

Commentary on the Management of Elevated Blood Pressure in Pregnancy

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The position paper on Hypertension in Pregnancy from the American Society of Hypertension (ASH), which was published in volume 11, issue 4 of *The Journal of Clinical Hypertension* represents the opinions of obstetrics, gynecology, and internal medicine experts. Current guidelines for the management of hypertension in pregnancy are clearly outlined by the authors who have had a long experience in managing this disease. Pathophysiologic changes that occur in normal pregnancy, hypertension in pregnancy, and toxemia of pregnancy are summarized.

It is difficult to critique a position paper that is well written and carefully evaluates this serious problem, but some specific comments regarding treatment are in order. These comments are based on a 2-year experience in a toxemia clinic and 50+ years of experience in treating elevated blood pressure (BP) in pregnant women. Since there are few well-controlled study data available regarding specific therapy, the comments made represent suggestions to rethink the presently recommended treatment of pregnant women who have or develop elevated BP.

It is well-known that BP decreases during the first trimester of pregnancy and that normal BPs during this time may be as low as 100/70 mm Hg. BP tends to rise to levels of about 120 to 125/80 to

85 mm Hg in the second and third trimester. Thus, systolic BPs of 130–140 mm Hg or diastolic BPs >85 mm Hg represent abnormally elevated BPs during this period. The ASH report notes that a systolic BP of 140 mm Hg represents an abnormal BP in a pregnant woman. There is some evidence that diastolic BP >85 mm Hg at any stage of pregnancy will increase fetal mortality. Many guidelines and the ASH paper do not, however, suggest treatment until levels of about 160/105 mm Hg are recorded. There also does not appear to be a major distinction made with regard to BP levels that require specific therapy between women who are hypertensive before pregnancy and those who develop toxemia with elevated BPs. Is this recommendation based on good science or fear that giving medication may result in a poor outcome that, as the position paper states, “may have to be defended before official boards or committees”?

The argument has been repeated that elevated BPs of 140 to 150/90 to 95 mm Hg over a short period probably do not result in any serious effects on the vascular system in pregnant women. Therefore, guidelines suggest that these levels of BP may not require treatment. Abnormal clinical findings may not be detectable in patients with these levels of BP. However, if careful vascular studies are done, endothelial dysfunction, early left atrial and perhaps left ventricular changes, and vascular changes in the kidney may be detected in people whose BPs have been elevated for just a 3- to 4-month period. There is also some suggestive evidence that long-term cardiovascular events may be more frequent in people who have untreated elevated BP for short periods.

We suggest, based on the available data but with little solid long-term evidence (which, by the way,

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may never be obtained) that lowering BPs that are higher than 130 to 140/80 to 85 mm Hg should be undertaken in pregnant women rather than waiting for higher levels, as suggested in the position paper. We are aware that lowering BP may not prevent toxemia of pregnancy or preeclampsia but we are also aware that this may prevent further elevations of BP and may delay the need for intervention. The position paper clearly states that “gestation is permitted to continue as long as BP is controlled” (and, of course, no overt signs of life-threatening maternal or fetal complications). If BP levels are controlled before reaching the presently recommended levels for treatment, it is possible that fewer instances of extremely high BP will occur.

The National High Blood Pressure Education Program (NHBPEP) and American College of Obstetricians and Gynecologists guidelines “accept withholding antihypertensive drugs unless diastolic levels are above 100 mm Hg (but support treatment at lower levels if there is evidence of end-organ damage or specific risk factors such as underlying renal disease, which often is clinically difficult to detect). They note that “end points” for reinstating treatment include exceeding a threshold BP of 150 to 160 mm Hg systolic and 100 to 110 mm Hg diastolic.

Obstetricians have a legitimate concern—medications that might adversely affect the fetus should be avoided. They continue to use alpha-methyl-dopa, hydralazine, and certain vasodilating β -blockers, medications that they are comfortable with. Of course, they have had more experience with these agents—these are the medications they have been recommending for years. In the Fifth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC), it was suggested that a hypertensive woman who becomes pregnant should continue taking her usual medication with the exception that the use of any renin-angiotensin aldosterone inhibitor (angiotensin-converting enzyme or angiotensin receptor blocker) be stopped. These have been associated with fetal abnormalities. The JNC report advocated the continued use of calcium channel blockers, β -blockers, or diuretics if necessary to keep BP below the goal of 140/90 mm Hg. As noted, it is recognized that during the first trimester, BP may decrease and medication may have to be decreased or temporarily discontinued. However, when BP begins to rise, even to levels of 130 to 140/80 to 85 mm Hg, it would appear logical that medications should again be increased or

restarted. At present, there is only limited evidence that this approach will prevent the development of higher levels of BP later on in pregnancy, but there is no firm evidence to suggest that lowering BP slowly will be detrimental to either the mother or the fetus. Quite the opposite might be true. In one meta-analysis of a large number of patients treated with a diuretic, for example, the occurrence of preeclampsia was reduced from 10.8% in a control group to 7.7% in the treated group.

The ASH position paper advised that the risk of complications “correlates with the age of the patient, the duration and degree of control of her BP, and the presence of organ damage.” They note that trials that have determined outcome in women with pre-pregnancy hypertension (chronic hypertension) whose BP is treated during pregnancy may be flawed but do suggest fewer hospitalizations of the mother, especially related to better BP control, with little definitive evidence of harm to the fetus. There is also little evidence, as noted, that this will prevent preeclampsia or toxemia. But treating elevated BPs below levels now advocated in the ASH position paper appears to be logical and without harm. Keeping BPs at more normal levels should help to prevent the rare cases of cerebrovascular hemorrhage or heart failure in pregnant women.

Our experience with the treatment of hypertension in pregnancy cannot possibly match that of the authors of the position paper. Our experience, however, in treating hypertension before a crisis occurs and maintaining BPs below 140/90 mm Hg throughout pregnancy or of treating women who develop toxemia before BP levels rise to presently recommended levels suggest that some cases of progressive hypertension may be prevented. We are aware of initial studies suggesting that the use of β -blockers during pregnancy may result in smaller birth weights. This, however, has not been confirmed in other studies. We are also aware that placental infarcts are often present in women who are hypertensive during pregnancy and that birth weights of babies born to women who are hypertensive are often lower than anticipated regardless of therapy. These findings and not the effects of specific medications may account for smaller babies in hypertensive women.

Some studies have suggested that a decrease in placental blood flow occurs when diuretics are used. These studies, however, have been questioned especially with regard to the methods used to determine flow. There is little evidence that the use of diuretics in pregnancy is dangerous and some evidence, as noted, that the BP-lowering effects of

Table. Suggested Simplified Approach to the Management of Hypertension in Pregnancy ^a		
	THERAPY	COMMENTS
Chronic hypertension (pre-pregnancy)	Continue medication: diuretics, CCB, or β -blocker (stop ACEI or ARB)	Dosage should be reduced during first and mid-second trimester; if BP >140/90 mm Hg, therapy should be increased or restarted
De novo elevation BP >20 weeks (with or without proteinuria or edema)	Specific antihypertensive therapy, CCBs, diuretics, β -blockers, if BPs >140/90 mm Hg ^b	Possibility that preeclampsia or eclampsia may not be prevented; reduction in maternal complications
Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BP, blood pressure; CCB, calcium channel blocker. Other measures that are currently used in treating toxemia of pregnancy and the use of intravenous therapy in eclampsia are clearly indicated. ^a This approach has not been approved by any national or society guideline committees. ^b Alpha-methyldopa and hydralazine may also be used.		

these agents might be beneficial. Yet, obstetric/gynecological textbooks suggest that the use of diuretics may be contraindicated. Since there is a tendency during pregnancy for a volume increase and sodium retention based on increased corticosteroids, it would seem logical that some diuretic use would be necessary. In our limited experience, controlling BP in pregnancy without a diuretic is often difficult.

Controversies remain as to whether and at what level to treat rapidly rising BP near term or during delivery (a phenomenon often indicating the appearance of pure or superimposed preeclampsia). There is further debate on how aggressively to lower the BP. The NHBPEP recommendations state that diastolic levels >105 mm Hg require treatment (although some contemporary texts still recommend >110 mm Hg), with some reservations. Circumstances, such as a young women whose recent BP levels were 110 to 120/70 to 80 mm Hg and have risen rapidly or patients demonstrating signs of cardiac decompensation or cerebral symptoms such as severe headaches, confusion, or somnolence, warrant treatment at lower levels. Perhaps keeping BP at lower levels throughout pregnancy might prevent at least some cases of rapidly rising BPs during the late third trimester.

The Table represents a simplified approach to managing elevated BP in pregnancy. Of course, if BPs rise (1) despite adequate therapy, or (2) if proteinuria increases or signs of platelet dysfunction occur, then other measures (ie, bed rest, magnesium sulfate) and early intervention may be necessary.

Dividing patients into subgroups such as preeclampsia or toxemia superimposed on chronic hypertension, for example, may not be necessary since levels of BP will mainly determine specific

therapy. Obviously, other treatments outlined in the position paper for preeclampsia or eclampsia should be followed.

SUMMARY

It may give physicians some comfort in the management of elevated BP in pregnancy to use drugs such as alpha-methyldopa (the agent of choice among obstetricians), which may or may not be effective in lowering BP adequately and often results in annoying side effects, but this may not be the best choice of therapy. Hydralazine, which has also been used for many years in the treatment of hypertension in pregnancy, often loses its effectiveness over time and may cause tachycardias and headaches; BP may not be lowered effectively. While this is presently recommended, it may also not be the drug of choice. Physicians are comfortable with these medications because they have been using them for many years. This, however, should not be the determining factor in choosing treatment. There are more effective and well-tolerated therapies—diuretics, calcium channel blockers and β -blockers—that should be given before BPs rise to levels currently recommended by some national guidelines. It is time to reexamine these recommendations. Angiotensin-converting enzyme inhibitors or angiotensin receptor blockers should not be given. Maintaining BP at more normal levels should prevent some episodes of maternal cerebral hemorrhage and congestive heart failure. While these are uncommon, they most probably are dependent on the actual levels of BP. Above all, it may be that controlling BP even for just 4 to 6 months may prevent vascular damage in the future. At present there is little evidence that careful lowering of BP with appropriate therapy will be detrimental to the mother or the fetus.