Left ventricular mass in relation to midlife blood pressure

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This editorial refers to ‘Midlife blood pressure change and left ventricular mass and remodelling in older age in the 1946 British Birth Cohort Study’†, by A.K. Ghosh et al. on page 3287.

The 1946 Medical Research Council National Survey of Health and Development (MRC-NSHD) is the longest running birth cohort in the UK. Making use of this unique resource, Ghosh and colleagues have now demonstrated that left ventricular mass index (LVMI) and relative wall thickness at age 60–64 were associated with blood pressure (BP) or use of antihypertensive drugs from 36 years of age onwards, independent of current BP or treatment status.1 Moreover, the rate of BP increase over time rather than the absolute BP level at a given age determined the LVMI at 60–64 years.1 Ghosh and colleagues have to be congratulated for their meticulous analysis of longitudinal data spanning 28 years. Nevertheless, several issues cannot be disregarded in the interpretation of the results. First, as in all longitudinal studies, the attrition rate was high. Of 5362 initially enrolled participants, only 2856 were invited for echocardiography from 2006 onwards, independent of current BP or treatment status.1 Moreover, the rate of BP increase over time rather than the absolute BP level at a given age determined the LVMI at 60–64 years.1 Ghosh and colleagues have to be congratulated for their meticulous analysis of longitudinal data spanning 28 years. Nevertheless, several issues cannot be disregarded in the interpretation of the results. First, as in all longitudinal studies, the attrition rate was high. Of 5362 initially enrolled participants, only 2856 were invited for echocardiography from 2006 onwards, independent of current BP or treatment status.1 Moreover, the rate of BP increase over time rather than the absolute BP level at a given age determined the LVMI at 60–64 years.1 Ghosh and colleagues have to be congratulated for their meticulous analysis of longitudinal data spanning 28 years. Nevertheless, several issues cannot be disregarded in the interpretation of the results. First, as in all longitudinal studies, the attrition rate was high. Of 5362 initially enrolled participants, only 2856 were invited for echocardiography from 2006 onwards, independent of current BP or treatment status.1 Moreover, the rate of BP increase over time rather than the absolute BP level at a given age determined the LVMI at 60–64 years.1

Another potentially clinically important observation emerging from the study of Ghosh et al. is that people with treated hypertension had higher LVMI, even after adjustment for current BP. However, the MRC-NSHD report provided no information on the persistence of treatment over time, the BP levels reached on treatment, or exception made for the treatment status at 60–64 years on the drug classes prescribed. Nevertheless, Ghosh’s observations1 are in line with the results of the Pressioni Arteriose Monitorate E Loro Associazioni (PAMELA) study.4 The 2051 participants were categorized based on their office, home, or 24 h BP into normotensive individuals and patients with hypertension, who were untreated, treated but not controlled, or treated and controlled.4 Compared with the normotensive group, LVMI was higher not only in untreated hypertensive patients, but also in treated uncontrolled patients and even in treated and controlled patients (79.7 vs. 103.4 and 94.3 g/m², respectively). The same was true for the prevalence of left ventricular hypertrophy (4.2 vs. 14.3, 28.7, and 19.0%, respectively).4 Ghosh and colleagues speculated that an elevated BP in midlife causes a ‘legacy’ of cardiac damage including interstitial and perivascular fibrosis that may be difficult to reverse even if BP is subsequently well controlled.

The opinions expressed in this article are not necessarily those of the Editors of the European Heart Journal or of the European Society of Cardiology.

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with medication. In line with this hypothesis, we previously demonstrated that normalization of the ambulatory BP on antihypertensive drug treatment does not eliminate the lifetime cardiovascular burden associated with the previous elevated BP, perhaps because blood pressure normalization by itself does not address other risk factors that cluster with hypertension.

Vasan and colleagues evaluated association of current BP (at baseline), recent antecedent BP (average of readings for all available examinations 1–10 years before baseline), and remote antecedent BP (average for all available examinations 11–20 years before baseline) with the 10-year cardiovascular risk in 2313 Framingham participants. They reported that antecedent BP is a major determinant of the future cardiovascular risk above and beyond current BP. In a subsequent report, the Framingham investigators noticed that higher midlife BP and body mass index predict heart failure later in life. A pooled analysis of seven US cohorts involving 61 585 subjects, 700 000 person-years of follow-up starting at age 55, and prior BP data over an average of 14 years. Individuals who maintained or decreased their BP to normal levels had the lowest remaining lifetime risk for cardiovascular disease, 22–41%, compared with individuals who had or developed hypertension by 55 years of age, 42–69%. Among 4217 Framingham study participants, who underwent up to four serial echocardiographic examinations, sex, age, body mass index, systolic BP, antihypertensive treatment, smoking, and diabetes mellitus were the determinants of tracking of left ventricular mass over the adult life course. At variance with Ghosh's findings, women experienced a steeper increase in left ventricular mass with advancing age and increasing body mass index compared with men. We also found in a large international database that the risk associated with the level of the ambulatory BP increased more steeply in women than in men (Figure 1).

In conclusion, the report by Ghosh and colleagues confirms the results of previously published cohort studies that all showed that BP early in life is a major determinant of adverse cardiovascular outcomes later in life. Investigators in the Global Burden of Diseases Study 2010 reported that high blood pressure is the leading risk factor for the global disease burden, and is estimated to cause 9.4 million deaths every year—more than half of the estimated 17 million deaths per year caused by cardiovascular disease. As demonstrated by Ghosh et al. and other investigators, antihypertensive drug treatment does not completely reverse the risk associated with the BP burden early in life, in particular when the BP load is allowed to persist for many years or in the presence of other risk factors. The take home message of Ghosh's report is therefore an appeal to overcome therapeutic inertia. Patients and caregivers should commence and maintain antihypertensive drug treatment as soon as the diagnosis of hypertension is confirmed, preferably by ambulatory BP monitoring, and should not rest until BP levels are at goal. In contrast to Ghosh's contention, women are a particularly vulnerable group and represent a group in whom early treatment of hypertension represents an opportunity to increase years and quality of life.

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**References**


