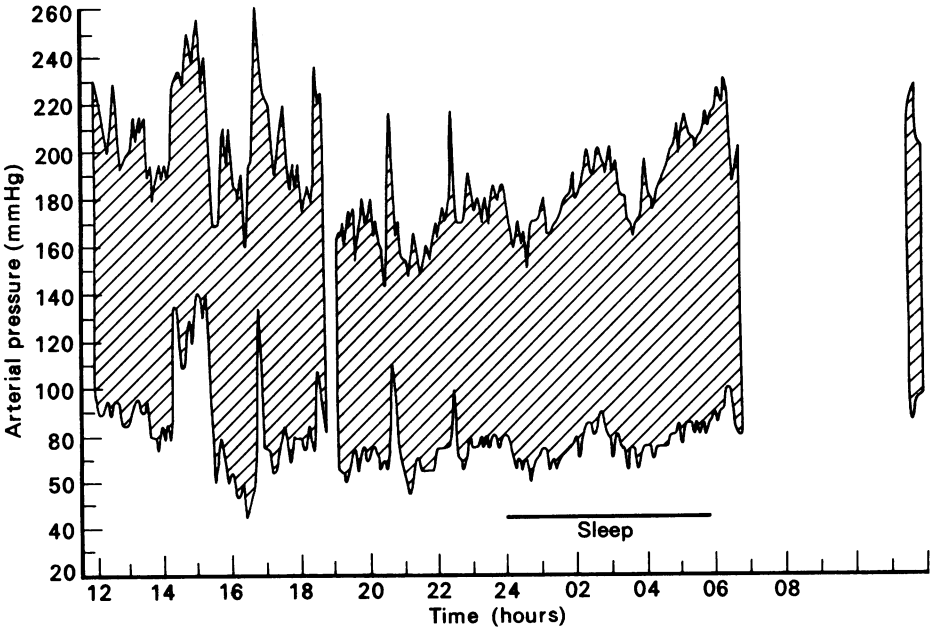

Figure 4a
Arterial pressure, plotted at 5 min. intervals, of subject F.C. The period of sleep is shown by the horizontal bar. (Chronic bronchitis).

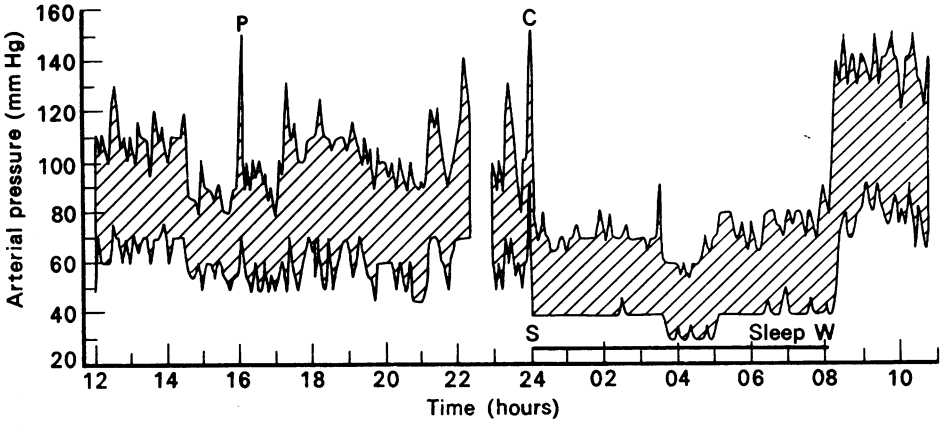

Figure 4b

Arterial pressure, plotted at 5 min. intervals, of subject A.B. The period of sleep is shown by the horizontal bar. The high pressures shown at 16.00 and 24.00 hours are due to a painful stimulus and coitus respectively.

It should not be too much to hope that such physiological education and re-education may one day be organised by health centres linked with sporting, cultural and recreational complexes geared to a common social aim, the full realisation of the whole physical and intellectual potential of every man and woman. Some new health centres should be located with such future possibilities in mind. Such a future may be difficult to conceive (particularly for doctors: lay people are more receptive), but less so, I hope, than a situation in which one third of the population is subjected to the crude and inflexible intrusion of drugs.

Handling data

Returning to our immediate future, there is a need for practical work on a large and reproducible scale, for the elaboration of simple systems of standardised data recording using agreed definitions, and for systems of data recall. Computers may not always provide an effective or economic answer to this problem, though they can be of enormous value in maintaining the accurate age-sex registers essential to serious primary care planning (Berkeley *et al.*, 1972).

The possibilities of mechanical data storage and retrieval systems have hardly been touched (Harden *et al.*, 1974) and are in many ways more appropriate to the local initiative on which nearly all innovation in primary care has so far depended. Above all, we need to bring together those practice teams who are seriously trying to deliver planned whole-population care in any dimension: to pool their knowledge of techniques for doing this, for expanded and imaginative delegation of work within teams, for new systems of data recording, retrieval and control, and, at the right time, agreement on standardised procedures and definitions. These measures are just as essential for effective care as they are for research.

Finally, we need far more initiative and experience in involving patients in their own care and in mutual patient education. Hypertensives are a large enough group for one of them to be a useful adviser to any practice in the planning of its work.

The next few years should see rapid progress in these aspects of the social organisation of care. In this way the care of hypertension may become a pathway for the later expansion of other forms of secondary prevention, as they become validated by convincing evidence, particularly, we must hope, in the prevention of ischaemic heart disease.

Such planning of essentially preventive work can not only be more exciting than crisis-oriented curative medicine, but more human as well, for at its heart lies a concern with real and individual people rather than with the gross pathology of more or less advanced disease. Speaking of the United States, Weed (1971) said:

> "There is no practical way for literally millions of patients to benefit from application of the highest standards except through the use of structured information... Whether this necessary reminding of the physician involves books, cartoons, an abacus, or a computer is not important. What is important is that the proper care gets to all the people. **We should continually remind ourselves that not to think quantitatively about the needs of all the people has qualitative implications for most of the people: and in our efforts we should neither worship nor fear the computer and technology, but we should simply use them as long as the benefits outweigh the losses in an honest accounting that does not leave, as many present methods do, thousands of people without care and without even being taken into account. . . .**"

This is equally true of the planned delivery of care for hypertensives in Britain. Individualised care, orientated towards effective influence on outcome rather than the satisfaction of the doctors' own emotional or other needs, is not denied by a basis of planned structure with defined sets of data; it needs them as the precondition of safe and effective care. Without such a genuine technical basis, the delivery of care can quickly degenerate into fraud.

Just as British research has in many ways led the world in the objective evaluation of care, British general practice can lead in the application of these conclusions, boldly discarding what is invalid, adopting those new techniques that are supported by real evidence, and then applying them to all those at risk. The most exciting field for such innovation lies in the care of hypertension, and those who pioneer this very arduous task will be at the leading edge of world medicine.

Addendum

In the course of writing this paper—in fact, because of writing it— I revised my opinion on the important matter of different criteria for treatment in men and in women. Because all my own data were accumulated on the basis of such a differential, it was difficult to revise the text.

If it is true that the treatment of hypertension does not reduce mortality from ischaemic heart disease, but only that from stroke, renal failure, and hypertensive heart failure; and that there is a sex differential in respect only of ischaemic heart disease, and not of these other outcomes; then differential criteria for men and for women are irrational. (Even if this is true, it does not justify a situation in which far more women than men are treated.)

Just as this conclusion became obvious, evidence began to accumulate of a useful effect of β-blockade on mortality from ischaemic heart disease. If this is confirmed, we may be back at square one!

All this merely confirms the thesis that diagnoses are not autonomous abstracts from nature, but plans for action related to human outcomes. Our definitions must change in relation to the accumulating evidence on the natural history of disease, and the new possibilities of effective intervention created by science. We can no longer afford to learn our diseases for life from a bestiary; we must learn to live with continual revision in this, as in all else in medicine. Stability is a necessary, useful, but ultimately destructive artefact, and we must learn to discard as well as impose it.

Acknowledgements

This essay derives from work carried out from 1962 onwards, with generous help from an Upjohn travelling fellowship, the Research Foundation of the Royal College of General Practitioners, the British Heart Foundation, the Welsh Regional Hospital Board, the Rudolf Friedlaender Trust, and the Medical Research Council's Epidemiology and Medical Care Unit.

I am indebted to my wife Mary, who did much of the screening work, Mrs Betty Ackery, my research secretary Mrs Joyce Rees, and my colleagues Dr Reg Saxton and Dr Andy Haines. I have had helpful advice on many occasions from Professor Archie Cochrane, Dr John Coope, Professor Sir Richard Doll, Drs Tom Meade, and Bill Miall, and ruthlessly exploited Miss Hammond of the College library, and Miss Gregory of the Pneumoconiosis Research Unit Library.

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THE ROLE OF RECEPTIONISTS IN HEALTH CENTRES

Receptionists derive their major job satisfaction from their interaction with patients. One specific aspect of this is the relationship developed by receptionists with a *family* over a period of time e.g. through the antenatal clinic, the well-baby clinic, and surgeries.

They also tend to derive their main job stress through interaction with some (female) patients. Some receptionists also derived stress from unreasonably aggressive behaviour from doctors.

Patients tend to see the most important part of the receptionist role to be a ' helping relationship ' towards themselves, this view is endorsed by receptionists. Both receptionists and patients are aware of the basic anxiety of many patients in their contact with the practice organisation, whether this is by telephone or face to face. Patients may often be both anxious because they are worried about their own, or their relative's health, and/or irritable because they are suffering pain or discomfort.

The first contact between the patient and the practice organisation will almost certainly be through the receptionist. This and subsequent contacts preceding the consultation may influence the actual value of the consultation for the patient. For example, if the patient feels embittered and humiliated by his treatment from the receptionist he may not feel very trusting towards the doctor who employs that receptionist. Thus in one sense the receptionist may be considered as a member of the clinical team.

Patients seem to have a clear notion of the service they feel they have a *right* to expect from a health centre e.g. the right of *immediate* access to a doctor. This notion may not be fully understood in the practice, and this contributes to the stress suffered by both patient and receptionist when it is not fulfilled.

Though job descriptions were not available, the observed differentiation of the receptionist role consisted of:

Nurse receptionist,
Receptionist,
Filing clerk,
Medical secretary/typist.

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