## Revisiting blood pressure thresholds to define hypertension during pregnancy: is 140/90 mmHg too high?







While definitions for pre-eclampsia and hypertensive disorders of pregnancy have drifted over several decades—from a triad of signs in the 1950s to hypertension and multi-organ involvement in 2018 one constant has been the cutoff to define hypertension: 140 mmHg systolic blood pressure (sBP) and 90 mmHg diastolic blood pressure (dBP). Long used as a reliable border separating normal from abnormal, this simple cutoff has been called into question.

In 2017, the American College of Cardiology and the American Heart Association lowered the threshold to diagnose hypertension outside of pregnancy. Stage 1 hypertension was introduced, defined as a sBP of 130-139 mmHg or dBP of 80-89 mmHg, or both. The reasoning seems sound: such degrees of blood pressure are associated with an increased lifetime risk of major cardiovascular events, such as a doubling of stroke risk.<sup>1</sup> The lowering of this threshold leads to the guestion: should we now lower the definition of hypertension to 130/80 mmHg or greater for pregnant women as well?

There have been reports linking blood pressures during pregnancy that would qualify as stage 1 hypertension with an increased risk of pre-eclampsia, gestational hypertension, and preterm birth.2-8 These studies have been retrospective and examined cohorts from highresource settings. Importantly, none of these studies examined potential associations between stage 1 hypertension and very severe adverse events (namely, hard endpoints such as maternal death, stillbirth, or lifethreatening complications), and none have investigated low-income and middle-income countries (LMICs), which shoulder most of the global burden of severe adverse outcomes arising from hypertensive disorders of pregnancy.

Deeming women with a sBP of 130-139 mmHg during pregnancy as hypertensive would incur substantial costs. Around 11-37% more women would be classified as having an increased blood pressure compared with when using the previous threshold, 4,6,9 and these women might need intensive antenatal monitoring and possibly an induction of labour upon reaching term gestation. This would drastically increase workload and drain resources, and in LMICs it could overwhelm

already under-resourced health-care facilities. Therefore, for a change such as this to be implemented—especially in LMICs—we would need to be sure that lowering the threshold to diagnose hypertension clearly improves maternal and neonatal outcomes (especially relative to the cost).

In The Lancet Global Health, Jeffrey Bone and colleagues<sup>10</sup> address the guestion of whether these lower blood pressure thresholds for hypertension in pregnancy are associated with adverse maternal and neonatal outcomes. The authors made use of data from the Community Level Interventions for Preeclampsia (CLIP) cluster randomised trial which was a prospective multicentre study that included more than 21000 women in India, Mozambique, and Pakistan. The pregnant women included were at high risk of adverse outcomes, with one-quarter delivering preterm and one-quarter suffering a severe maternal morbidity (such as eclampsia, stroke, antepartum haemorrhage, or disseminated intravascular coagulation, among others) or a perinatal death. Importantly, blood pressure measurements were prospectively taken (in clinical trial conditions) with semi-automated pregnancyvalidated devices in a standardised method and were simultaneously recorded by trained community health workers.

This study did not identify significant associations between stage 1 hypertension and major adverse maternal (maternal composite, or a maternal composite of adverse neurological events) or perinatal (stillbirth, or fetal or neonatal death) outcomes compared with women with normal blood pressure. By contrast, women with sBP greater than 140 mmHg or dBP greater than 90 mmHq, or both, had two to six times increased risk of these major adverse outcomes.

It is reassuring that no significant links were found between sBP 130-139 mmHg and poor obstetric outcomes. However, we have noticed a dose-dependent increase in all adverse outcomes with worsening levels of hypertension, including stage 1 hypertension, in the findings of Bone and colleagues. Indeed, even for stage 1 hypertension, the four major adverse outcomes they examined all trend towards an increase and in

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the higher blood pressure categories (non-severe stage 2 hypertension, and severe stage 2 hypertension), the associations became stronger and significant. It is therefore plausible that the study was underpowered to detect a true link that exists between stage 1 hypertension and severe adverse outcomes. Even if so, the absolute risk is likely to be low, meaning it would remain debatable whether labelling an additional 17-8% of the entire cohort as hypertensive would be worthwhile.

On balance, we believe the current evidence, including this important contribution by Bone and colleagues, to does not support decreasing the threshold for the diagnosis of hypertension in pregnancy, especially in LMICs. The threshold should only ever be shifted if it were shown that the costs of labelling a large proportion of the pregnant population as hypertensive would be clearly balanced by significant improvements in clinically important adverse outcomes. Otherwise we risk pouring scarce resources into a large population in whom they were not required.

We declare no competing interests.

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