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RAPID COMMUNICATION
CVD risk factors

Within-visit blood pressure reactivity and incident hypertension: findings from the prospective KORA cohort

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Hypertension remains a leading modifiable cardiovascular risk factor worldwide.[1] Current guidelines recommend averaging the second and third blood pressure (BP) readings during a clinical visit, thereby discounting the typically highest first measurement.[2,3] This transient initial elevation, termed the alerting response (AR), is distinct from white-coat hypertension: it is captured within a single visit rather than by contrasting office and ambulatory values. Although generally regarded as a benign measurement artefact, an AR observed in approximately 10% of a community sample was associated with roughly doubled cardiovascular mortality over 20 years, independent of mean BP.[4] We therefore examined whether within-visit BP reactivity was associated with incident hypertension in the population-based Cooperative Health Research in the Region of Augsburg (KORA) study cohort.

We used data from the population-based KORA S3 survey (1994/95) in Augsburg, Germany.[5] Three BP readings were obtained at 3 min intervals according to the WHO-MONICA protocol. AR was defined as BP in the hypertensive range (≥140 systolic and/or ≥90 mmHg diastolic) at the first reading with normotension at the mean of readings 2 and 3, so affected participants would be classified as normotensive by guideline-based assessment. Hypertension at the F3 follow-up was defined as mean systolic BP ≥140 mmHg and/or mean diastolic BP ≥90 mmHg based on readings 2 and 3, or current antihypertensive treatment, consistent with the established KORA definition; no age-specific thresholds were applied. BP phenotypes were defined from measured values irrespective of medication use. Multivariable logistic regression examined AR and follow-up hypertension, adjusting for sociodemographic, lifestyle, cardiometabolic, and psychosocial factors. Sensitivity analyses restricted the sample to participants treatment-naive at baseline and additionally adjusted for baseline mean systolic and diastolic BP. Systolic reactivity, defined as the change in systolic blood pressure (ΔSBP), was also modelled continuously as the first systolic reading minus the mean of readings 2 and 3, per 5 mmHg.

Among 2847 participants, 287 (10.1%) met AR criteria, with higher prevalence in men than women (11.5% vs. 8.8%, $P = 0.018$). Compared with normotensive individuals, those with AR had higher body mass index (BMI), greater physical inactivity, more frequent total-to-HDL cholesterol ratio ≥5, and approximately twice the prevalence of cardiovascular disease (8.0% vs. 4.3%) and type 2 diabetes (4.2% vs. 1.9%). Antihypertensive use was also more common, consistent with the greater cardiovascular disease and diabetes burden. Depressive symptoms, sleep complaints, somatic symptom burden, and resting pulse did not differ.

Over approximately 10 years, the treatment-inclusive hypertension outcome occurred in 28.7% of the normal-BP group, 61.8% of the AR group, and 84.5% of participants with baseline hypertension (*Figure 1*, panel A). Regression analyses were restricted to 1912 participants without measured

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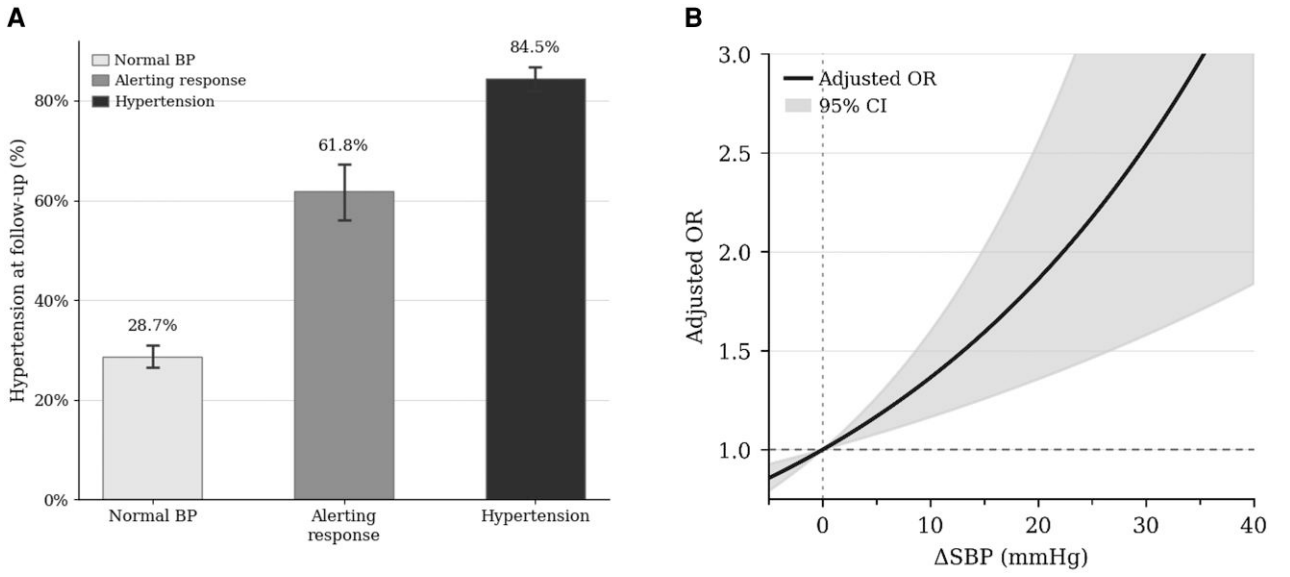

Figure 1 Within-visit blood pressure reactivity and incident hypertension over 10 years. (*A*) Proportion of participants meeting the primary hypertension outcome at F3, defined as mean systolic blood pressure ≥140 mmHg and/or mean diastolic blood pressure ≥90 mmHg based on readings 2 and 3, or current antihypertensive treatment, according to baseline blood pressure phenotype: normal blood pressure (first reading and mean of readings 2–3 both normotensive), alerting response (first reading ≥140 systolic and/or ≥90 mmHg diastolic with mean of readings 2–3 normotensive), and hypertension (mean of readings 2–3 ≥ 140 systolic and/or ≥90 mmHg diastolic). Bars show observed proportions; error bars indicate 95% Wilson confidence intervals. Panel A uses the same treatment-inclusive outcome definition as the regression analyses. (*B*) Continuous association between within-visit systolic blood pressure reactivity (ΔSBP, defined as the first systolic reading minus the mean of the second and third systolic readings, in mmHg) and the adjusted odds of incident hypertension at follow-up, among participants without hypertension at baseline. The solid line represents the adjusted odds ratio per 5-mmHg increase in ΔSBP, with the 95% confidence interval shaded. Estimates are derived from a multivariable logistic regression model adjusted for age, sex, body mass index category, alcohol consumption, smoking status, education, sleep complaints, depressive symptoms, parental history of hypertension, cholesterol ratio, physical activity, and history of cardiovascular disease. The dashed horizontal line indicates an odds ratio of 1.0; the vertical dotted line indicates a ΔSBP of 0.

hypertension at baseline. After full adjustment, AR was associated with nearly twice the odds of hypertension (odds ratio [OR] 1.90, 95% confidence interval [CI] 1.43–2.51; $P < 0.001$), with no evidence of interaction by sex ($P = 0.28$). The association was stronger among participants treatment-naive at baseline (OR 2.74, 95% CI 2.03–3.70) and persisted after adjustment for baseline mean systolic and diastolic BP (OR 1.43, 95% CI 1.04–1.97; $P = 0.029$). Each 5-mmHg increase in ΔSBP was associated with higher odds of follow-up hypertension (OR 1.17, 95% CI 1.08–1.26; $P < 0.001$; *Figure 1*, panel B).

An AR during routine BP measurement was independently associated with incident hypertension in a normotensive community sample. It therefore identifies individuals whose first measurement, despite being discarded in guideline-based averaging, carries prognostic information. Prior studies have linked cardiovascular reactivity with subsequent cardiovascular risk and have reported associations of ARs with target-organ outcomes.[6–8] Mental health and cardiovascular reactivity have also been examined in relation to hypertension development.[9,10] These findings provide context for the observed prognostic association but do not establish its mechanism. In our study, psychosocial burden and resting pulse did not differ between AR and normotensive groups; these descriptive findings do not allow us to distinguish vascular, autonomic, behavioural, or other explanations. As with any within-visit difference score, reactivity is subject to regression towards the mean; nevertheless, random baseline fluctuation would not be expected to predict hypertension a decade later, and the association persisted after adjustment for baseline mean BP.

Strengths include the prospective design, long follow-up, standardized BP assessment, comprehensive adjustment, and consistent categorical and continuous results. Limitations include the observational design, precluding causal inference, and assessment at a single baseline visit, preventing evaluation of AR stability. Data characterizing participants who did not return were unavailable, so differential attrition cannot be excluded. Although less healthy participants were preferentially lost in KORA, the direction and magnitude of any resulting bias cannot be determined without knowing how attrition jointly related to baseline AR and subsequent hypertension risk. Drug classes and treatment thresholds have changed since the study period, although results were consistent among treatment-naive participants. BP and treatment changes during follow-up were not fully captured, and generalizability may be limited to similar populations.

Together with our prior cardiovascular-mortality findings,[4] these results suggest that AR may be an early marker in the development of hypertension. Among individuals classified as normotensive by guideline-based averaging, 10-year

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hypertension incidence was 61.8% with AR vs. 28.7% without, an absolute difference of 33.0 percentage points. This does not imply that AR warrants immediate treatment; rather, the routinely discarded first reading may aid risk stratification and identify individuals whose BP trajectory merits closer attention.

Author contributions

Seryan Atasoy (Conceptualization [lead], Formal analysis [lead], Writing-original draft [lead]), Karl-Heinz Ladwig (Writing-review & editing [equal]), Heribert Sattel (Formal analysis [supporting], Writing-review & editing [supporting]), Annette Peters (Data curation [equal], Resources [lead], Writing-review & editing [supporting]), Ina-Maria Rückert-Eheberg (Data curation [lead], Writing-review & editing [supporting]), Margit Heier (Data curation [lead], Writing-review & editing [supporting]), Birgit Linkohr (Data curation [lead], Writing-review & editing [supporting]), and Peter Henningsen (Supervision [lead], Writing-review & editing [equal])

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Conflict of interest: None declared.

Data availability

The data underlying this study cannot be made publicly available due to data protection regulations. Data are available upon request by means of a project agreement, subject to approval by the KORA Board. Applications and further information on the data access procedure can be found on the KORA-PASST platform (https://epi.helmholtz-muenchen.de/) or by contacting the KORA Study Centre.

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