Although Yogi Berra may not have been thinking about postpartum hypertension when he first made this prescient observation, he pretty much nailed it. Older textbooks of obstetrics barely mention elevated blood pressure (BP) as a concern after delivery, and they even opine that, in the majority of cases of preeclampsia, the manifestations of the disease remit within 48 hours.1 We now know that in some women hypertension persists for weeks and even months after delivery,2 and that, if not addressed, postpartum hypertension may lead to lethal cerebrovascular sequelae.3

Twenty-three percent of the women with antepartum hypertension were diagnosed with preeclampsia, 60% with transient hypertension, 9.4% with gestational hypertension, and 7.5% with chronic hypertension.

These antepartum clinical diagnoses were based on the International Classification of Diseases, Ninth Revision codes, and are thus subject to error.7 The most recent American College of Obstetricians and Gynecologists guidelines8 recommends abandoning the older designation transient hypertension (hypertension diagnosed after 20 weeks gestation, without other laboratory signs of preeclampsia or systemic organ dysfunction),9 in favor of the term gestational hypertension. In the Goel article,4 both categories (transient and gestational hypertension) are reported, and the majority of subjects were actually diagnosed with transient hypertension. The clinical characteristics of this group are not presented; thus, one wonders whether these women represented a heterogeneous group of disorders: eg, gestational hypertension and preeclampsia without proteinuria. If we exclude women with transient hypertension, the breakdown of hypertension etiology described in this study would be 57.7% with preeclampsia, 23.5% with gestational hypertension, and 18.8% with chronic hypertension. This is similar to the diagnoses assigned in the 1998 to 2006 Nationwide Inpatient Sample study (45%, 36.8%, and 18% respectively).6 The slightly higher proportion of women with preeclampsia again could be attributed to the cesarean delivery–only population that is studied here.

The patients with preeclampsia and transient hypertension appeared more likely to develop postpartum hypertension (PP HTN) in comparison with women with chronic or gestational hypertension, and it would be interesting if the angiogenic factors were more abnormal in these groups in comparison with the other 2 categories. These results are consistent with the possibility that the transient hypertension subgroup is a heterogeneous group and may include women with undiagnosed preeclampsia. Although it is reported that women with gestational hypertension have lower maternal morbidity, including cerebrovascular disorders and mortality, than eclampsia/pre eclampsia,10 it is well recognized that some (10%–25%) will develop preeclampsia before delivery. The uncertainties in the diagnostic categories in the Goel study4 preclude making conclusions regarding the association of diagnostic category with risk of PP HTN. However, one could argue that postpartum, the level of BP, rather than the diagnostic category, is more relevant, because fetal well-being is no longer a concern, and the prevention of maternal cerebrovascular and cardiovascular events is the goal of treatment.

Among the women who were normotensive before delivery, 10% (n=77) developed de novo hypertension postpartum (BP 140/90 mm Hg 48 hours or more after cesarean delivery and up to 6 weeks postpartum). Among the women who already had a diagnosis of hypertension before delivery, 50% (n=107) continued to have hypertension in the postpartum period. Women with de novo, and persistent postpartum hypertension, as well, had a higher body mass index at delivery and were more likely to have a history of diabetes mellitus than women who never developed PP HTN or who became normotensive after delivery. These observations are important...
in identifying subgroups that may require greater surveillance in the postpartum period.

Goel and colleagues have made an important contribution by highlighting the relatively high incidence of postpartum hypertension. Until now, the focus of pregnancy-associated hypertension research has been on antepartum hypertension, with only a few studies and reviews addressing the postpartum period. There is no doubt that antepartum hypertension is a high-risk situation, and that delivery of a healthy, live baby is of utmost importance. Fortunately, maternal catastrophes such as death, stroke, and seizures are rare, but there is mounting evidence that the incidence of postpartum thrombotic and hemorrhagic strokes is increasing just as maternal hypertension is rising. Data from the Nationwide Inpatient Sample demonstrated that between 1994 to 1995 and 2006 to 2007 the rate of postpartum hospitalizations for stroke increased from 0.12 to 0.22 per 1000 deliveries (83%).

Postpartum is a hypercoagulable state, the risk being greatest in the puerperium. The risk of thrombotic events substantively increases within 6 weeks postpartum (odds ratio, 22.8) and normalizes after 12 weeks postpartum (odds ratio, 1.0). Indeed, the majority of pregnancy-associated strokes occur in the first 48 hours after delivery, and hypertension is the strongest risk factor. Hypertensive disorders or heart disease was present in 53% of cases of stroke, and 10% to 13% of women died. A Swedish cohort study also reported that cerebral infarction was 33 times more likely to develop in the 3 days surrounding delivery, and 8.3 times more likely in the subsequent 6 weeks postpartum. Similarly, the risk of intracerebral hemorrhage rises sharply in the puerperium and is 11.7 times more likely than antenatally. Therefore, early diagnosis and management of PP HTN has the potential to prevent morbidity and even lethal sequelae. Hypertension, especially preeclampsia, is clearly one of the most significant risk factors for pregnancy-associated stroke and maternal death. In the United Kingdom, between 2006 and 2008, the majority of the maternal deaths from severe preeclampsia and eclampsia were related to either intracranial hemorrhage or eclampsia.

The observation by Goel et al that abnormalities in angiogenic factors are associated with postpartum hypertension suggests that women with pathophysiologic features of preeclampsia are especially at risk, because these biomarkers have been shown to reliably distinguish preeclampsia from other forms of hypertensive disorders in pregnancy. In their study, antepartum sFlt1 levels were higher and PlGF levels were lower in women with postpartum hypertension than in women who were normotensive postpartum. In multivariable analysis, being in the highest tertile of antepartum, sFlt1/PlGF was associated with the development of PP HTN, odds ratio 2.25 (1.19–4.25; P=0.02). The observation that these alterations in angiogenic factors were present even in women who were not diagnosed with preeclampsia before delivery suggests that a subclinical form of the disease may have been present. Indeed, postpartum preeclampsia has always been an enigmatic syndrome, and although most women who are given this diagnosis do not show signs of preeclampsia antepartum, the prevailing wisdom/suspicion is that the disease was present but not detected. The current findings are consistent with this view.

Although not emphasized by the authors, their results suggest a possible predisposing factor for the high incidence of PP HTN. Ninety percent of their patients were treated with nonsteroidal anti-inflammatory drugs (NSAIDs) for post–cesarean delivery analgesia, a practice that has become widespread in the past decade. Parenteral ketorolac and oral ibuprofen (600–800 mg every 6 or 8 hours) are routinely prescribed for pain control in the postdelivery period. That NSAIDs can increase BP in nonpregnant subjects, especially those with hypertension, is well established. Although not well studied, there are conflicting data about their contribution to BP elevation in the puerperium. The recent American College of Obstetricians and Gynecologists guidelines discourage the use of NSAIDs in patients with diagnosed hypertension, and we endorse this position and would recommend the discontinuation of NSAIDs if PP HTN develops. In our experience, hypertension usually becomes apparent a few days (2–4) after surgery once the effects of magnesium, anesthesia, and fasting have subsided. This also corresponds to the time when extracellular fluid relocates into the vascular space and the increased BP associated with NSAIDs would likely be detected. The effect of these factors has not been carefully studied in clinical trials; nevertheless, we suggest that a potential strategy for minimizing PP HTN would be to use alternative postpartum analgesia such as acetaminophen and narcotics. Given the rare, but devastating potential consequences of PP HTN, we congratulate Goel et al for highlighting the high prevalence and the need for additional investigation regarding prevention, pathophysiology, and treatment.

Disclosures
None.

References


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Postpartum Hypertension: "It Ain't Over 'til It's Over"
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