Why stable blood pressure isn't always stable: A hidden stroke risk?

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Blood pressure variability between clinic visits has been linked to vascular events before, but whether it matters independently of the average reading itself, and whether that risk differs by glycaemic status, has been unclear. A new UK Biobank analysis presented at the EASD Annual Meeting in Milan puts numbers on both questions, using visit-to-visit changes in blood pressure tracked over a median of five years.

KEY TAKEAWAYS

- The Pivot: Blood pressure variability between visits predicts death and stroke independently of the average blood pressure reading, a factor not routinely considered in clinical assessment.
- The Data: 10-year model-predicted mortality ranged from 10.3 per 1,000 (normoglycaemia, low variability) to 28.6 per 1,000 (type 2 diabetes, high variability).
- The Action: Dr Ahmadizar's team suggests variability could help flag high-risk patients missed by average blood pressure alone, though clinical use isn't recommended yet.

Researchers led by Dr Fariba Ahmadizar at Amsterdam University Medical Centre included 57,611 UK Biobank participants with blood pressure measured at baseline and at least one repeat visit, spanning normoglycaemia, prediabetes and type 2 diabetes. Annualised absolute proportional changes between consecutive visits in systolic, diastolic, mean arterial and pulse pressure were modelled as time-updated exposures, with age-timescale Cox models adjusting for interval mean blood pressure, glycaemic status, and standard cardiovascular risk factors.

Variability mattered even after accounting for average blood pressure

Over a median of 5.1 years, 319 participants developed dementia, 571 had a stroke, and 1,460 died. Every blood pressure variability measure was independently associated with all-cause mortality (hazard ratio per 1-SD increase, 1.08 to 1.11, all significant after false discovery rate correction). Diastolic and mean arterial pressure variability were also associated with stroke (HR 1.12 and 1.10 respectively). Associations with dementia did not survive correction for multiple testing. Combining glycaemic status with blood pressure variability sharpened the picture considerably. Relative to normoglycaemia with low variability, prediabetes with high systolic variability carried a 62% higher mortality risk (HR 1.62). Type 2 diabetes with high variability in any of the four blood pressure measures carried roughly two-and-a-half to nearly three times the mortality risk: systolic HR 2.80, diastolic HR 2.59, mean arterial HR 2.60, and pulse pressure HR 2.34, all statistically significant after correction. No multiplicative interaction between glycaemic status and variability reached significance, and adding blood pressure variability to the model barely changed the glycaemia-associated risk

estimates (attenuation of only 0-2%), meaning variability adds independent information rather than explaining away the risk already known to come from glycaemic status.

What the absolute numbers look like

Translated into model-predicted 10-year mortality per 1,000 people, by glycaemic status and systolic blood pressure variability tertile:

Glycaemic statusLow variabilityIntermediate variabilityHigh variabilityNormoglycaemia10.311.113.4Prediabetes12.512.616.6Type 2 diabetes17.922.328.6The gap between the lowest-risk cell (normoglycaemia, low variability, 10.3 per 1,000) and the highest (type 2 diabetes, high variability, 28.6 per 1,000) is nearly three-fold. No conflicts of interest were declared by the authors.

The findings underscore the critical importance of considering blood pressure variability (BPV) as an independent risk factor, particularly in individuals with prediabetes and type 2 diabetes. While average blood pressure remains a cornerstone of cardiovascular risk assessment, this study strongly suggests that the dynamic fluctuations in blood pressure over time contribute significantly to adverse outcomes, even when mean values are within target ranges. This adds a crucial layer of complexity to traditional risk stratification models, moving beyond a static snapshot of blood pressure to incorporate its temporal instability.

Clinically, these results advocate for a more nuanced approach to blood pressure monitoring and management. For patients with prediabetes or type 2 diabetes, routine office blood pressure measurements might not fully capture their cardiovascular risk profile. The study's emphasis on annualised changes between consecutive visits suggests that serial measurements over time, rather than isolated readings, are essential for identifying individuals at higher risk due to elevated BPV. This could involve encouraging patients to engage in home blood pressure monitoring, with a focus not just on the average readings but also on the consistency and range of their measurements. Healthcare professionals should be alert to significant fluctuations in blood pressure between visits, even if the mean values appear controlled.

Clinical Implications and Future Directions

The study's robust findings, particularly the independent association of BPV with all-cause mortality and stroke, necessitate a re-evaluation of current clinical guidelines for hypertension management, especially in the context of impaired glucose metabolism. While existing guidelines primarily focus on achieving specific blood pressure targets, they often do not explicitly address the management of BPV as a distinct therapeutic goal. This research suggests that reducing BPV could represent an unexploited therapeutic avenue to mitigate cardiovascular risk in vulnerable populations.

However, it is crucial to acknowledge that the study, while large and well-conducted, is observational. It establishes an association between BPV and adverse outcomes but does not definitively prove causality. Therefore, further interventional trials are warranted to determine whether specific pharmacological or non-pharmacological interventions

aimed at reducing BPV can translate into improved clinical outcomes, particularly a reduction in stroke and mortality. Such trials would need to carefully define and measure BPV, select appropriate interventions (e.g., specific antihypertensive drug classes, lifestyle modifications), and track long-term cardiovascular events. For instance, some evidence suggests that certain classes of antihypertensives, such as calcium channel blockers and angiotensin receptor blockers, may exert a greater effect on reducing BPV compared to others, but this requires confirmation in dedicated outcome trials.

Moreover, the precise mechanisms linking BPV to increased stroke risk remain an area of active investigation. Hypotheses include increased shear stress on arterial walls leading to endothelial dysfunction and atherosclerosis, impaired cerebral autoregulation making the brain more vulnerable to perfusion fluctuations, and microvascular damage. Understanding these underlying pathophysiological pathways could inform the development of targeted therapies. The study's inability to establish a significant association with dementia after correction for multiple testing also highlights the complexity of cerebrovascular disease and the need for further research into the specific impact of BPV on cognitive decline, potentially with longer follow-up periods or more granular cognitive assessments.

In the interim, clinicians should consider BPV as an additional layer of risk assessment, particularly for patients with prediabetes and type 2 diabetes. While there are no established guidelines for directly treating BPV, a holistic approach to cardiovascular risk reduction, including optimal glycemic control, lipid management, and consistent blood pressure control with appropriate medication choices, may indirectly contribute to reducing BPV. Educating patients about the importance of consistent blood pressure readings and encouraging regular, accurate home monitoring can empower them to be more engaged in their care and provide valuable data for clinicians to identify patterns of high variability. This proactive monitoring and personalized risk assessment represent a significant step forward in preventing adverse cardiovascular events.

> **CLINICAL IMPLICATIONS**
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> This data argues for looking at the pattern of blood pressure readings over time, not just the latest number or the running average, particularly in patients with prediabetes or type 2 diabetes, where the combination with high variability carried the steepest risk in this analysis. Dr Ahmadizar herself is measured about next steps: "more research is needed before blood pressure variability can be recommended for routine clinical use. Questions that need answering include whether it is possible to reduce these fluctuations, and if doing so reduces the risk of stroke or death."
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> As an observational study, this cannot establish that reducing blood pressure variability would itself lower risk, variability could be a marker of underlying vascular fragility or autonomic dysfunction rather than a modifiable target in its own right. The authors are explicit that the study did not prove causality.
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> The practical takeaway for now is a research one rather than a bedside protocol change: blood pressure variability is a real independent mortality signal on this data, but no intervention trial yet exists to show that acting on it changes outcomes.

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