Hypertension: Essential hypertension: the heart and hypertension

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The heart and hypertension are intimately linked. Hypertension predisposes to coronary heart disease, myocardial hypertrophy, and cardiac dysfunction. Other organs and systems are also important in hypertension but this article concentrates on the effect of hypertension on the heart. The impact of hypertension on the heart is much more important than its effect in causing stroke and renal failure in terms of numbers of patients affected. There is still undue emphasis on diastolic pressure, with little attention paid to isolated systolic hypertension, and treatment remains inadequate for many patients. Cardiologists have a responsibility in this regard. The perspective taken in this article is that of the physician in the outpatient clinic.

Background

General practitioners deal with most hypertension. Patients are usually sent to the hospital clinic because the blood pressure has not been controlled despite multiple drug treatment, for loss of previously good control, where it is felt that a cause for hypertension should be sought, or because of an overt cardiovascular event. The extent and tempo of investigation of the elevated pressure are determined by the clinical situation. Hypertension also presents as an incidental finding in other clinical situations and as a result is often suboptimally managed or even ignored. Attention to blood pressure is surprisingly cavalier in cardiac clinics given its importance as a risk factor. Undoubtedly, it is more difficult to deal with than the measurement of cholesterol and the reflex—albeit appropriate—prescribing of a statin in the patient with known ischaemic heart disease. In light of the cost of coronary angioplasty and bypass graft surgery, especially to the patient, it seems inappropriate not to pursue rigorously the best management of hypertension. Blood pressure recordings after myocardial infarction, revascularisation, and rest in hospital are unlikely to be representative of subsequent levels. Follow up is essential.

Even when undertaken, the measurement of blood pressure is sometimes casual and imprecise. Nonetheless, attaching a number to the reading is important. Inspection of the hospital notes for previous recordings can be useful in determining past levels and the need for treatment. The British Society of Hypertension (BSH) guidelines have been incorporated into the joint British guidelines on the prevention of coronary heart disease,1 and recommend formal assessment of 10 year coronary heart disease risk as a guide to the treatment of high blood pressure.2 Cardiac hypertrophy can alter that risk and is not accounted for in the standard risk tables currently in use for primary prevention. Other articles in this series deal with risk assessment and drug treatment.

The level of the pressure and time of exposure appear to be key factors in determining the effect on the heart. However the response of the individual seems varied and the length of exposure is rarely known accurately. This leads to an imprecise relation between the level of pressure recorded in the office and the state of the heart. The underlying cause of the hypertension seems unimportant in the development of cardiac problems, except in the rare case of phaeochromocytoma. Here a cardiomyopathy may ensue of such severity that the patient may no longer have raised blood pressure. All patients with cardiomyopathy, irrespective of blood pressure, should therefore have a 24 hour urine collection for measurement of noradrenaline and adrenaline (norepinephrine and epinephrine). Renal artery stenosis should be sought in hypertensive patients with vascular disease, particularly if presenting with recurrent pulmonary oedema and relatively preserved left ventricular systolic function.

Isolated systolic hypertension

Elevation of systolic pressure not accompanied by the expected diastolic rise carries cardiovascular risk, which can be reduced with treatment. In large population surveys systolic pressure continues to rise whereas diastolic pressure peaks in the sixth decade (50–59 years). Systolic pressure is the major determinant of the workload of the heart and cardiac hypertrophy. The wider pulse pressure associated with isolated systolic hypertension (ISH) is associated with an increase in risks from cardiovascular disease and mortality (fig 1). Systolic pressures continue to be ignored by some practitioners and patients, but in numerical terms ISH is probably responsible for more morbidity than the less frequent diastolic hypertension.
Coronary artery disease is one of the most frequent accompaniments of raised arterial pressure. Atheroma is not seen in the pulmonary arteries unless there is pulmonary hypertension, indicating a central role for pressure itself in the genesis of atherosclerotic lesions. Even so, the importance of large vessel coronary heart disease complicating hypertension has probably been underestimated. Modest pressure elevation is common in the population. In this substantial group coronary heart disease, not stroke, is the major clinical issue. In severe pressure elevation the problems of stroke and cardiac failure dominate. Framingham data showed that hypertension was the most common cause of heart failure, but this reflects the poor detection and treatment of hypertension, 30–50 years ago. Hypertension is now second to ischaemic heart disease as a cause of heart failure. However, the two conditions frequently co-exist. High pressure initially induces useful compensatory hypertrophy but later decompensation results in heart failure. Myocardial infarction may also play an important part in this decompensation.

Structural changes
Increase in left ventricular mass is a consistent feature of hypertension. Cardiac myocyte cell number does not increase but there is cell hypertrophy. In addition there is considerable interstitial change and fibroblast proliferation. The myocytes account for 70% of the normal cardiac mass but represent only 25% of the cell content.

The changes in small vessel structure are akin to those seen in other tissues in response to pressure elevation, with increase of wall thickness and relative reduction of lumen. However, the haemodynamics of the coronary vessels and the smaller arterioles are different from other organs. Flow is greatest in diastole but the pressure curve in the epicardial coronary arteries follows that of the proximal aorta with which they are contiguous. The smaller intracardiac vessels are subject to extrinsic pressure from contracting cardiac muscle, their feeding pressure and pressure within the ventricular cavity.

An increasing mass of myocardium, made up of larger cells with increased deposition of surrounding collagen, requires more blood supply and relative ischaemia ensues. Exercise tests may indicate ischaemia when the epicardial vessels show no narrowing. Compensatory hypertrophy turns to myocardial failure, with increasing subendocardial ischaemia and subsequent fibrosis.

Additional ischaemia occurs from narrowing of the large epicardial arteries. A stenosis of less than 70% is usually compensated for by dilatation of the smaller arterioles distal to the lesion. However, when the small distal vessels are hypertrophied and subject to increased extrinsic pressure from hypertrophied myocardium, along with raised ventricular pressure, the flow reserve diminishes. Ischaemia occurs at lower workloads. Occlusion of a major vessel may further damage heart muscle and precipitate overt failure in an already compromised vulnerable hypertrophied heart.

Numerous hormonal and neurogenic factors have been postulated to contribute to these changes. The benefit in morbidity and mortality from treatment with both β antagonists and angiotensin converting enzyme (ACE) inhibitors in patients with impaired systolic function, clinical heart failure after myocardial infarction or at high cardiovascular risk suggest at least two important adverse processes amenable to partial correction with treatment. Nevertheless, the reduction of blood pressure itself, irrespective of the mechanism of drug action, appears to both prevent myocardial infarction and reduce the incidence of heart failure.

Hypertrophy is initially concentric. Wall thickness and muscle mass increase, systolic wall stress remains unchanged. At this stage coronary reserve is already compromised. Asymmetric hypertrophy is reported in some young adults with established hypertension, increased heart rate and stroke volume with normal peripheral resistance are reported. Increased peripheral resistance (from the smaller resistance vessels) and loss of compliance (increased stiffness) of large arteries is seen with a consequent increase of mean pressure and pulse pressure, largely through systolic elevation (fig 1). Endothelial dysfunction plays a part in flow modulation via impaired nitric oxide synthesis by the coronary endothelium. Coronary reserve decreases. Myocardial compliance (ventricular distensibility),
measured by the pressure–volume relation, can remain normal even with severe hypertrophy. Decompensation of the ventricle is associated with loss of compliance. The product of the systolic wall stress and the stroke volume is increased but the forward pump function is lessened. The heart function can be supported by reducing afterload, reducing preload (thereby reducing volume so increasing the mass:volume ratio), and inotropes.

Causes of cardiac and vascular changes
Undoubtedly increased blood pressure itself enhances the vessel and cardiac changes alluded to above. The proof is the reversibility of many of these by the lowering of pressure by a variety of means. What initiates and augments the elevation remains largely unknown though extensively investigated.

The ECG
The ECG is inexpensive, informative, and available routinely. However, lead placement is too often approximate, even on the limbs. This can alter interpretation, particularly for voltage measurement but also for ischaemia (poor R wave progression in the chest leads may be caused by incorrect placement of V2–4). Computer algorithms for interpretation can help to alert the clinician to left ventricular hypertrophy (LVH), which is sometimes overlooked. Silent myocardial infarction is surprisingly common in males in the 40–59 year age group and may be more common in the hypertensive population.

Interpretation, particularly for LVH, must take account of the patient’s age and build. In particular the chest lead voltages are increased in young, slim, and athletic individuals and reduced in obesity. Racial differences alter the usefulness of the standard ECG criteria of hypertrophy. Specificity is decreased in blacks. See table 1 for commonly used voltage criteria and fig 2 for ECG examples.

The overall reliability of the ECG in the detection of hypertrophy ranges from less than 10% up to 50% when compared to measurement by cardiac ultrasound, depending on the population screened and ECG criteria chosen. This is well illustrated when electrocardiographic criteria were compared with ultrasound derived evidence of cardiac hypertrophy in 4684 subjects of the Framingham heart study. Voltage criteria combined with borderline and definite repolarisation changes had a sensitivity of only 6.9% but a specificity of 98.8%. Nevertheless, the presence of voltage criteria of LVH and repolarisation changes (fig 2) on ECG criteria adds a risk similar to that for a patient with a previously documented myocardial infarction. Indeed, investigation of asymptomatic hypertensive patients with ECG LVH strain shows a high prevalence of epicardial coronary disease. Sudden death is claimed to be six times more common for any given level of blood pressure and is thought more likely to relate to ischaemia rather than a primary arrhythmia, although long QT intervals are seen. LVH based on voltage criteria
without ST/T wave change carries less risk and seems to reflect largely the risk associated with the duration and severity of the hypertension. Non-specific ST/T changes alone carry no more risk than the presence of voltage criteria alone and are less clearly related to pressure levels. The finding of left bundle branch block (LBBB) or left axis deviation in hypertension is not uncommon but the significance is uncertain unless caused by ischaemia. Finally, a normal ECG cannot exclude significant ischaemic heart disease or heart failure in the patient with high blood pressure.

The calculation of independent risk associated with ECG change depends on the use of multiple logistic regression analysis to take account of other factors—for example, age—which themselves exert notable effects. The approach has well described limitations and serves to emphasise the importance of the controlled clinical trial to determine best clinical practice. Observational studies support the notion that antihypertensive treatment reduces the prevalence of high blood pressure and ECG voltage evidence of LVH with mild/moderate repolarisation changes.12

### Chest x ray

An enlarged heart shadow may represent LVH but equally may be caused by chamber dilatation, pericardial fat or technical factors such as poor inspiration and projection. Conversely, an apparently normal sized heart may be hypertrophied or have impairment of function, especially if induced through ischaemia. However, chest radiography can still be important in the assessment of the hypertensive patient and may show left atrial enlargement, pulmonary venous hypertension as a consequence of increased left atrial pressure, abnormalities of the aorta and rarely rib notching.

### Cardiac ultrasound

Cardiac ultrasound is not generally recommended in the assessment of all hypertensive patients, but it can be informative in certain situations. The assessment of the LVH is an important but difficult task. Modern ultrasound machines have better capability for

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**Table 1 Commonly used criteria for ECG left ventricular hypertrophy (LVH)**

<table>
<thead>
<tr>
<th>Type</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chest lead</strong></td>
<td>R wave in V1 or V2 exceeds 25 mm&lt;br&gt;S wave in V1 or V2 exceeds 25 mm&lt;br&gt;Depressed S wave in V1 or V2 exceeds 35 mm&lt;br&gt;Ventricular activation time (onset of QRS to peak R) exceeds 0.04 s</td>
</tr>
<tr>
<td><strong>Limb lead</strong></td>
<td>R in aVL exceeds 11 mm&lt;br&gt;R in I exceeds 12 mm&lt;br&gt;R in aVF exceeds 20 mm&lt;br&gt;R in I + S in III exceeds 25 mm&lt;br&gt;R in aVL + S in V5 exceeds 13 mm</td>
</tr>
<tr>
<td><strong>Repolarisation changes (see note)</strong></td>
<td>ST depression with inverted or biphasic T waves&lt;br&gt;1 and aVL (facing left ventricle when heart horizontal) or 11 and aVF (facing left ventricle when heart vertical)</td>
</tr>
</tbody>
</table>

Additional points

LVH results in only slight shift to the left of the frontal plane QRS axis
- Horizontal heart: axis = +30° to −30°
- Vertical axis: axis = +40° to +90°

There is often counterclockwise rotation—that is, qR complexes appear in the chest leads before the usual V1 to V5. Prominent u waves may be seen in the mid and right precordial leads in LVH. Remember digoxin can produce ST/T wave changes and u waves

*Vary with criteria used and population screened—see text.

Note: “strain” refers to the additional presence of ST/T wave changes, usually definite ST depression (1 mm) and T wave inversion or biphasic T wave, which are of particular prognostic importance in the presence of voltage changes—see text.

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**Figure 2.** (A) Twelve lead ECG showing left ventricular hypertrophy (LVH) in the limb leads only. The patient was obese, which reduces the sensitivity of the chest leads for LVH. (B) Twelve lead ECG showing LVH and widespread strain pattern. The strain pattern denotes a worse prognosis. Ischaemia (large and/or small vessel) is likely to be present.
Table 2  

Echo criteria for LVH and formula for left ventricular mass calculation

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal and posterior wall thickness</td>
<td>Abnormal S &gt; 13 mm men; &gt; 12 mm women</td>
</tr>
<tr>
<td>Abnormal PW &gt; 12 mm men; &gt; 11 mm women</td>
<td></td>
</tr>
<tr>
<td>Left ventricular mass formula (using American Society of Cardiology guidelines, measuring leading edge to leading edge)</td>
<td>Left ventricular mass ( (g) = 0.83 \left( S + PW + LVDD \right)^3 - LVDD^3 + 0.6 )</td>
</tr>
</tbody>
</table>

*Corrected for body surface area. Correction by height in metres is preferred in Framingham outcome studies.

S, septal thickness; PW, posterior wall thickness; LVDD, left ventricular diastolic diameter.

Figure 3. M mode (left panel) and two dimensional (right panel) echocardiography (parasternal long axis view) demonstrating concentric LVH in a female patient (septum 13 mm; left ventricular (LV) cavity 48 mm; posterior wall 13 mm). The measurements (leading edge to leading edge) are taken just beyond the tips of the mitral valve (MV) leaflets at end diastole. Care must be taken not to include the right ventricular band in the septal measurement. RV, right ventricle; AV, aortic valve.

fibrilliation. Detection of regional wall motion abnormalities is useful confirmatory evidence for associated ischaemic damage and may be found in the absence of a history of coronary ischaemia. Increased left atrial size and pulmonary artery pressure may indicate hypertensive damage but are not specific. Non-invasive estimates of left atrial or end diastolic ventricular pressures are not sufficiently robust for clinical use. Calcified aortic cusps—"aortic sclerosis"—are found more frequently in the hypertensive and may be a marker of increased risk through coronary disease."

The ultrasound examination may also give useful information on cardiac function and consequently prognosis. Should the presence of myocardial dysfunction influence the choice of treatment or management? Impaired systolic ventricular function was a major reason to justify treatment with an ACE inhibitor and perhaps to avoid treatment with a β-blocker. The recent HOPE (heart outcomes prevention evaluation) study has emphasised that high cardiovascular risk alone is sufficient to warrant use of an ACE inhibitor. The recent β-blocker studies now mandate the use of β-blockade in patients with heart failure, assuming clinical stability and careful titration of dose. Thus knowledge of left ventricular systolic function does not necessarily influence treatment decisions.

Cardiac ultrasound can sometimes help sort out the breathless hypertensive patient, particularly if there is clear cut evidence of systolic dysfunction or significant valve disease. Patients may, however, be more breathless than expected for a given level of systolic dysfunction, raising the possibility of associated diastolic dysfunction, particularly if LVH is present. Patients with hypertension are often old, and age and hypertension are particularly associated with "stiff ventricles" and consequent breathlessness. The assessment of diastolic dysfunction is controversial and has been the subject of many reviews. Its reported frequency ranges from 10–40%, depending on selection criteria and measurement techniques. Diastolic dysfunction is difficult to establish using current echocardiography techniques, many of which are unduly influenced by fluid loading conditions in the patient. The finding of an apparently normal heart or one with obvious hypertrophy, dilatation or impaired systolic function in the patient. Indeterminate findings are more difficult to interpret clinically.

Magnetic resonance imaging

Assessment of left ventricular wall thickness and overall left ventricular mass using magnetic resonance imaging (MRI) is probably the most accurate non-invasive method for the assessment of LVH. Assessment of left ventricular systolic function and tissue blood flow at rest and with pharmacological stress can also be undertaken. However, there are no substantial studies linking measurements by this approach to outcome. Limited availability and patient acceptance restrict its use at present, but future
protocols may allow for a single complete investigation of the hypertensive patient, giving information not only on the presence of LVH, but also coronary disease, myocardial structure and function, valve disease, and other important pathology including evidence of renal artery narrowing or adrenal pathology (fig 4).

Ambulatory blood pressure measurement
It seems logical that multiple measurements of blood pressure give a better estimate of “hypertensive load” than a single measurement. This is borne out in the consistency of such 24 hour readings, which have allowed definitions of day and night time normality, and exhibit a closer relation to ventricular mass estimates and coronary events. The daytime average is most usefully compared with the clinic reading. Guidelines suggest adding 10/5 mm Hg to accord with clinic/office readings. The early readings after initial cuff application, if unduly elevated, indicate the presence of the “alerting reaction”; it may be seen again in the hour preceding return of the pressure measuring device. This measurement seems most useful when clinic readings show unusual variability, when blood pressure is resistant to treatment, when there are symptoms suggestive of hypotension in the absence of an obvious postural fall, or when “white coat hypertension” is suspected.

Stress testing and the hypertensive
Exercise ECG can be helpful in the diagnosis of associated ischaemia. Generally, the stress test should not be undertaken if the blood pressure is very high (> 220 mm Hg systolic or 115 mm Hg diastolic, or both) and should be stopped if the pressure increases greatly during exercise. It may not be possible to stop antihypertensive drug treatment before exercise testing, thus reducing the sensitivity of the test. The prognostic value of the increase in blood pressure with exercise does not seem greater than for resting blood pressure, even though it is claimed to relate more closely to cardiac hypertrophy. A fall in the level of the patient’s usual pressure (in contrast to the “settling” of pressure elevated by anxiety) with increased

Figure 4. Magnetic resonance images. (A) Long axis view showing pronounced left ventricular dilatation and thinning of the interventricular septum (arrow) (True-FISP acquisition). (B) Mid-ventricular short axis view of the same patient as in (A), showing the dilated left ventricle with thinning (arrow) of the septum (True-FISP acquisition). (C) Another patient but a similar view to (B) acquired from a single phase gradient echo cine and in contrast showing pronounced hypertrophy of the left ventricle. The thin walled right ventricle can be seen wrapping around the left ventricle, lying superiorly and to the left on the cross sectional views. The views are similar to a cross sectional two echo view. Note the clarity of the endocardial border, allowing accurate estimation of left ventricular volume and mass. LA, left atrium; LV, left ventricle, RV, right ventricle. (Images provided by Dr U M Savananthan.)
workload can indicate serious cardiac impair-
ment. The test can be repeated after improved
blood pressure control or an alternative stress
test method used. The ECG may be difficult to
interpret if repolarisation abnormalities or
LBBB are present, but the exercise time, symp-
toms, and blood pressure response can still
provide useful information. Alternative stress
tests using either pharmacological agents or
exercise with echocardiography or nuclear
imaging can improve the sensitivity and
specificity of ischaemia detection, but are more
costly and generally less available. ST segment
depression, abnormal response of ejection
fraction, and perfusion defects occur without
evidence of obstructive epicardial vessel disease
in the presence of hypertyrophy. Stress echo
techniques may be more specific than nuclear
techniques for epicardial vessel as opposed to
small vessel narrowing.

Coronary and left ventricular angiography
This can be undertaken when significant
 coronary and/or valve disease is suspected.
Blood pressure control should be optimised
before arterial puncture. LVH and dysfunction
may be evident from the left ventricular angio-
gram, but in general the non-invasive tests pro-
vide this information. Increases in left atrial
and left ventricular end diastolic pressures may
indicate cardiac involvement in hypertention.
Small vessel disease is inferred when stress tests
are abnormal but no narrowing of major vessels
is seen on angiography.

**Left ventricular hypertrophy**

How important is the presence of LVH? The
relation of LVH with the level of pressure is
complex. In part this may relate to never knowing
how long blood pressure elevation has
been present at any particular level in an individ-
ual. The prevalence of hypertyrophy in-
creases considerably with age. Many other fac-
tors are involved in its development. Black
patients were thought to be more at risk for
developing LVH for a given level of pressure,
but Lee9 has shown reduced specificity for
ECG criteria for LVH in black patients. In
general, hypertensive black patients have a
lower incidence of cardiac events than whites.19
In 3220 subjects in the Framingham heart
study, apparently free of cardiovascular disease
and over the age of 40 years at enrolment, the
relative risk of an increment in cardiac mass of
50 g per metre height (substantial), adjusted
for age, diastolic blood pressure, pulse pres-
sure, antihypertensive treatment, cholesterol,
cigarette smoking, diabetes, body mass index,
and ECG evidence of LVH with repolarisation
change, was associated with a 49% risk increase
in cardiovascular disease in men but at the
same time a 49% risk increase in mortality
from all causes. For women the cardiovascular
and all cause mortality rates were doubled.21
Given that the accepted clinical approach is to
produce optimal pressure reduction in all patients, especially encouraged by the findings
of the recent HOT (hypertension optimal treat-
ment) trial, how useful is the knowledge
about the presence of LVH in the individual
patient? There are no drugs currently to treat
cardiac hypertyrophy itself, even if echo diag-
nosed hypertyrophy is accepted as carrying risk
additional to the pressure measurement. Its
findings may encourage the physician and
patient to try harder and with more drugs, and
sway a treatment decision for “borderline”
pressure recordings, but the guide will remain
the level of pressure achieved. At the moment
there is no convincing trial evidence that
reduction of hypertrophy carries any more
benefit than that from reduction of the blood
pressure per se. It is difficult to see how this
hypothesis can be tested without the use of
agents that reduce ventricular hypertyrophy
independent of their effect on blood pressure.

Reduction of blood pressure leads to
regression of hypertyrophy, measured by a vari-
ety of techniques. Extrapolation from animal
studies suggests that the reversal is only partial
both in the quantity of mass reduced and the
quality of the remaining myocardial tissue
compared with a normal heart. The dominat-
ing factor is the extent of blood pressure
reduction. Claims for the superiority of one
agent compared with another in reducing
hypertrophy, particularly the effectiveness of
ACE inhibitors as a group compared with
other agents, continue to be debated.11

Modern treatment of hypertension is centred
on the concept of treating according to risk, not
simply the pressure level. Traditional trials in
hypertension are characterised by the low car-
diac event rates, and the evidence for reduction
in myocardial infarction or death has been dis-
appointing compared to prevention of stroke.
However, the consensus now from many stud-
ies is that reduction of blood pressure reduces
not only the occurrence of heart failure but also
myocardial infarction rates. Fears associated
with the J shaped curve and reduction of blood
pressure have been largely allayed by the HOT
study.2 Nevertheless patients with a critical ste-
nosis of a coronary vessel, especially if flow
reserve is compromised by LVH and its
accompanying pathological changes, can de-
velop worsening ischaemia if pressure is greatly
lowered.

The HOPE population carried a risk of
death, stroke or myocardial infarction of about
4% per year.13 Those included with hyper-
tension were treated with conventional drugs
excluding an ACE inhibitor. Overall blood
pressure control was better in the ACE inhibi-
tor treated patients, but those randomised to
the ACE inhibitor showed a significant reduc-
tion in cardiac event rates well beyond that
predicted from the small added blood pressure
fall. Previous trials to find superiority of one
drug over another have not found differences;
the reduction of pressure rather than the agent
seems paramount. However, this does not sit easily with the studies after myocardial infarction and in heart failure patients where clear benefit has been seen from ACE inhibitors and β blockers in reducing cardiovascular mortality and myocardial infarction rates. The recent CAPP (captopril prevention project) study specifically compared treatment including or excluding the ACE inhibitor captopril and found no difference in events. However, the study has been heavily criticised. The Swedish STOP-2 (Swedish trial in old patients with hypertension 2) study superficially also appeared to find no difference between agents on event rates. Nevertheless, subgroup analysis does suggest that a regimen including an ACE inhibitor might be beneficial in reducing rates of myocardial infarction. Furthermore the doxazosin arm of the ALLHAT (antihypertensive and lipid lowering treatment to prevent heart attack) study has been stopped, on the basis that fewer patients in the diuretic arm developed heart failure than those on doxazosin. Whether this is because of “pre-treatment” of heart failure by the diuretic or a genuine lack of effect of doxazosin is speculative. The overall mortality and cardiovascular risk is not reduced by treatment to the level of the normotensive. Clearly attention to other risk factors (diabetes, smoking, increased cholesterol) is also important and absolute risk varies widely between populations. Most patients require at least two agents to control blood pressure adequately, and some three or more. A regimen that includes a β blocker and an ACE inhibitor might be expected to offer some cardiac protection when risk is present. The β blocker will also help control the symptoms of angina. Addition of a vasodilating calcium antagonist may improve symptoms and further reduce pressure. Diuretics remain unequalled for symptom control in the patient breathless from heart failure, offer blood pressure control equal to other agents used alone, combine well with other drugs, and—although they have side effects—are remarkably well tolerated by the majority of patients. Spironolactone no longer has a licence in the UK for the treatment of hypertension, but the recent findings of benefit from this drug in those with severe stable chronic heart failure would justify its use in the hypertensive with heart failure. Lifestyle measures are also important in the reduction of blood pressure levels and thereby LVH.

Aspirin and heart disease
Aspirin is usually prescribed in those with known ischaemic heart disease. Clopidogrel is an alternative in patients genuinely intolerant of aspirin. Aspirin as primary prevention is controversial. The HOT study suggests a relative risk reduction of about 15% for major cardiovascular events, but at the cost of an excess of major bleeding events. When the individual’s overall absolute cardiovascular risk is high then the 15% relative risk reduction becomes worthwhile when set against the risk of a serious bleed. The BHS guidelines’ advocate statin prescription to those with angina or previous myocardial infarction at a total cholesterol concentration of 5 mmol/l or higher under the age of 75 years.

Treatment of hypertension:
- By any agent, if effective, will reduce risk of ischaemic heart disease (IHD)
- Should include β blocker or ACE inhibitor, or both, if heart disease present
- Usually requires two or more agents
- Should include attention to lifestyle and modifiable risk factors
- Should include aspirin for those with IHD, or at high risk of developing IHD

Summary
The effect of hypertension on the heart and therefore prognosis is highly variable and depends not only on the pressure level but also on other factors including age, sex, cholesterol, and smoking. High blood pressure can severely damage the heart, reducing the quality of life as well as longevity. Significant protection is offered to the heart by good control of blood pressure and other risk factors. The higher the risk the greater the absolute benefit the patient can expect. Doctors should not exaggerate the risks of pressure elevation to the individual patient and thereby over claim the benefit likely to accrue from treatment; however good the treatment, it cannot be expected to more than compensate for the associated risk. Understanding of these concepts by both patients and physicians should lead to improved care and protection of the heart through the early detection and rigorous control of high blood pressure.

4. A meta-analysis of three large trials in systolic hypertension showed that after controlling for mean pressure, a 10 mm wider pulse pressure increased the risk of cardiovascular mortality by 20%. The probability of a cardiovascular end point increased with lower diastolic pressures for any given systolic pressure suggesting that the wider pulse pressure is driving the risk of major complications.
Education in Heart


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Heart 2001 86: 467-475
doi: 10.1136/heart.86.4.467

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