A New Look at Hypertension

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Summary

Progress in hypertension has produced benefits for many people but still there are unresolved problems. We still have to decide: at what level to treat hypertension; and what are the longer risks of drugs which so effectively control blood pressure. Research is in progress in the fascinating abnormality of cellular sodium transport; prostaglandin/Kallikrin - Bradykinin system, and the renin angiotensin cascade. We have not achieved perfection; controversy continues which is the stimulus to progress in medicine.

Introduction

Hypertension is a major cause of mortality responsible for 44% of deaths from cardio-vascular diseases as revealed by the famous Framingham Massachussetts study.

The bulk of the deaths were with no warning and could have been entirely prevented by treatment.

1980 saw two large trials on mild hypertensives (90-105 mm Hg of diastolic pressure), confirming the reduction in death from complications of hypertension i.e. coronary artery disease, strokes, heart failure and atheromatous arterial disease.

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<th>DEFINITION</th>
<th>BLOOD PRESSURE</th>
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<td>(MM HG)</td>
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<td>NORMOTENSIVE</td>
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<td>HYPERTENSIVE</td>
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<td>BORDERLINE</td>
<td>S 140 - 160</td>
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As defined by W.H.O.

Etiology

Framingham study, conducted on 5000 people and followed-up for almost 20 years, suggested that less than 5% of hypertensive had any discernable cause: 95% were essential hypertensives.

Secondary causes like renal or endocrinological causes or coarctation of aorta are rarities to look for.

In general most physicians are less obsessional in their research in older subjects and agree to investigate those under forty years of age.

The etiology of essential hypertension continues to be debated.

Multifactorial Etiology

Significant co-relations have been demonstrated between the blood pressure of family members, identical twins and siblings.

Platt proposed a single dominant gene. Pickering on the famous Pickering and Platt controversy of the 1960's refuted this hypothesis based on a fallacy arising from the statistical unreliability of the relatively small number in Platt survey.
Pickering proposed the theory of multiple genetic influences on arterial pressure.

There is now agreement that hypertension has multifactorial etiology.

There is also evidence to suggest that the blood pressure of an individual is determined soon after birth and is modified subsequently by environmental factors which include age, stress, atheroma, diet and salt.

**Salt Intake**

Study of spouses of hypertensive husbands with similar salt intake has not shown any striking influence on arterial pressure.

De-Wardner has postulated defect in the renal excretion of sodium in hypertension which leads to an increase in circulating natriuretic hormone. Similarly work on red cell sodium fluxes has demonstrated defect in excretion of sodium.

This defect in the presence of increased salt intake can eventually lead to essential hypertension. There is evidence that increased intracellular sodium in paralleled by increased intracellular calci. If such a defect is present in arterial smooth muscle cell, it will be a plausible explanation for increased arterial tone and so hypertension.

Severe restriction of sodium in some patients with renal failure, has led to deterioration in renal functions. Non-the-less some restriction of salt intake can reasonably be encouraged.

There is evidence to suggest that a high potassium intake and similarly a high calcium intake may be protective against hypertension. Epidemiological data suggest that hard water protects against cardio-vascular disease and that magnesium deficiency is associated with hypertension.
Risk Factors

Factors which may accelerate the damage from hypertension are:

1. Sex: Women withstand raised arterial pressure better than men. The risk of death or mortality is about half of that of men.

2. Abnormal Blood Lipids: At the same arterial pressure, serum cholesterol of 8.6 m mol/l is associated with 5-fold increased mortality from cardio-vascular disease than serum cholesterol of 4.7 m mol/l.

3. Cigarette Smoking;

4. E.C.G. evidence of left ventricular hypertrophy;

5. Glucose Tolerance and

6. Obesity.

These factors act as "Multipliers: in determining whether to treat a certain level of hypertension.

NON-DRUG APPROACH

1. Weight reduction will result in reduction of arterial pressure.

2. Now the recommendation is decrease in carbohydrate to reduce sympathetic output.

3. Sodium restriction is no more advised; however, excessive salt intake should be discouraged.

4. Attention to patient's life style; however, it is usually difficult to change.

5. Mental relaxation; easy to say, difficult to achieve.
6. Exercise has little value in bringing down blood pressure; however, graded exercise will cause a feeling of well-being.

7. Smoking should be given up.

8. Future surveillance, that is constant check on blood pressure, is essential.

9. Sedation for those who cannot relax has a modest effect on blood pressure.

Today's drugs are safe, effective and well tolerated. The present drug therapy began 30 years ago when Smirk and his colleagues from New Zealand demonstrated reversal of malignant hypertension by hexamethone compounds.

The Australian Blood Pressure Trial,\(^4\) carried on 3560 patients (age 30-39 and BP 90-105 diastolic) and followed-up for 4 years, showed 2/3 reduction from cardio-vascular mortality in these mildly hypertensive patients.

The question is should symptomless mildly hypertensive patients be submitted to the disadvantages and worries of 30 years pill taking in order to give an extra few years of life. However, when one looks at Framingham study, the incidence of stroke was 60 per 10,000 and coronary heart disease in 306 patients per 10,000.

The above findings have been substantiated by Veterans Administration study of Fries and his colleagues\(^6\) showing highly significant improvement in the treated group.

Drugs

THIAZIDES are ideal for mild hypertension. However, a recent BMRC trial showed men giving up diuretics because of impotence.\(^1\)

B-BLOCKERS have brought a revolution in the management of hypertension and now with the long acting preparation, it is more or less
the first choice. BMRC trial has shown poor compliance in females because of cold fingers and toes.¹

VASODILATORS are a separate group of drugs and have given a great versatility to hypertension management. Hydralazine, Prazosine and Minoxadil are a good addition. Apart from reflex tachycardia, which can be counteracted by B- Blockers, Minoxadil may cause excessive hair growth and this hars its use in women. This side effect is now put to therapeutic use for balding men. Vasodilators are the drugs of choice for chronic renal failure.

CENTRALLY ACTING DRUGS are Clonidine and Methyldopa. Methyldopa is one of the most extensively used drug, safe and effective. Clonidine can be given when hypertension coexists with migraine.

CALCIUM ANTAGONISTS: Verapamil, Nefedipine and Nitrendipine have established a definite place in the management of hypertension. They have the additional advantage of the ability to suppress SVT’s.

RENNIN ANGIOTENSIN SYSTEM BLOCKING DRUGS are a separate superior group. Captopril and Analapril are effective in resistant hypertension.

Malignant hypertension per se is not an indication for parenteral therapy. Malignant hypertension associated with left ventricular failure or encephalopathy is a medical emergency needing urgent but not drastic steps recommended in the literature. Enthusiasm in bringing down the blood pressure suddenly has produced neurological damage to the water-shed areas in the brain with visual loss, hemiplegia and sudden death.

SUDDEN REDUCTION OF BLOOD PRESSURE CAN BE ACHIEVED BY:

1. Crushed Adalat (Nefedipine) capsule given sublingually.

2. Diazoxide 75 mg I.V. followed by 150 mg five minutes later. 300mg I.V. rapidly was associated in the past with morbidity and mortality.
3. Hydralazine I.V. upt 20 mg is also ideal for control of malignant hypertension.

4. Sodium Nitroprusside is an I.V. preparation for rapid control of B.P. It is cumbersome to use as cyanide level needs monitoring.

5. Labetalol, the alpha and beta blocker, can be given slow I.V. in the drip for rapid control of blood pressure.

References

1. Medical Research Council working party. 1977, Randomized controlled trial of treatment for mild hypertension in B.M.J.I., 1437.


