Risk Factors in Ischaemic Heart Disease

SURGEON CAPTAIN G. KUPPUSWAMY, VSM, IN*

Introduction

OF the various risk factors in Ischaemic heart disease, Hypertension, Diabetes Mellitus and Obesity are among the more important ones. It is proposed to discuss the method of their early detection and their influence on the occurrence of I.H.D.

Hypertension

Varying levels of blood pressure have been quoted by various authors to indicate hypertensive status. The Framingham study group in their adult population (32-60 years) study has arbitrarily taken the following figures;

(a) Less than 140/90 mm of Hg....Normal

(b) $\frac{140-160}{90-95}$ mm Hg .....Borderline

(c) More than 160/95 mm Hg.....Hypertensive

The figures given in the Director General Armed Forces Medical Services Memorandum on Hypertension are very close to the above figures.

These figures are probably lower than those generally accepted by various authors; nevertheless since the issue is whether Hypertension—particularly the borderline hypertensive, contributes to a greater incidence of IHD, consideration of lower levels of blood pressure is probably of more value. It is well known and fully confirmed that the incidence of IHD in the grossly hypertensive is very much higher than in the normal population. Figures 1 & 2 show the incidence of IHD vis-a-vis the basal status of blood pressure in both men and women, based on the Framingham Study.

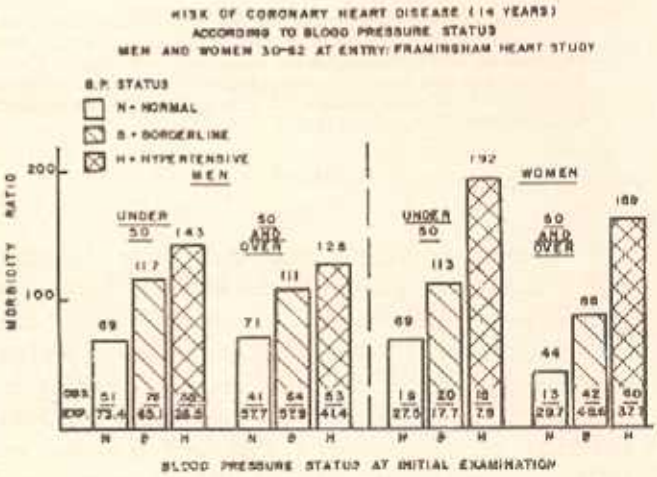


Fig. 1

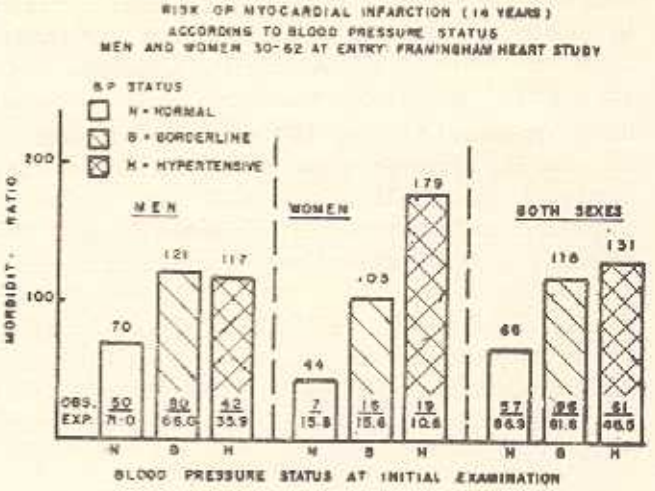
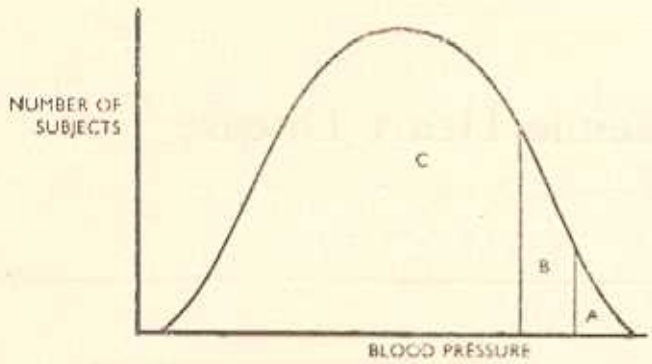


Fig. 2

* Senior Adviser in Medicine, ATNK & KK Area, Air Force Hospital Bangalore.



Schematic representation of blood pressure values in a large population. Patients in Group A are known to be at risk and known to benefit from treatment. Those in Group B are also at risk but it is not yet known if reduction of blood pressure would be of benefit. Those in Group C are subjects with "normal" blood pressure.

Fig. 3

Figure 3 represents graphically how a population could be divided into the normal, borderline and Hypertensive subjects. It is statistically confirmed that group A—the well confirmed hypertensive is very much at risk and certainly benefits by treatment. It is group B the borderline hypertensive that we are concerned with and it is not yet fully established that treatment of this group would indeed reduce the incidence of IHD.

Actuarial data clearly show mortality ratios which can be assigned to relatively modest increases in either systolic or diastolic blood pressures. Thus the Society of Actuaries, Chicago, have statistically confirmed that men with a casual blood pressure of over 140 mm Hg systolic or 90 mm Hg diastolic have a 50% increase in the mortality due to IHD over a 20 year period as compared to the expected incidence in the normotensive general population. There is an increase over 100% for values of 160 mm Hg systolic and 100 mm Hg diastolic. EOSTEUB et al have again statistically proved by postmortem studies that modest elevations of blood pressure are associated with accelerated atherosclerosis of the coronary arteries.

Apparently therefore, there is a clear case for treating the borderline hypertensive with drug therapy. However, FRIES et al have noted in a double blind 4 year study, no differences either in the incidence of, or in the mortality by IHD in both the treated and untreated groups of borderline hypertensives. This is probably because 4 years is too short a period in the evolution of coronary atherosclerosis. This was in 1965. However, in 1970 the Veterans Administrative Co-operative Study Group claimed a reduction in the morbidity due to IHD from 55% to 18% by treating borderline hypertensives with a diastolic pressure between 90 to 114 mm Hg. It would appear that the higher the pretreatment level of blood pressure, the greater the benefit to accrue. By and large, therefore, with the evidences at our disposal it would be appropriate to start therapy for the borderline hypertensives with diastolic pressure of over 90 mm Hg and a systolic pressure of over 150 mm Hg.

Diabetes Mellitus

It is impossible to discuss Diabetes Mellitus as a separate risk factor as the disease is very often associated with hypertension, hypercholesterolaemia and hyperlipidaemia and therefore it is necessary to emphasise that recognition, assessment and treatment of these associated factors should go hand in hand with the treatment of Diabetes Mellitus itself, in whatever stage it is recognised.

The increased frequency of atherosclerotic heart disease and the association of Diabetes Mellitus and Ischaemic Heart Disease with chemical and clinical Diabetes has been extensively documented and statistically confirmed by various workers. With respect to asymptomatic hyperglycaemia, as distinct from clinical diabetes, the TECUMSEH study reported an association between hyperglycaemia and coronary artery disease prevalence for both men and women. The findings were independent of and additive to the effect of raised serum cholesterol and blood pressure.

The result from the Framingham study however, suggests that hyperglycaemia may not be an independent risk factor, atleast not for the atherosclerotic coronary disease. STAMLER et al reported that higher blood glucose levels were correlated to higher blood pressure levels in proven diabetics. Men with hyperglycaemia with a blood glucose level greater than 170 mg% had a preval-

ence rate of hypertension of 32% compared to 18% in men with glucose values less than 170 mg%. Hyperglycaemia was also associated with hypercholesterolaemia and hyperuricaemia.

The mortality rate from IHD was 42 per 1000 for hyperglycaemic men and 16 per 1000 for normoglycaemic men.

It is obvious that

(1) The incidence of IHD and associated hypertension, hypercholesterolaemia and hyperuricaemia is significantly higher in both clinical and chemical diabetics.

(2) The mortality from IHD is definitely more amongst diabetics, both chemical and clinical.

Therefore there is a need for early detection and treatment of diabetics.

The point for consideration is whether treatment of early diabetics would have a beneficial effect in, (a) preventing and postponing IHD, or (b) reducing the mortality from IHD or (c) regressing or stabilising atherosclerotic coronary artery disease if it has already commenced.

KEEN et al in 1968 reported that treatment with Tolbutamide of mildly hyperglycaemic patients,—chemical diabetics—appears to protect them against the manifestations of IHD over a 5 year period as compared to the untreated group. But he could not confirm this impression after seven years follow-up.

PAASIKIVI in 1970 reported beneficial results with treatment by Tolbutamide in patients showing abnormal glucose tolerance after myocardial infarctions. In his impression, therapy with Tolbutamide postponed non-fatal relapses of Myocardial Infarction. In 1973, the pendulum seems to have swung. The University Group Diabetes Programme in their study felt that both Sulphonylureas (Tolbutamide) and Biguanides (Phenformin) exerted a deleterious effect on Hyperglycaemics and overt diabetics by favouring an earlier onset of IHD. But these findings have been challenged by KEEN et al, PAASIKIVI, STOWERS and others in the same year and the consensus seems to be leaning towards emphasis on the treatment of early diabetics or asymptomatic (chemical) diabetics with diet and oral hypoglycaemic agents.

It is therefore suggested that it will be desirable to screen all aircrew (it would be utopian to try and screen all personnel) above the age of 30 years by carrying out pre and post prandial blood sugar estimation. Patients who are hyperglycaemic should be subjected to immediate treatment either with diet alone or with Tolbutamide and reviewed at 3 monthly intervals.

Obesity

There is indirect evidence based on Life Insurance and autopsy statistics that Obesity predisposes to Coronary Artery Disease. Only in the extremely overweight persons does Obesity become a risk factor per se. The risk involved is possibly due to the close association of Hypertension, Hyperglycaemia, Hyperlipidaemia and Hyperuricaemia with Obesity. Chronic caloric imbalance contributes to the development of these traits. The overall mortality in obese persons is indeed reduced with weight reduction. Figure 4 shows the correlation between Obesity and mortality rates.

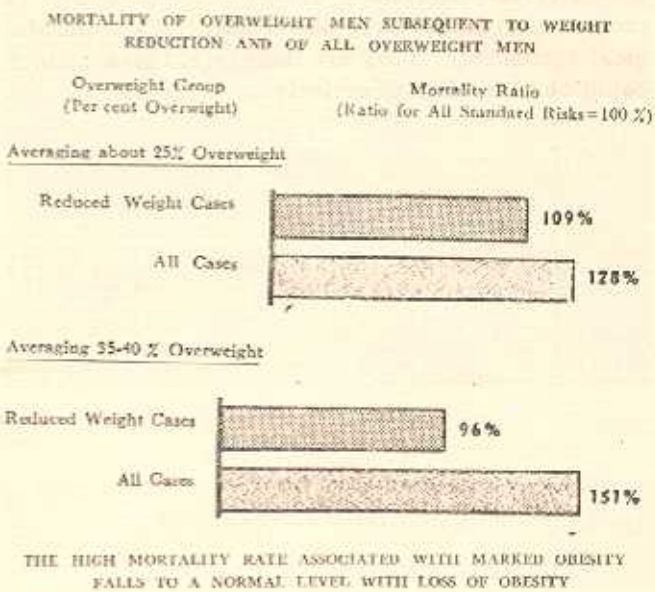


Fig. 4

Therefore it would be sound advice that weight should at all times be maintained at optimal levels.

Gout

Both Gout and Independent — asymptomatic — Hyperuricaemia with serum Uric acid levels greater than 7.0 mgms % are considered as risk factors associated with increased IHD. Increased levels of Serum Uric Acid are seen both in Gouty and non-Gouty patients and therefore hyperuricaemia per se is an independent risk factor and whenever encountered, hyperuricaemia must be controlled.

It is relevant to point out that small doses of Salicylates, the Thiazide diuretics, Acetazolamide and pyrazinamide increases serum uric acid. Large doses of Salicylates, Phenylbutazone, Probenecid, Allopurinol and Coumarin compounds reduce serum uric acid. It may be noted that Colchicine used in acute Gout does not reduce Serum Uric Acid concentrations. Hyperuricaemia associated with Obesity will respond to mere weight reduction.

Conclusion

The various conditions which have been discussed are without any doubt risk factors, promoting the incidence, morbidity and mortality due to Ischaemic Heart Disease. Furthermore, these are risk factors involving endogenous biochemical and physiological regulatory mechanisms amenable to exogenous influences like diet control, Pharmacological agents etc. They are therefore, factors which could be controlled effectively.

Early detection, and proper control of these factors at the earliest opportunity is a mandatory requirement in good medical practice.

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