Casual, basal and supplemental blood pressures in 519 first-degree relatives of substantial hypertensive patients and in 350 population controls

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Summary

1. The casual blood pressure is the sum of the relatively stable basal pressure taken under defined conditions of rest and the labile supplemental pressure (casual minus basal), which represents the response to the current degree of physical, mental and probably metabolic stimulation.

2. The basal and supplemental blood pressures behave differently and it seems likely that different factors are involved in their pathogenesis.

3. The 5 and 8 years follow-up mortality is closely related to the basal pressure but not to the supplemental pressure.

4. The rise with age in the basal blood pressure is greater in the relatives of substantial hypertensive patients than in population control subjects.

5. Above the age group 30-39 years there is an increase in the rate of rise of the mean basal blood pressure with age among the first-degree relatives of hypertensive patients. In a population control group an acceleration in the rate of rise of the mean basal blood pressure with age also occurs but a decade or more later than in the relatives of hypertensive patients.

6. In males the mean supplemental pressures (systolic and diastolic) do not rise appreciably with age and the mean supplemental pressures of first-degree relatives and control subjects do not differ appreciably.

7. In females the mean supplemental pressures rise with age but, except after age 60 years, the pressure rise in first-degree relatives is only a little greater than in control subjects.

8. When hypertensive patients with similar casual blood pressures are compared the basal blood pressures are higher in patients with glomerulonephritis than in essential hypertensive patients.

9. In the first-degree relatives of substantial hypertensive patients high-ranking basal blood pressures occur much more frequently than in general population control subjects.

10. The close resemblance of the blood pressures in like twins indicates that genetic or familial factors have an important influence on blood pressure, and on the occurrence of frank hypertension.

Key words: basal blood pressure, casual blood pressure, familial aggregation, inheritance, supplemental blood pressure.

Introduction

In 1938 Alam & Smirk compared the effects of blood pressure-raising reflexes in health and in essential and renal hypertension. This led to a study (Alam & Smirk, 1943; Smirk, 1944; Kilpatrick, 1948) of casual, basal and supplemental pressures. We introduced the term 'casual blood pressure' for the blood pressure as ordinarily measured. Addis (1922) had already made observations on the basal blood pressure.

About 1946, before any of the modern effective blood pressure drugs became available, Alstad & Smirk, joined later by Veale, began a study of the relationship between basal and supplemental pressures and the subsequent mortality in a series of 315 hypertensive patients (Smirk, Veale & Alstad, 1959). During the follow-up period these patients were under the care of their own doctors. There was no restriction on the use of the then-available therapeutic measures but patients treated by surgery...
or by very strict restriction of salt intake were not included. In terms of present-day standards we regard these patients as virtually untreated.

Patients who started with a high basal blood pressure had a much greater morbidity and mortality in the subsequent 5 or 8 follow-up years than those whose basal blood pressures were low; but patients starting with a high supplemental blood pressure had at least as good an outlook for 5 or 8 years as those with a low supplemental pressure. The basal and supplemental blood pressures were almost independent variables. Further studies (Smirk, and supplemental blood pressures were almost independent variables. Further studies (Smirk, 1964) confirmed a close relationship between basal blood pressure and subsequent mortality, not only in virtually untreated hypertensive patients but in hypertensive patients treated with effective hypotensive drugs. No such close relationship is apparent in a 5 and 8 years follow-up period, between the supplemental pressure and the subsequent mortality of hypertensive patients.

It seemed appropriate to compare the casual, basal and supplemental pressures of first-degree relatives of substantial hypertensive patients with corresponding observations on population control subjects. Such observations relate to early stages in the development of hypertension.

Methods

Basal blood pressures on our first-degree relatives of hypertensive and population control subjects were obtained after volunteers had spent a night in a single room under quiet conditions with a mild sedative (pentobarbitone, 100 mg), and next morning they were habituated to the blood pressure procedure by repeated measurement of the blood pressure at intervals of about half a minute, for a period usually of 10 or 15 min. Habituation is an important part of the procedure. This was done in a single quiet room with no interruptions and no conversation apart from an initial greeting. This technique gave lower basal pressures than those reported by Addis. We used the term supplemental pressure for the casual minus the basal blood pressure; this supplemental pressure is the more variable or labile part of the casual blood pressure. It represents the response of the individual to the current environment: physical, psychological and probably metabolic.

519 relatives were studied: of these 252 (48.6%) were siblings, 247 (47.6%) were children and 20 (3.8%) were parents of patients with hypertension.

Results

In Fig. 1 the results in males are set out on the left and in females on the right, for age groups 15–19 years up to 70–79 years. The top pair of graphs compare the casual systolic blood pressures of the first-degree relatives of substantial hypertensive patients (R) with the casual systolic blood pressures of population control subjects (C). The second pair set out the basal systolic blood pressures of relatives and control subjects. The third pair show the basal diastolic blood pressures of relatives and control subjects. The fourth pair show the supplemental systolic blood pressures of the relatives of hypertensive patients and population control subjects. The fifth pair compare the supplemental diastolic blood pressures of the relatives of hypertensive patients and population control subjects.

The casual diastolic pressures are not shown but can be constructed from the sum of the supplemental and basal diastolic pressures.

In both males and females the casual and basal systolic blood pressures of the first-degree relatives of hypertensive patients exceeded those of the population control subjects.

In the age groups 15–19, 20–29 and 30–39 years of males and females the casual systolic, basal systolic and basal diastolic pressures of the relatives of hypertensive patients do not exceed the corresponding blood pressures of the population control subjects by more than 12 mmHg.

After age group 30–39 years in the relatives there is an acceleration in the rate of rise with age of the casual and basal systolic pressures in males and to a lesser degree in the basal diastolic pressures. In the population control subjects there is also an acceleration in the rate of rise of the blood pressure with age, affecting casual and basal systolic pressures and to a lesser degree the basal diastolic pressures; but this acceleration in the rate of rise of the blood pressure begins 10–20 years later in the population control subjects than in the relatives of hypertensive patients.

In males neither the supplemental systolic nor the supplemental diastolic blood pressure shows any large rise with age. Furthermore the acceleration in the rate of rise of the basal pressure after age group 30–39 years is inconspicuous or absent in the male supplemental systolic and diastolic pressures. Also in contrast to the basal pressures of
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males the supplemental pressures, systolic and diastolic, of the first-degree male relatives show no consistent elevation above those of the male population control subjects.

In the female relatives of hypertensive patients, as in males, there is an acceleration in the rate of rise of the casual and basal systolic and pressures after age group 30–39 years. In the female population control subjects there is also an acceleration in the rate of rise of the casual and basal systolic and diastolic blood pressures with age but they start about 10 years later than in the relatives of hypertensive patients.

In general the basal blood pressures of males and females behave similarly. There are differences, however, in the behaviour of male and female supplemental blood pressures. In females there is a rise with age in the supplemental systolic blood pressure of the first-degree relatives of substantial hypertensive patients and a smaller rise with age in the population control subjects. In the supplemental diastolic blood pressure there is also a rise with age but no consistent difference between the blood pressures of first-degree relatives of hypertensive patients and control subjects. The only large supplemental pressure difference between relatives and the control group was in age group 70–79 years, of females.

A survey of the literature yielded casual blood pressures of fifty-three identical twin pairs. The

Fig. 1. Comparison of blood pressures of relatives (○, R) of patients with essential hypertension and pressures of population control subjects (△, C).
writer encountered twelve additional identical twin pairs. The blood pressures of the like twins were similar. We looked out for hypertensive and among the above found fourteen twin pairs in which both twins were hypertensive, but only one twin pair in which one twin was hypertensive without the other twin being hypertensive.

Discussion

It appears that genetic or, just possibly, familial factors have a strong influence on the basal blood pressure, but little influence on the supplemental pressures.

On clinical evidence we know that hypertension may arise in many disorders, but irrespective of the underlying cause or causes, the eventual consequences of continued high blood pressure, especially the basal blood pressure, are similar, namely: cardiac hypertrophy, ECG changes, congestive heart failure, left ventricular failure, strokes and retinal changes.

These manifestations are secondary to the mechanical event, a rise of pressure, whatever the cause of the mechanical event may be.

Now if high blood pressure as such, irrespective of the cause, leads on to these characteristic manifestations of hypertension, it follows that over-average blood pressures from physiological causes will also contribute to the pathogenesis of essential hypertension and of other types of hypertension.

Elevated blood pressures, even though produced genetically and physiologically, predispose to and lead in the course of time to characteristic pathological changes in the heart and circulation (Smirk, 1946, 1949, 1967, 1972). Some of the changes represent work hypertrophy of heart and blood vessels and others are accelerated involutionary changes.

There is evidence that hypertension accelerates the development of renal ischaemia, and this may cause a further blood pressure elevation at a later stage in the disorder by a renal mechanism (Goldblatt, 1947).

Also, increase in the vascular and cardiovascular reactivity may be secondary to early rises of blood pressure or might be more directly of genetic origin. The blood vessels of our rats with spontaneous genetic hypertension certainly show increased reactivity to pressor agents.

Doyle & Fraser (1961) made a most important observation which links vascular reactivity with genetic factors in man. They infused noradrenaline into the brachial artery and found greater vasoconstriction in the forearm in the normotensive offspring of hypertensive parents than in a control group with normotensive parents. It appears that the phenomenon of increased vascular reactivity may precede any rise of blood pressure.

Obesity and broad body build are influenced genetically and this may make a small contribution to the level of the blood pressure in some hypertensive patients.

The hardening of the arterial reservoir, i.e. the aorta and large vessels, which occurs with age and is accelerated by hypertension is likely to be one of the changes which when accelerated by over-average blood pressure leads on to further rises of pressure as the result of increased resistance to distension of the arterial reservoir during systole.

Many other factors have been described which may contribute to this multifactorial disorder. A question may be raised—how many such factors are influenced genetically?

Using hexamethonium, Doyle & Smirk (1955) showed that an important part of the blood pressure, in essential and other types of hypertension, is neurogenically maintained. A part is also not neurogenically maintained. It is possible that here a genetic background may be involved in that hexamethonium drops the blood pressure considerably in our genetically hypertensive rats (Phelan, Eryetisher & Smirk, 1962; Phelan, 1966).

It is suggested that genetic factors are responsible for much of the initial blood pressure increase in essential hypertension, probably, in the first place, by altering the balance of physiological factors, some tending to raise and others tending to reduce blood pressure levels.

Such a process may help to explain why it has been possible in New Zealand and Japan to develop hypertensive rat colonies by selective breeding (Smirk & Hall, 1958; Phelan, 1966, 1968; Okamoto & Aoki, 1963; Smirk, 1972).

Over-average blood pressures and also changes associated with age induce a number of cardiovascular changes which become responsible eventually for further blood pressure increases (Smirk, 1967).

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